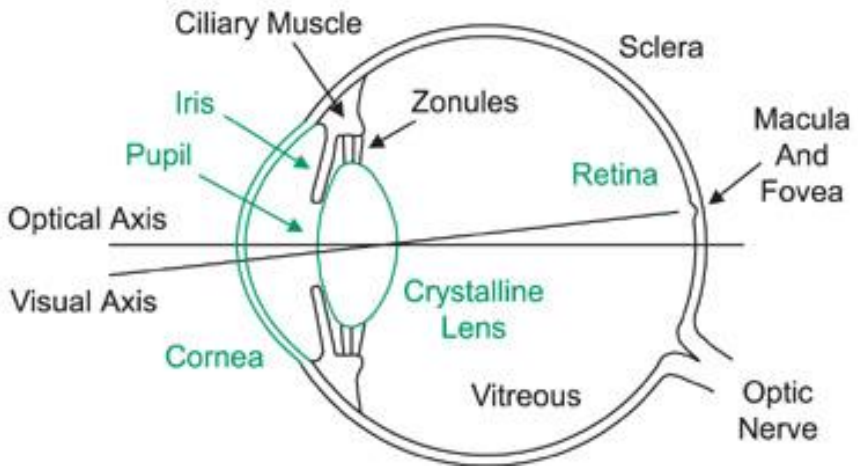
Special sensory: Optics of Eye

The optics of the eye involve the process where light enters through the cornea and lens, creating an inverted, real image on the retina for the brain to interpret. The cornea performs the majority of light refraction, while the adjustable crystalline lens fine-tunes the focus by changing its shape through ciliary muscles. The iris controls the amount of light entering the eye, and the light-sensing retina converts the image into neural signals sent to the brain via the optic nerve.



How Light Enters the Eye

Cornea:

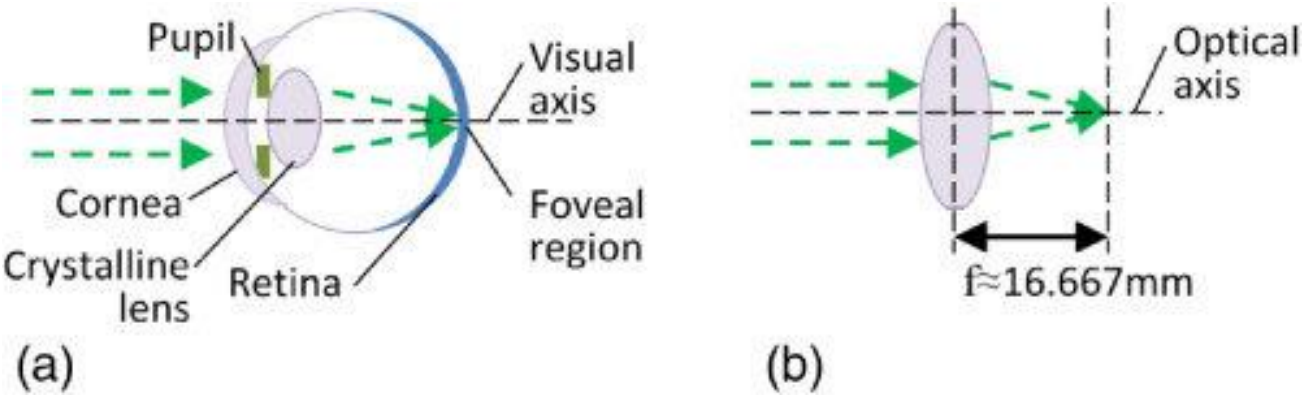
Light enters the eye through the cornea, the clear, curved outer surface at the front of the eye.

Pupil and Iris:

The light then passes through the pupil, the dark opening in the center of the iris. The iris adjusts the size of the pupil to regulate the amount of light reaching the retina.

Lens:

Next, light passes through the lens, a transparent, adjustable structure inside the eye.



Refraction and Image Formation

Refraction:

As light passes through the cornea and lens, it is bent or refracted. The cornea does most of the bending, while the lens provides a smaller, adjustable component of this power.

Accommodation:

The eye's lens changes its shape to adjust its focal length. This process, called accommodation, allows the eye to focus on objects at different distances, ensuring a sharp image is formed on the retina.

Vitreous Humor:

The clear, jelly-like substance filling the space behind the lens is the vitreous humor.

The Retina and Brain

Retina:

The light converges to form an inverted, real image on the retina, the light-sensitive layer at the back of the eye.

Optic Nerve:

The retina converts this light into electrical signals, which are then transmitted to the brain via the optic nerve.

Perception:

The brain processes these signals to form the upright, clear image we perceive as vision.

