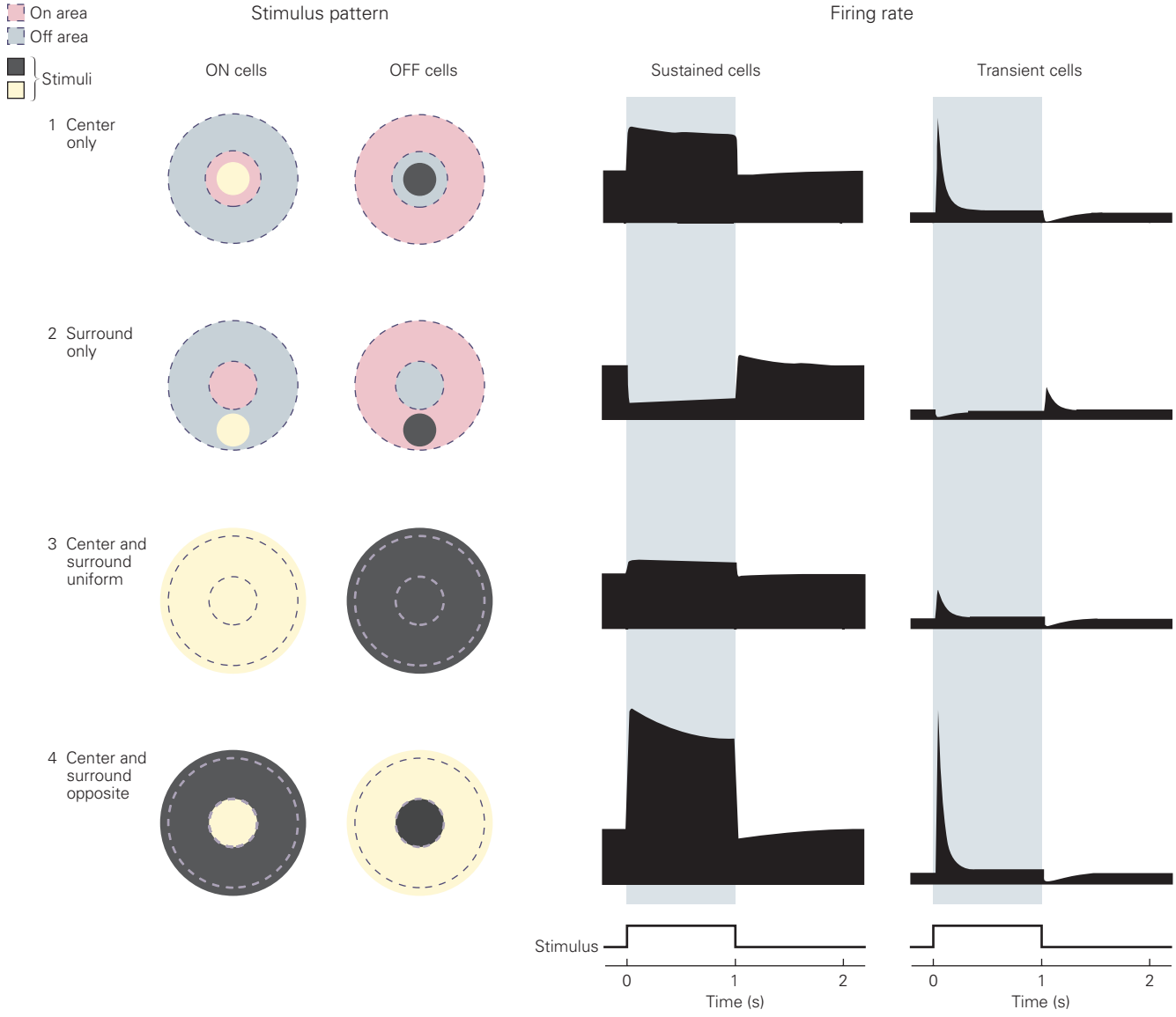

Figure 22–10 **Responses of retinal ganglion cells with center-surround receptive fields.** In these idealized experiments, the stimulus changes from a uniform gray field to the pattern of bright (**yellow**) and dark (**black**) regions indicated on the *left*. This leads to the firing rate responses shown on the *right*. **1.** ON cells are excited by a bright spot in the receptive field center, OFF cells by a dark spot. In *sustained cells*, the excitation persists throughout stimulation, whereas in *transient cells*, a brief burst of spikes occurs just after the onset of stimulation. **2.** If the same stimulus that excites the center is applied to the surround, firing is suppressed. **3.** Uniform stimulation of both center and surround elicits a response like that of the center, but much smaller in amplitude. **4.** Stimulation of the center combined with the opposite stimulus in the surround produces the strongest response.

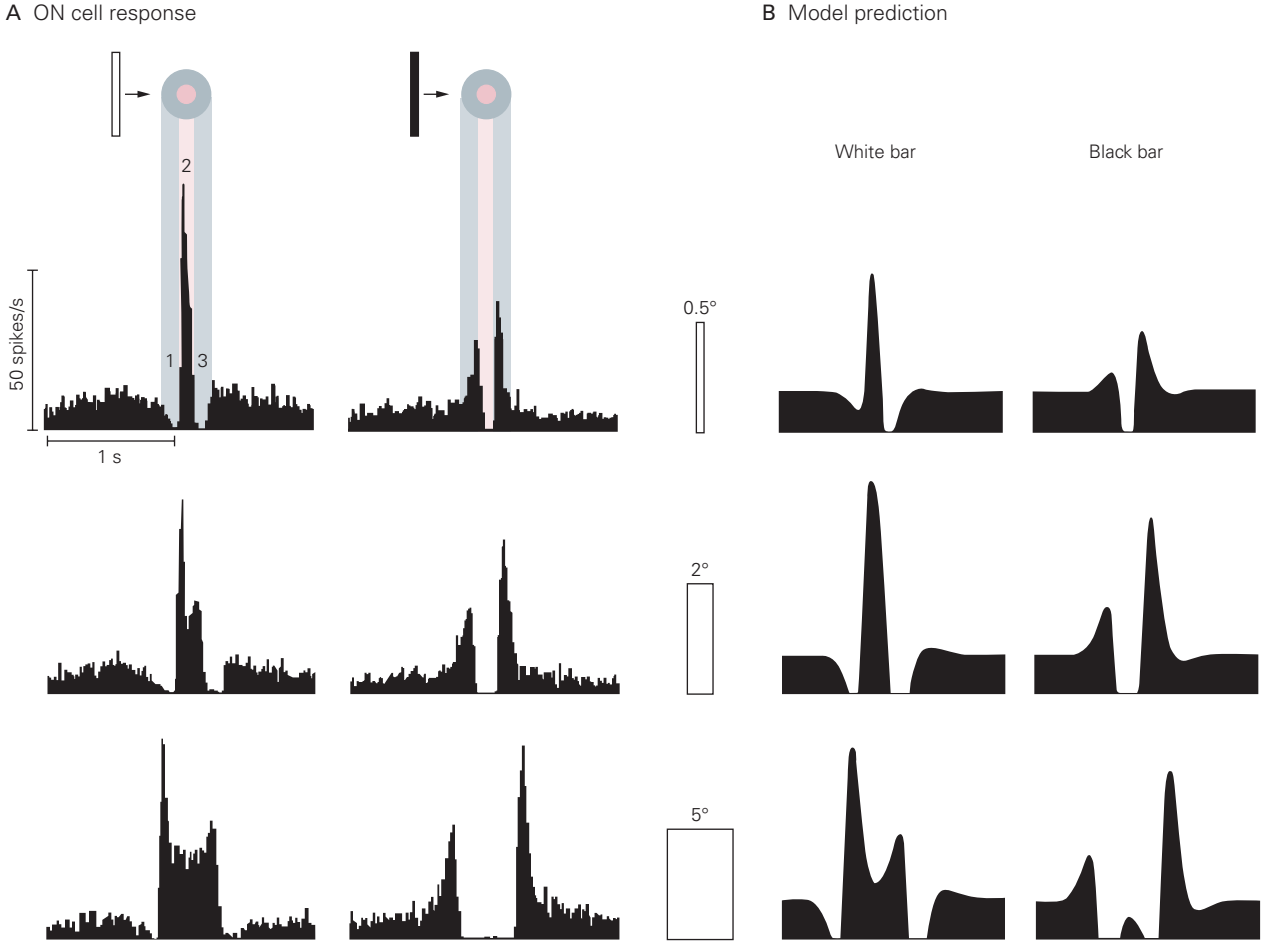

Figure 22–11 Responses of ganglion cells in the cat retina to moving objects.

A. The firing rate of an ON ganglion cell in response to a variety of bars (white or black, various widths) moving across the retina. Each bar moves at 10° per second; 1° corresponds to 180 μm on the retina. In response to the white bar, the firing rate first decreases as the bar passes over the receptive-field surround (**1**), increases as the bar enters the center (**2**), and decreases again as the bar passes through the surround on the opposite side (**3**). The dark bar elicits responses of the opposite sign. Because ganglion cells similar to this one are distributed throughout the retina, one can also interpret this curve as an instantaneous snapshot of activity in a population of ganglion cells, where the horizontal axis represents location on the retina. In effect, this activity profile is the neural representation of the moving bar transmitted to the brain. A complementary population of OFF ganglion cells (not shown here) conveys another neural activity profile in parallel. In this way, both bright and dark edges can be signaled by a sharp increase in firing.