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Retina

The **retina** (from Latin *rete* 'net'; pl. **retinae** or **retinas**) is the innermost, light-sensitive layer of tissue of the eye of most vertebrates and some molluscs. The optics of the eye create a focused two-dimensional image of the visual world on the retina, which then processes that image within the retina and sends nerve impulses along the optic nerve to the visual cortex to create visual perception. The retina serves a function which is in many ways analogous to that of the film or image sensor in a camera.

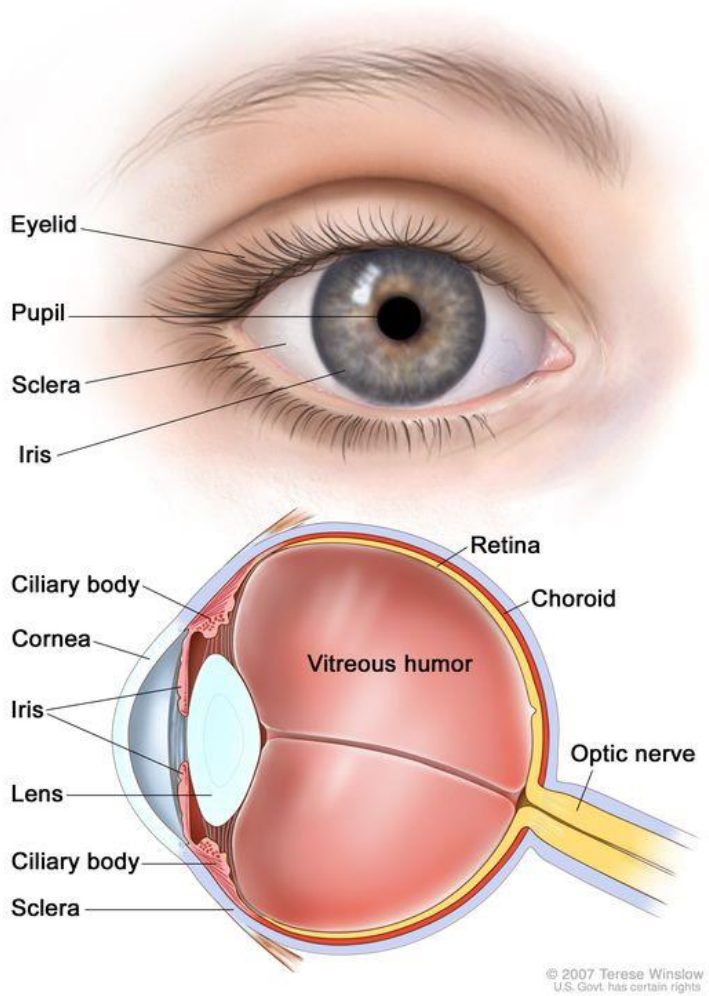
The neural retina consists of several layers of neurons interconnected by synapses and is supported by an outer layer of pigmented epithelial cells.

The primary light-sensing cells in the retina are the photoreceptor cells, which are of two types: rods and cones.

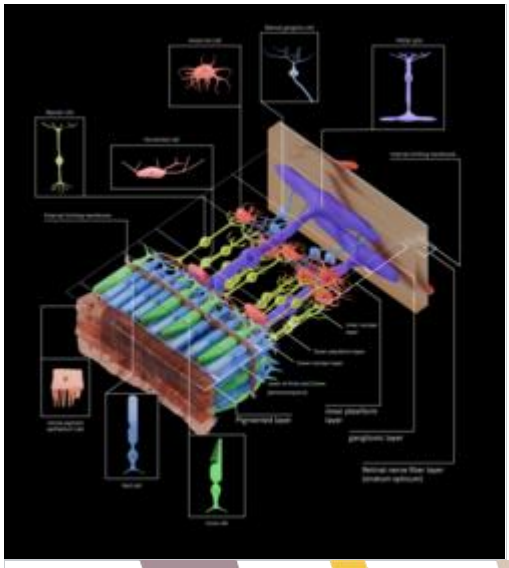
Rods function mainly in dim light and provide monochromatic vision.

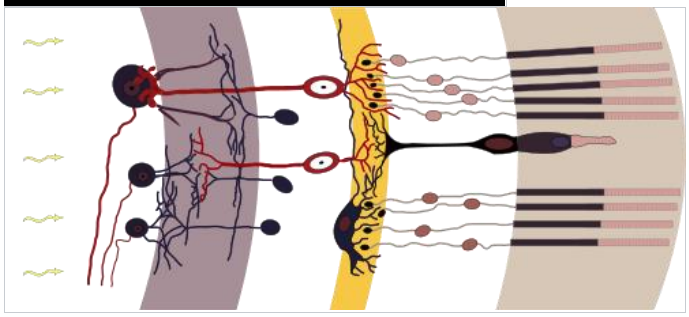
Cones function in well-lit conditions and are responsible for the perception of colour through the use of a range of opsins, as well as high-acuity vision used for tasks such as reading.