B. A simple model of retinal processing that incorporates center-surround antagonism and a transient temporal filter is used to predict ganglion-cell firing rates. The predictions match the essential features of the responses in part **A**. (Reproduced, with permission, from Rodieck 1965. Copyright © 1965 Elsevier Ltd.)

Box 22–1 Spatiotemporal Sensitivity of Human Perception

Although small spots of light are useful for probing the receptive fields of single neurons in visual pathways, different stimuli are needed to learn about human visual perception. Grating stimuli are commonly used to probe how our visual system deals with spatial and temporal patterns.

The subject views a display in which the intensity varies about the mean as a sinusoidal function of space (Figure 22–12). Then the contrast of the display—defined as the peak-to-peak amplitude of the sinusoid divided by the mean—is reduced to a threshold at which the grating is barely visible. This measurement is repeated for gratings of different spatial frequencies.

When the inverse of this threshold is plotted against the spatial frequency, the resulting *contrast sensitivity curve* provides a measure of sensitivity of visual perception to patterns of different scales (Figure 22–13A). When measured at high light intensity, sensitivity declines sharply at high spatial frequencies, with an absolute threshold at approximately 50 cycles per degree. This sensitivity is limited fundamentally by the quality of the optical image and the spacing of cone cells in the fovea (see Figure 22–1C).

Interestingly, sensitivity also declines at low spatial frequencies. Patterns with a frequency of approximately 5 cycles per degree are most visible. The visual system is said to have *band-pass* behavior because it rejects all but a band of spatial frequencies.

One can use the same techniques to measure the sensitivity of individual retinal ganglion cells in primates. The results resemble those for human subjects (Figure 22–13), suggesting that these basic features of visual perception are determined by the retina.

The band-pass behavior can be understood on the basis of spatial antagonism in center-surround receptive fields. A very fine grating presents many dark and bright stripes within the receptive-field center; their effects cancel one another and thus provide no net excitation. A very coarse grating presents a single stripe to both the center and surround of the receptive field, and their antagonism again provides the ganglion cell little net excitation. The strongest response is produced by a grating of intermediate spatial frequency that just covers the center with one stripe and most of the surround with stripes of the opposite polarity (Figure 22–13B).

In dim light, the visual system's contrast sensitivity declines, but more so at high than at low spatial frequencies (Figure 22–13A). Thus, the peak sensitivity shifts to lower spatial frequencies, and eventually the curve loses its peak altogether. In this state, the visual system has so-called *low-pass* behavior, for it preferentially encodes stimuli of low spatial frequency. The fact that in dim light the receptive fields of ganglion cells lose their antagonistic surrounds explains the transition from band-pass to low-pass spatial filtering (Figure 22–13B).

Similar experiments can be done to test visual sensitivity to temporal patterns. Here, the intensity of a test stimulus flickers sinusoidally in time, while the contrast is gradually brought to the threshold level of detection. For humans, contrast sensitivity declines sharply at very high flicker frequencies, but declines also at very low frequencies (Figure 22–14A). Flicker at approximately 10 Hz is the most effective stimulus. One finds similar band-pass behavior in the flicker sensitivity of macaque retinal ganglion cells (Figure 22–14B).

Sensitivity to temporal contrast also depends on the mean light level. For human subjects, the optimum flicker frequency shifts downward at lower stimulus intensities and the peak in the curve becomes less and less prominent (Figure 22–14). The fact that primate retinal ganglion cells duplicate this behavior suggests that retinal processing limits the performance of the entire visual system in these simple tasks.

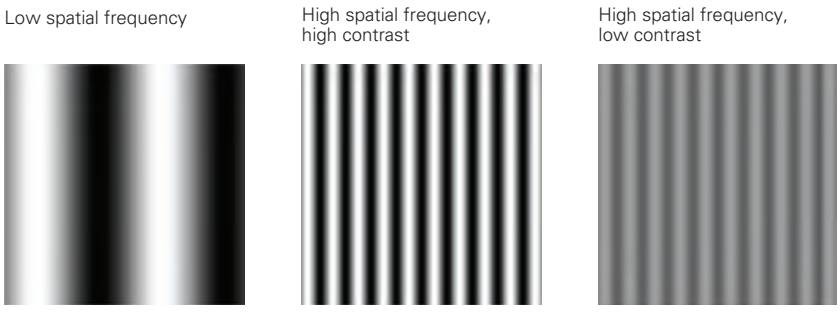

Figure 22–12 Sinusoid grating displays used in psychophysical experiments with human subjects. Such stimuli were used in the experiments discussed in Figure 22–13.

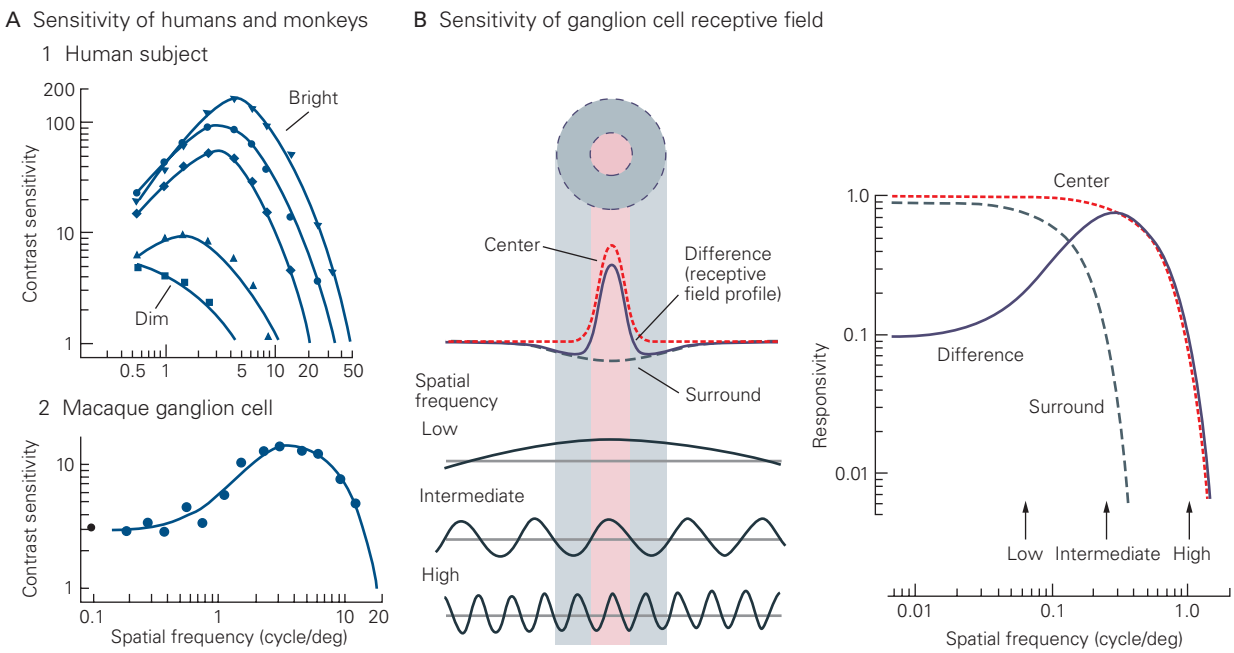

Figure 22–13 Spatial contrast sensitivity.

A. 1. The contrast sensitivity of human subjects was measured using gratings with different spatial frequencies (see Figure 22–12). At each frequency, the contrast was increased to the threshold for detection, and the inverse of that contrast value was plotted against spatial frequency, as shown here. The curves were obtained at different mean intensities, decreasing by factors of 10 from the top to the bottom curve. (Reproduced, with permission, from De Valois, Morgan, and Snodderly 1974.) 2. Contrast sensitivity of a P-type ganglion cell in the macaque retina measured at high intensity. At each spatial frequency, the contrast was gradually increased until it produced a detectable change in the neuron's firing rate. The inverse of that threshold contrast was plotted as in part A-1. The isolated dot at left marks the sensitivity at zero spatial frequency, a spatially uniform field. (Reproduced, with permission, from Derrington and Lennie 1984.)

B. Stimulation of a center-surround receptive field with sinusoid gratings. The neuron's sensitivity to light at different points on the retina is modeled as a "difference-of-Gaussians" receptive field, with a narrow positive Gaussian for the excitatory center and a broad negative Gaussian for the inhibitory surround. Multiplying the profile of the grating stimulus (intensity vs position) with the profile of the receptive field (sensitivity vs position) and integrating over all space calculates the stimulus strength delivered by a particular grating. The resulting sensitivity of the receptive field to gratings of different frequency is shown in the plot on the right. At low spatial frequencies, the negative contribution from the surround cancels the contribution from the center, leading to a drop in the difference curve. (Reproduced, with permission, from Enroth-Cugell and Robson 1984.)