A third type of light-sensing cell, the photosensitive ganglion cell, is important for entrainment of circadian rhythms and reflexive responses such as the pupillary light reflex.



Retinal layers





an orthographic cross-section of the layers of the human retina labeling various elements

Rods, cones, and nerve layers in the retina:

The front (anterior) of the eye is on the left.

Light (from the left) passes through several transparent nerve layers to reach the rods and cones (far right).

Chemical changes in the rods and cones send a signal back to the nerves.

The signal goes first to the bipolar and horizontal cells (yellow layer), then to the amacrine cells and ganglion cells (purple layer), then to the optic nerve fibres. The signals are processed in these layers.

First, the signals start as raw outputs of points in the rod and cone cells.

Then, the nerve layers identify simple shapes, such as bright points surrounded by dark points, edges, and movement. (Based on a drawing by Ramón y Cajal, 1911)

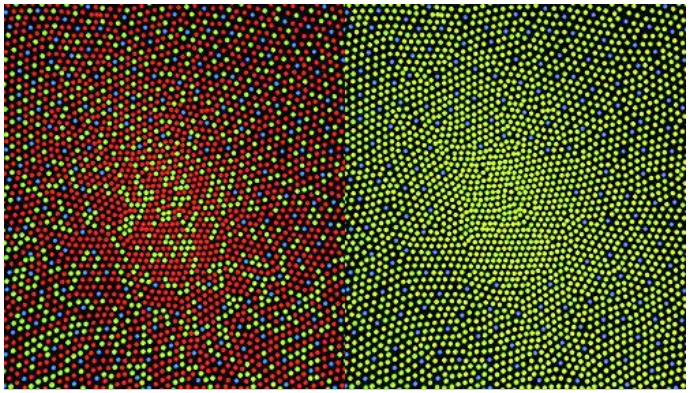
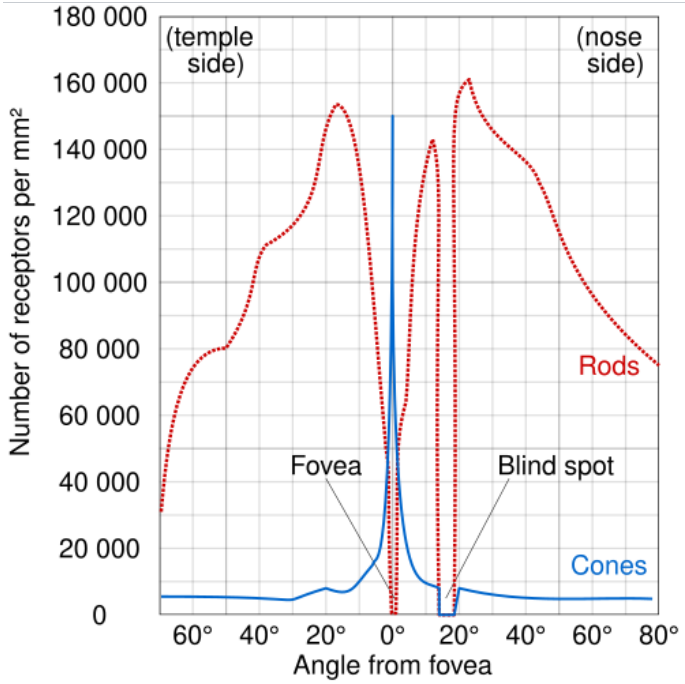


Illustration of the distribution of cone cells in the fovea of an individual with normal colour vision (left), and a colourblind (protanopic) retina. The center of the fovea holds very few blue-sensitive cones.



Distribution of rods and cones along a line passing through the fovea and the blind spot of a human eye

The vertebrate retina has 10 distinct layers.

From closest to farthest from the vitreous body:

1. Inner limiting membrane – basement membrane elaborated by Müller cells
2. Nerve fiber layer – axons of the ganglion cell bodies (a thin layer of Müller cell footplates exists between this layer and the inner limiting membrane)

3. Ganglion cell layer – contains nuclei of ganglion cells, the axons of which become the optic nerve fibres, and some displaced amacrine cells
4. Inner plexiform layer – contains the synapse between the bipolar cell axons and the dendrites of the ganglion and amacrine cells
5. Inner nuclear layer – contains the nuclei and surrounding cell bodies (perikarya) of the amacrine cells, bipolar cells, and horizontal cells
6. Outer plexiform layer – projections of rods and cones ending in the rod spherule and cone pedicle, respectively, these make synapses with dendrites of bipolar cells and horizontal cells.[2] In the macular region, this is known as the f*iber layer of Henle*.
7. Outer nuclear layer – cell bodies of rods and cones
8. External limiting membrane – layer that separates the inner segment portions of the photoreceptors from their cell nuclei
9. Inner segment / outer segment layer – inner segments and outer segments of rods and cones, the outer segments contain a highly specialized light-sensing apparatus.
10. Retinal pigment epithelium – single layer of cuboidal epithelial cells (with extrusions not shown in diagram). This layer is closest to the choroid, and provides nourishment and supportive functions to the neural retina, The black pigment melanin in the pigment layer prevents light reflection throughout the globe of the eyeball; this is extremely important for clear vision.