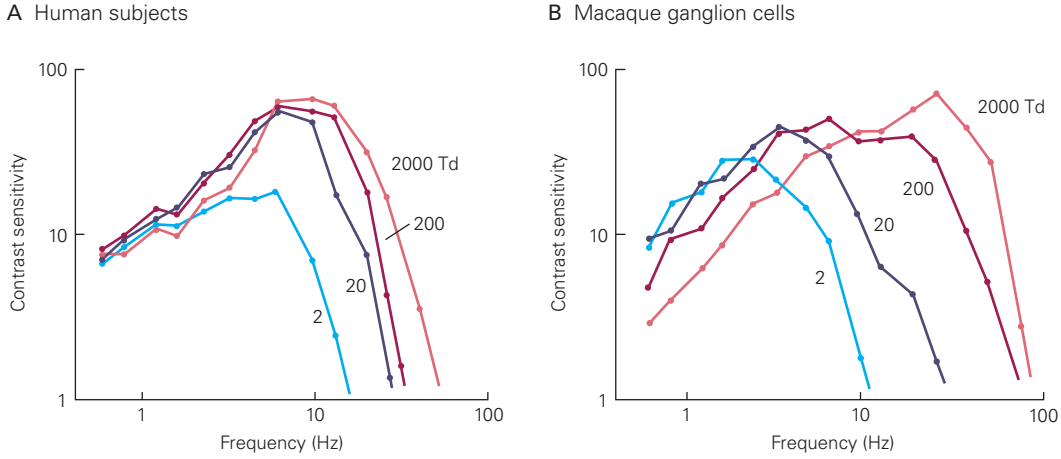

Figure 22–14 Temporal contrast sensitivity. (Reproduced, with permission, from Lee et al. 1990.)

A. The sensitivity of human subjects to temporal flicker was measured by methods similar to those in Figure 22–13A, but the stimulus was a large spot whose intensity varied sinusoidally in time rather than in space. The inverse of the threshold contrast required for detection is plotted against the frequency of the sinusoidal flicker. Sensitivity declines at both high and low frequencies. The mean light level varied, decreasing by factors of 10 from the top to the bottom trace.

B. The flicker sensitivity of M-type ganglion cells in the macaque retina was measured by the same method applied to human subjects in part A. The detection threshold for the neural response was defined as a variation of 20 spikes per second in the cell's firing rate in phase with the flicker.

(sustained or transient), spectral filtering (broadband or dominated by red, green, or blue), and selectivity for other image features such as motion.

These neural representations are directed to various visual centers in the brain, including the lateral geniculate nucleus of the thalamus, a relay to the visual cortex; the superior colliculus, a midbrain region involved in spatial attention and orienting movements; the pretectum, involved in control of the pupil; the accessory optic system, which analyzes self-motion to stabilize gaze; and the suprachiasmatic nucleus, a central clock that directs circadian rhythm and whose phase can be set by light cues (Chapter 44). In many cases, the axons of one type of ganglion cell extend collaterals to multiple areas of the central nervous system. M-cells, for example, project to the thalamus and the superior colliculus.

A Network of Interneurons Shapes the Retinal Output

We now consider in more detail the retinal circuit and how it accounts for the intricate response properties of retinal ganglion cells.

Parallel Pathways Originate in Bipolar Cells

The photoreceptor forms synapses with bipolar cells and horizontal cells (see Figure 22–3A). In the dark, the photoreceptor's synaptic terminal releases glutamate continuously. When stimulated by light, the photoreceptor hyperpolarizes, less calcium enters the terminal, and the terminal releases less glutamate. Photoreceptors do not fire action potentials; like bipolar cells, they release neurotransmitter in a graded fashion using a specialized structure, the *ribbon synapse.* In fact, most retinal processing is accomplished with graded membrane potentials: Action potentials occur primarily in certain amacrine cells and in the retinal ganglion cells.

The two principal varieties of bipolar cells, ON and OFF cells, respond to glutamate at the synapse through distinct mechanisms. The OFF cells use ionotropic receptors, namely glutamate-gated cation channels of the AMPA-kainate variety (AMPA = α-amino-3-hydroxy-5-methylisoxazole-4-propionate). The glutamate released in darkness depolarizes these cells. The ON cells use metabotropic receptors that are linked to a G protein whose action ultimately closes cation channels. Glutamate activation of these receptors thus hyperpolarizes the cells in the dark.

Bipolar ON and OFF cells differ in shape and especially in the levels within the inner plexiform layer where their axons terminate. The axons of ON cells end in the proximal (lower) half, while those of OFF cells end in the distal (upper) half (Figure 22–15). There, they form specific synaptic connections on the dendrites of amacrine and ganglion cells. The ON bipolar cells excite ON ganglion cells, while OFF bipolar cells excite OFF ganglion cells (see Figure 22–3A). Thus, the two principal subdivisions of retinal output, the ON and OFF pathways, are already established at the level of bipolar cells.