These layers can be grouped into four main processing stages—photoreception; transmission to bipolar cells; transmission to ganglion cells, which also contain photoreceptors, the photosensitive ganglion cells; and transmission along the optic nerve. At each synaptic stage, horizontal and amacrine cells also are laterally connected.

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Photochemistry of Vision

Both rods and cones contain chemicals that decom-pose on exposure to light and, in the process, excite the nerve fibers leading from the eye. The light-sensitive chemical in the *rods* is called *rhodopsin;* the light-sensitive chemicals in the *cones,* called *cone pigments* or *color pigments,* have compositions only slightly dif-ferent from that of rhodopsin.

In this section, we discuss principally the photo-chemistry of rhodopsin, but the same principles can be applied to the cone pigments.

Rhodopsin-Retinal Visual Cycle, and Excitation of the Rods

Rhodopsin and Its Decomposition by Light Energy. The outersegment of the rod that projects into the pigment layer of the retina has a concentration of about 40 per cent of the light-sensitive pigment called *rhodopsin,* or *visual purple*. This substance is a combination of theprotein *scotopsin* and the carotenoid pigment *retinal* (also called "retinene"). Furthermore, the retinal is a particular type called 11-*cis* retinal.

This *cis* form of retinal is important because only this form can bind with scotopsin to synthesize rhodopsin.

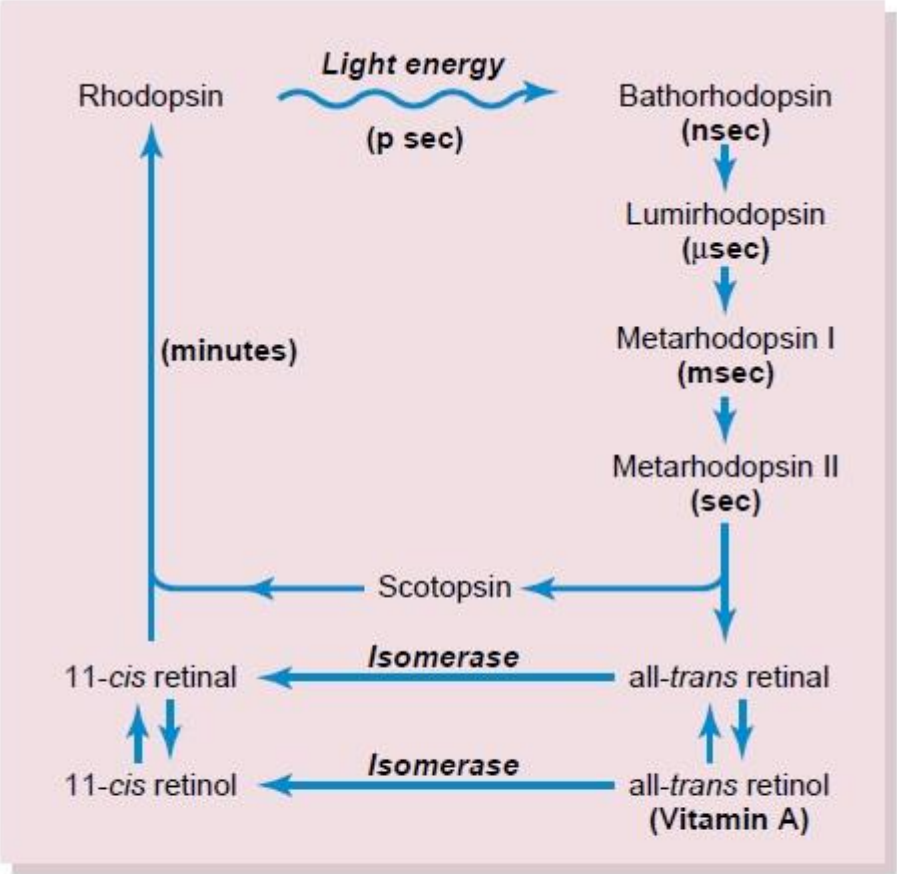

Figure 50–5

Rhodopsin-retinal visual cycle in the rod, showing decomposition of rhodopsin during exposure to light and subsequent slow re-formation of rhodopsin by the chemical processes.

When light energy is absorbed by rhodopsin, the rhodopsin begins to decompose within a very small fraction of a second, as shown at the top of Figure 50–5. The cause of this is photoactivation of electrons in the retinal portion of the rhodopsin, which leads to instantaneous change of the *cis* form of retinal into an all-*trans* form that still has the same chemical structure as the *cis* form but has a different physical structure— a straight molecule rather than an angulated molecule. Because the three-dimensional orientation of the reactive sites of the all-*trans* retinal no longer fits with the orientation of the reactive sites on the protein *scotopsin*, the all-*trans* retinal begins to pullaway from the scotopsin. The immediate product is *bathorhodopsin*, which is a partially split combinationof the all-*trans* retinal and scotopsin. Bathorhodopsin is extremely unstable and decays in nanoseconds to *lumirhodopsin*. This then decays in microsecondsto *metarhodopsin I*, then in about a millisecond to *metarhodopsin II*,and finally, much more slowly (inseconds), into the completely split products *scotopsin* and all-*trans* retinal.

It is the metarhodopsin II, also called *activatedrhodopsin,* that excites electrical changes in the rods,and the rods then transmit the visual image into the central nervous system in the form of optic nerve action potential, as we discuss later.

Re-formation of Rhodopsin. The first stage in re-formationof rhodopsin, as shown in Figure 50–5, is to reconvert the all-*trans* retinal into 11-*cis* retinal. This process requires metabolic energy and is catalyzed by the enzyme *retinal isomerase.* Once the 11-*cis* retinal is formed, it automatically recombines with the scotopsin to re-form rhodopsin, which then remains stable until its decomposition is again triggered by absorption of light energy.

Role of Vitamin A for Formation of Rhodopsin. Note inFigure 50–5 that there is a second chemical route by which all-*trans* retinal can be converted into 11-*cis* retinal.This is by conversion of the all-*trans* retinal first into all-*trans* retinol, which is one form of vitamin A. Then the all-*trans* retinol is converted into 11-*cis* retinol under the influence of the enzyme isomerase. Finally, the 11-*cis* retinol is converted into 11-*cis* retinal, which combines with scotopsin to form new rhodopsin.

Vitamin A is present both in the cytoplasm of the rods and in the pigment layer of the retina. Therefore, vitamin A is normally always available to form new retinal when needed. Conversely, when there is excess retinal in the retina, it is converted back into vitamin A, thus reducing the amount of light-sensitive pigment in the retina. We shall see later that this interconver-sion between retinal and vitamin A is especially impor-tant in long-term adaptation of the retina to different light intensities.

Night Blindness. Night blindness occurs in any personwith severe vitamin A deficiency. The simple reason for this is that without vitamin A, the amounts of retinal and rhodopsin that can be formed are severely depressed. This condition is called *night blindness* because the amount of light available at night is too little to permit adequate vision in vitamin A–deficient persons.

For night blindness to occur, a person usually must remain on a vitamin A–deficient diet for months, because large quantities of vitamin A are normally stored in the liver and can be made available to the eyes. Once night blindness develops, it can sometimes be reversed in less than 1 hour by intravenous injection of vitamin A.

Excitation of the Rod When Rhodopsin Is Activated by Light

The Rod Receptor Potential Is Hyperpolarizing, Not Depolariz-ing. When the rod is exposed to light, the resulting receptor potential is different from the receptor poten-tials in almost all other sensory receptors. That is, exci-tation of the rod causes *increased negativity* of the intrarod membrane potential, which is a state of *hyper-polarization,* meaning that there is more negativitythan normal *inside* the rod membrane. This is exactly opposite to the decreased negativity (the process of "depolarization") that occurs in almost all other sensory receptors.

But how does activation of rhodopsin cause hyper-polarization? The answer is that *when rhodopsindecomposes, it decreases the rod membrane conduc-tance for sodium ions in the outer segment of the rod.*

This causes hyperpolarization of the entire rod mem-brane in the following way.

Figure 50–6 shows movement of sodium ions in a complete electrical circuit through the inner and outer segments of the rod. The inner segment continually pumps sodium from inside the rod to the outside, thereby creating a negative potential on the inside of the entire cell. However, the outer segment of the rod, where the photoreceptor discs are located, is entirely different; here, the rod membrane, in the *dark* state, is very leaky to sodium ions. Therefore, positively charged sodium ions continually leak back to the inside of the rod and thereby neutralize much of the negativity on the inside of the entire cell. Thus, *undernormal dark conditions, when the rod is not excited*

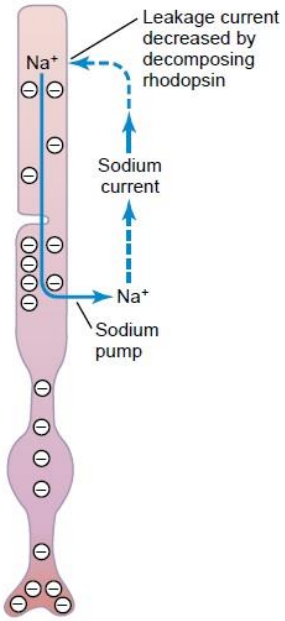

Figure 50–6

Theoretical basis for generation of a "hyperpolarization receptor potential" caused by rhodopsin decomposition, which *decreases the flow of positively charged sodium ions* into the outer segment of the rod.