Bipolar cells can also be distinguished by the morphology of their dendrites (Figure 22–15). In the central region of the primate retina, the *midget bipolar cell* receives input from a single cone and excites a P-type ganglion cell. This explains why the centers of P-cell receptive fields are so small. The *diffuse bipolar cell* receives input from many cones and excites an M-type ganglion cell. Accordingly, the receptive-field centers of M-cells are much larger. Thus, stimulus representations in the ganglion cell population originate in dedicated bipolar cell pathways that are differentiated by their selective connections to photoreceptors and postsynaptic targets.

Spatial Filtering Is Accomplished by Lateral Inhibition

Signals in the parallel on and off pathways are modified by interactions with horizontal and amacrine cells (see Figure 22–3A). Horizontal cells have broadly arborizing dendrites that spread laterally in the outer plexiform layer. Photoreceptors contact the tips of these arbors at glutamatergic terminals shared with bipolar cells. In addition, horizontal cells are electrically coupled to each other through gap junctions.

A horizontal cell effectively measures the average level of excitation of the photoreceptor population over a broad region. This signal is fed back to the photoreceptor terminal through an inhibitory synapse. Thus, the photoreceptor terminal is under two opposing influences: light falling on the receptor hyperpolarizes it, but light falling on the surrounding region depolarizes it through the sign-inverting synapses from horizontal cells. As a result, the bipolar cell has an antagonistic receptive-field structure.

This spatial antagonism in the receptive field is enhanced by lateral inhibition from amacrine cells in the inner retina. Amacrine cells are neurons whose processes ramify only in the inner plexiform layer. Approximately 30 types of amacrine cells are known, some with small arbors only tens of micrometers across, and others with processes that extend across the entire retina. Amacrine cells generally receive

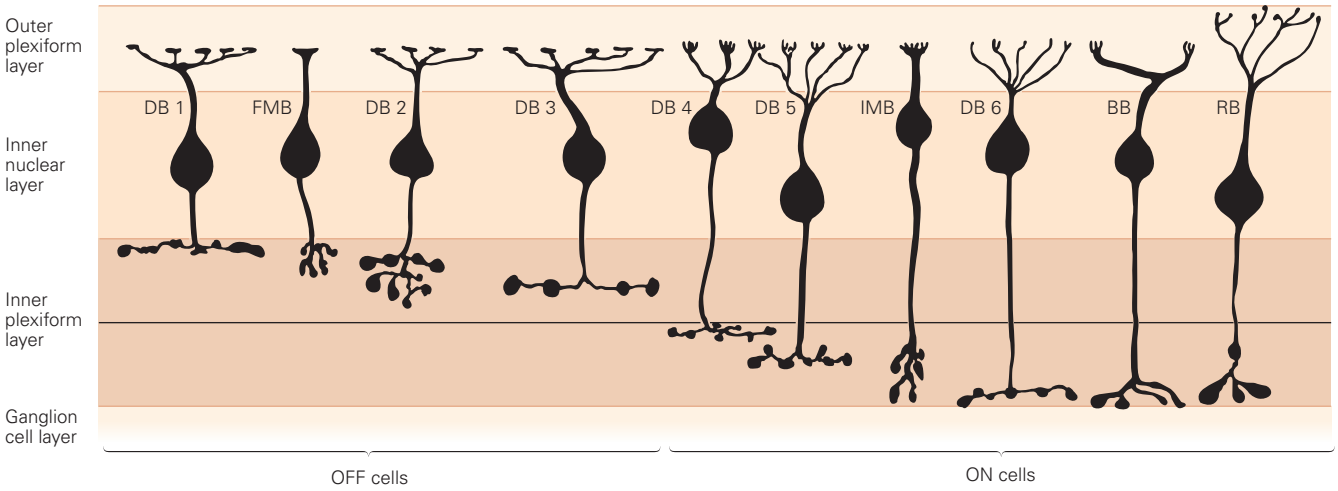

Figure 22–15 Bipolar cells in the macaque retina. The cells are arranged according to the depth of their terminal arbors in the inner plexiform layer. The horizontal line dividing the distal (**upper**) and proximal (**lower**) levels of this layer represents the border between the axonal terminals of OFF and ON type cells. Terminals in the upper half are presumed to be those of OFF cells, and those in the lower half ON cells. Cell types are diffuse bipolar cells (**DB**), ON and OFF midget bipolars (**IMB, FMB**), S-cone ON bipolar (**BB**), and rod bipolar (**RB**). (Reproduced, with permission, from Boycott and Wässle 1999.)

excitatory signals from bipolar cells at glutamatergic synapses. Some amacrine cells feed back directly to the presynaptic bipolar cell at a *reciprocal inhibitory synapse.* Some amacrine cells are electrically coupled to others of the same type, forming an electrical network much like that of the horizontal cells.

Through this inhibitory network, a bipolar cell terminal can receive inhibition from distant bipolar cells, in a manner closely analogous to the lateral inhibition of photoreceptor terminals (see Figure 22–3A). Amacrine cells also inhibit retinal ganglion cells directly. These lateral inhibitory connections contribute substantially to the antagonistic receptive field component of retinal ganglion cells.

Temporal Filtering Occurs in Synapses and Feedback Circuits

For many ganglion cells, a step change in light intensity produces a transient response, an initial peak in firing that declines to a smaller steady rate (see Figure 22–10). Part of this sensitivity originates in the negative-feedback circuits involving horizontal and amacrine cells. For example, a sudden decrease in light intensity depolarizes the cone terminal, which excites the horizontal cell, which in turn repolarizes the cone terminal (see Figure 22–3A). Because this feedback loop involves a brief delay, the voltage response of the cone peaks abruptly and then settles to a smaller steady level. Similar processing occurs at the reciprocal synapses between bipolar and amacrine cells in the inner retina.

In both cases, the delayed-inhibition circuit favors rapidly changing inputs over slowly changing inputs. The effects of this filtering, which can be observed in visual perception, are most pronounced for large stimuli that drive the horizontal and amacrine cell networks most effectively. For example, a large spot can be seen easily when it flickers at a rate of 10 Hz but not at a low rate (see Figure 22–14).

In addition to these circuit properties, certain cellular processes contribute to shaping the temporal response. For example, the AMPA-kainate type of glutamate receptor undergoes strong desensitization. A step increase in the concentration of glutamate at the dendrite of a bipolar or ganglion cell leads to an immediate opening of additional glutamate receptors. As these receptors desensitize, the postsynaptic conductance decreases again. The effect is to render a step response more transient.

Retinal circuits seem to go to great lengths to speed up their responses and emphasize temporal changes. One likely reason is that the very first cell in the retinal circuit, the photoreceptor, is exceptionally slow (see Figure 22–7C). Following a flash of light, a cone takes about 40 ms to reach the peak response, an intolerable delay for proper visual function. Through the various filtering mechanisms in retinal circuitry, subsequent neurons respond most vigorously during the rising phase of the cone's response. Indeed, some ganglion cells have a response peak only 20 ms after the flash. Temporal processing in the retina clearly helps to reduce visual reaction times, a life-extending trait as

important in highway traffic as on the savannas of our ancestors.

Color Vision Begins in Cone-Selective Circuits

Throughout recorded history, philosophers and scientists have been fascinated by color perception. This interest was originally driven by the relevance of color to art, later by its relation to the physical properties of light, and finally by commercial interests in television and photography. The 19th century witnessed a profusion of theories to explain color perception, of which two have survived modern scrutiny. They are based on careful psychophysics that placed strong constraints on the underlying neural mechanisms.

Early experiments demonstrated that any given natural light could be color-matched by mixing together appropriate amounts of three primary lights. This led to the trichromatic theory of color perception based on absorption of light by three mechanisms, each with a different sensitivity spectrum. These correspond to the three cone types (see Figure 22–6), whose measured absorption spectra fully explain the color-matching results both in normal individuals and those with genetic anomalies in the pigment genes.

The so-called opponent-process theory was proposed to explain our perception of different hues. According to this theory, color vision involves three processes that respond in opposite ways to light of different colors: (y–b) would be stimulated by yellow and inhibited by blue light; (r–g) stimulated by red and inhibited by green; and (w–bk) stimulated by white and inhibited by black. We recognize some of these 19th century postulates in the postreceptor circuitry of the retina.

In the central 10° of the human retina, a single midget bipolar cell that receives input from a single cone excites each P-type ganglion cell. An L-ON ganglion cell, for example, has a receptive field center consisting of a single L cone and an antagonistic surround involving a mixture of L and M cones. When this neuron's receptive field is stimulated with a large uniform spot of light that extends over both the center and the surround, this neuron is depolarized by red light and hyperpolarized by green light. Similar antagonism holds for the three other P-cells: L-OFF, M-ON, and M-OFF. These P-cells send their signals to the parvocellular layers of the lateral geniculate nucleus.