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there is reduced electronegativity inside the membraneof the rod, measuring about –40 millivolts rather than the usual –70 to –80 millivolts found in most sensory receptors.

Then, when the rhodopsin in the outer segment of the rod is exposed to light, the rhodopsin begins to decompose, and this *decreases* the outer segment membrane conductance of sodium to the interior of the rod, even though sodium ions continue to be pumped outward through the membrane of the inner segment. Thus, more sodium ions now leave the rod than leak back in. Because they are positive ions, their loss from inside the rod creates increased negativity inside the membrane, and the greater the amount of light energy striking the rod, the greater the elec-tronegativity becomes—that is, the greater is the degree of *hyperpolarization.* At maximum light inten-sity, the membrane potential approaches –70 to –80 millivolts, which is near the equilibrium potential for potassium ions across the membrane.

Duration of the Receptor Potential, and Logarithmic Relation of the Receptor Potential to Light Intensity. When a sudden pulse of light strikes the retina, the transient hyperpolarization that occurs in the rods— that is, the *receptor potential* that

occurs—reaches a peak in about 0.3 second and lasts for more than a second. In cones, the change occurs four times as fast as in the rods. A visual image impinged on the rods of the retina for only one millionth of a second can sometimes cause the sensation of seeing the image for longer than a second.

Another characteristic of the receptor potential is that it is approximately proportional to the logarithm of the light intensity. This is exceedingly important, because it allows the eye to discriminate light intensi-ties through a range many thousand times as great as would be possible otherwise.

Mechanism by Which Rhodopsin Decomposition *Decreases* Membrane Sodium Conductance—TheExcitation "Cascade." Under optimal conditions, a single photon of light, the smallest possible quantal unit of light energy, can cause a measurable receptor potential in a rod of about 1 millivolt. Only 30 photons of light will cause half saturation of the rod. How can such a small amount of light cause such great excitation? The answer is that the photoreceptors have an extremely sensitive chemical cascade that amplifies the stimulatory effects about a millionfold, as follows:

1.The *photon activates an electron* in the 11-*cis* retinal portion of the rhodopsin; this leads to the formation of *metarhodopsin II*, which is the active form of rhodopsin, as already discussed and shown in Figure 50–5.

2.The *activated rhodopsin* functions as an enzyme to activate many molecules of *transducin,* a protein present in an inactive form in the membranes of the discs and cell membrane of the rod.

3.The *activated transducin* activates many more molecules of *phosphodiesterase.*

4.Activated phosphodiesterase is another enzyme;it immediately hydrolyzes many molecules of *cyclic guanosine monophosphate (cGMP),* thusdestroying it. Before being destroyed, the cGMP had been bound with the sodium channel protein of the rod's outer membrane in a way that "splints" it in the open state. But in light, when phosphodiesterase hydrolyzes the cGMP, this removes the splinting and allows the sodium channels to close. Several hundred channels close for each originally activated molecule of rhodopsin. Because the sodium flux through each of these channels has been extremely rapid, flow of more than a million sodium ions is blocked by the channel closure before the channel opens again. This diminution of sodium ion flow is what excites the rod, as already discussed.

5.Within about a second, another enzyme, *rhodopsin kinase,* which is always present inthe rod, inactivates the activated rhodopsin (the metarhodopsin II), and the entire cascade reverses back to the normal state with open sodium channels.

Thus, the rods have developed an important chemi-cal cascade that amplifies the effect of a single photon of light to cause movement of millions of sodium ions. This explains the extreme sensitivity of the rods under dark conditions.

The cones are about 30 to 300 times less sensitive than the rods, but even this allows color vision at any intensity of light greater than extremely dim twilight.

Photochemistry of Color Vision by the Cones

It was pointed out at the outset of this discussion that the photochemicals in the cones have almost exactly the same chemical composition as that of rhodopsin in the rods. The only difference is that the protein por-tions, or the opsins—called *photopsins* in the cones— are slightly different from the scotopsin of the rods. The *retinal* portion of all the visual pigments is exactly the same in the cones as in the rods. The color-sensitive pigments of the cones, therefore, are combi-nations of retinal and photopsins.

In the discussion of color vision later, it will become evident that only one of three types of color pigments is present in each of the different cones, thus making the cones selectively sensitive to different colors: blue, green, or red. These color pigments are called, respectively, *blue-sensitive pigment, green-sensitive pigment,* and *red-sensitive pigment.* Theabsorption characteristics of the pigments in the three types of cones show peak absorbencies at light wave-lengths of 445, 535, and 570 nanometers, respectively.

These are also the wavelengths for peak light sensitiv-ity for each type of cone, which begins to explain how the retina differentiates the colors. The approximate absorption curves for these three pigments are shown in Figure 50–7. Also shown is the absorption curve for the rhodopsin of the rods, with a peak at 505 nanometers.

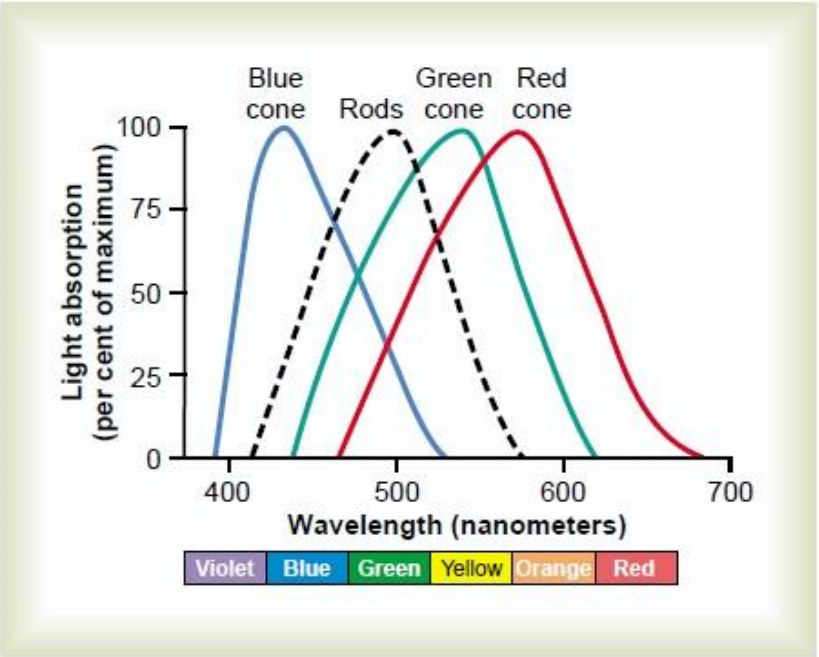

Figure 50–7

Light absorption by the pigment of the rods and by the pigments of the three color-receptive cones of the human retina. (Drawn from curves recorded by Marks WB, Dobelle WH, MacNichol EF Jr: Visual pigments of single primate cones. Science 143:1181, 1964, and by Brown PK, Wald G: Visual pigments in single rods and cones of the human retina: direct measurements reveal mechanisms of human night and color vision. Science 144:45, 1964. ® 1964 by the American Association for the Advancement of Science.)

Automatic Regulation of Retinal Sensitivity—Light and Dark Adaptation

Light and Dark Adaptation. If a person has been in brightlight for hours, large portions of the photochemicals in both the rods and the cones will have been reduced to retinal and opsins. Furthermore, much of the retinal of both the rods and the cones will have been converted into vitamin A. Because of these two effects, the concentrations of the photosensitive chemicals remaining in the rods and cones are considerably reduced, and the sensitivity of the eye to light is correspondingly reduced. This is called *light adaptation.*

Conversely, if a person remains in darkness for a long time, the retinal and opsins in the rods and cones are converted back into the light-sensitive pigments. Furthermore, vitamin A is converted back into retinal to give still more light-sensitive pigments, the final limit being determined by the amount of opsins in the rods and cones to combine with the retinal. This is called *dark adaptation.*

Figure 50–8 shows the course of dark adaptation when a person is exposed to total darkness after having been exposed to bright light for several hours.

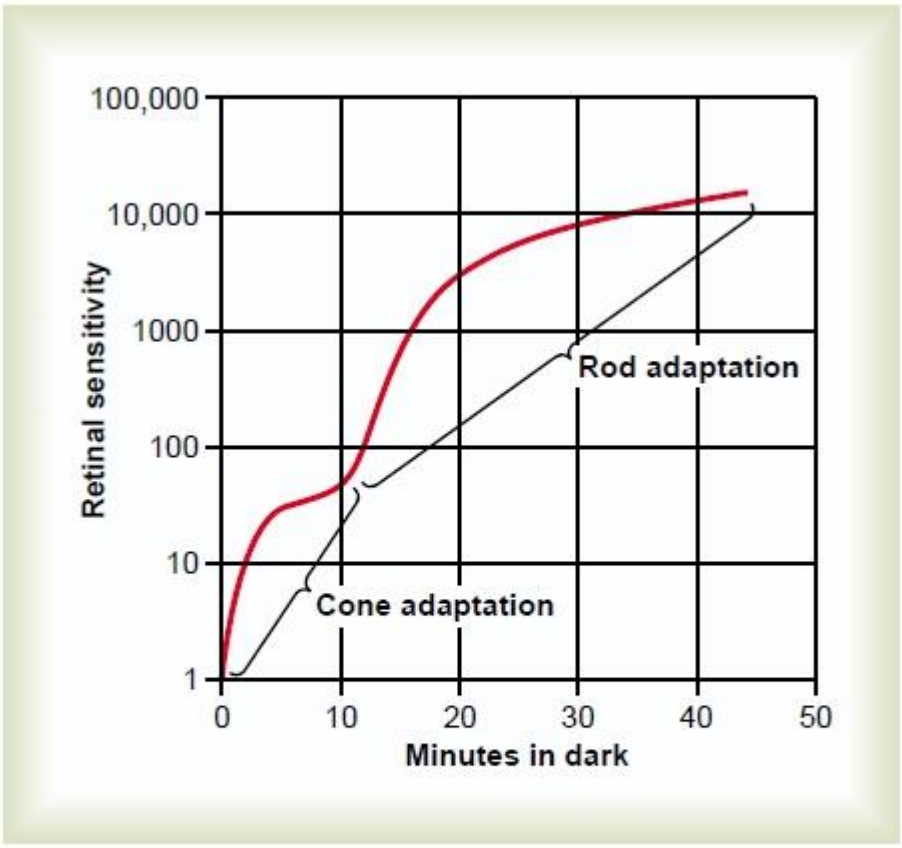

Figure 50–8

Dark adaptation, demonstrating the relation of cone adaptation to rod adaptation.

Note that the sensitivity of the retina is very low on first entering the darkness, but within 1 minute, the sensitivity has already increased 10-fold—that is, the retina can respond to light of one tenth the previously required intensity. At the end of 20 minutes, the sensi-tivity has increased about 6000-fold, and at the end of 40 minutes, about 25,000-fold.

The resulting curve of Figure 50–8 is called the *darkadaptation curve.* Note, however, the inflection in thecurve.The early portion of the curve is caused by adap-tation of the cones, because all the chemical events of vision, including adaptation, occur about four times as rapidly in cones as in rods. However, the cones do not achieve anywhere near the same degree of sensitivity change in darkness as the rods do. Therefore, despite rapid adaptation, the cones cease adapting after only a few minutes, while the slowly adapting rods continue to adapt for many minutes and even hours, their sen-sitivity increasing tremendously. In addition, still more sensitivity of the rods is caused by neuronal signal con-vergence of 100 or more rods onto a single ganglion cell in the retina; these rods summate to increase their sensitivity.

Other Mechanisms of Light and Dark Adaptation. In additionto adaptation caused by changes in concentrations of rhodopsin or color photochemicals, the eye has two

other mechanisms for light and dark adaptation. The first of these is a *change in pupillary size*. This can cause adaptation of approxi-mately 30-fold within a fraction of a second, because of changes in the amount of light allowed through the pupillary opening.

The other mechanism is *neural adaptation*, involving the neurons in the successive stages of the visual chain in the retina itself and in the brain. That is, when light intensity first increases, the signals transmitted by the bipolar cells, horizontal cells, amacrine cells, and gan-glion cells are all intense. However, most of these signals decrease rapidly at different stages of transmission in the neural circuit. Although the degree of adaptation is only a fewfold rather than the many thousandfold that occurs during adaptation of the photochemical system, neural adaptation occurs in a fraction of a second, in contrast to the many minutes to hours required for full adaptation by the photochemicals.

Value of Light and Dark Adaptation in Vision. Between thelimits of maximal dark adaptation and maximal light adaptation, the eye can change its sensitivity to light as much as 500,000 to 1 million times, the sensitivity automatically adjusting to changes in illumination.

Because registration of images by the retina requires detection of both dark and light spots in the image, it is essential that the sensitivity of the retina always be adjusted so that the receptors respond to the lighter areas but not to the darker areas. An example of maladjustment of retinal adaptation occurs when a person leaves a movie theater and enters the bright sunlight. Then, even the dark spots in the images seem exceedingly bright, and as a consequence, the entire visual image is bleached, having little contrast among its different parts. This is poor vision, and it remains poor until the retina has adapted sufficiently so that the darker areas of the image no longer stimulate the receptors excessively.

Conversely, when a person first enters darkness, the sensitivity of the retina is usually so slight that even the light spots in the image cannot excite the retina. After dark adaptation, the light spots begin to regis-ter. As an example of the extremes of light and dark adaptation, the intensity of sunlight is about 10 billion times that of starlight, yet the eye can function both in bright sunlight after light adaptation and in starlight after dark adaptation.