A dedicated type of S-ON bipolar cell collects the signals of S-cones selectively and transmits them to ganglion cells of the small bistratified type. Because this ganglion cell also receives excitation from L-OFF and M-OFF bipolar cells, it is depolarized by blue light and hyperpolarized by yellow light. Another type of ganglion cell shows the opposite signature: S-OFF and (L + M)-ON. These signals are transmitted to the koniocellular layers of the lateral geniculate nucleus.

The M-cells are excited by diffuse bipolar cells, which in turn collect inputs from many cones regardless of pigment type. These ganglion cells therefore have large receptive fields with broad spectral sensitivity. Their axons project to the magnocellular layers of the lateral geniculate nucleus.

In this way, chromatic signals are combined and encoded by the retina for transmission to the thalamus and cortex. In circuits of the primary visual cortex, these signals are recombined in different ways, leading to a great variety of receptive field layouts. Only about 10% of cortical neurons are preferentially driven by color contrast rather than luminance contrast. This likely reflects the fact that color vision—despite its great aesthetic appeal—makes only a small contribution to our overall fitness. As an illustration of this, recall that colorblind individuals, who in a sense have lost half of their color space, can grow up without ever noticing that defect.

Congenital Color Blindness Takes Several Forms

Few people are truly colorblind in the sense of being wholly unable to distinguish a change in color from a

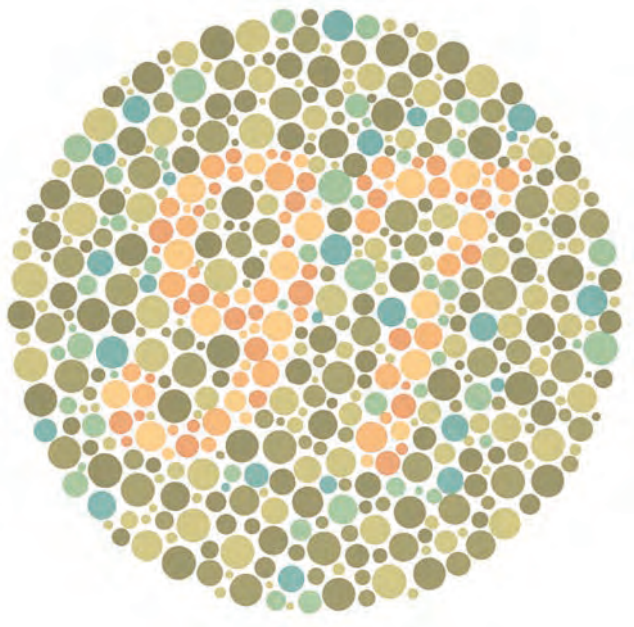

Figure 22–16 A test for some forms of color blindness. The numerals embedded in this color pattern can be distinguished by people with trichromatic vision but not by dichromats who are weak in red–green discrimination. If you don't see any numbers please have your vision tested. (Reproduced, with permission, from Ishihara 1993.)

change in the intensity of light, but many individuals have impaired color vision and experience difficulties in making distinctions that for most of us are trivial, for example between red and green. Most such abnormalities of color vision are congenital and have been characterized in detail; some other abnormalities result from injury or disease of the visual pathway.

Some people have only two classes of cones instead of three. These dichromats find it difficult or impossible to distinguish some surfaces whose colors appear distinct to trichromats. The dichromat's problem is that every surface reflectance function is represented by a two-value description rather than a three-value one, and this reduced description causes dichromats to confuse many more surfaces than do trichromats. Simple tests for color blindness exploit this fact (Figure 22–16).

Although there are three forms of dichromacy, corresponding to the loss of each of the three types of cones, two kinds are much more common than the third. The common forms correspond to the loss of the L cones or M cones and are called *protanopia* and *deuteranopia*, respectively. Protanopia and deuteranopia almost always occur in males, each with a frequency of about 1%. The conditions are transmitted by women who are not themselves affected, and so implicate genes on the X chromosome. A third form of dichromacy, *tritanopia*, involves loss or dysfunction of the S cone. It affects only about 1 in 10,000 people, afflicts women and men with equal frequency, and involves a gene on chromosome 7.

Because the L and M cones exist in large numbers, one might think that the loss of one or the other type would impair vision more broadly than just weakening color vision. In fact, this does not happen because the total number of L and M cones in the dichromat retina is not altered. All cells destined to become L or M cones are probably converted to L cones in deuteranopes and to M cones in protanopes.

In addition to the relatively severe forms of colorblindness represented by dichromacy, there are milder forms, again affecting mostly males. These so-called anomalous trichromats have cones whose spectral sensitivities differ from those in normal trichromats. Anomalous trichromacy results from the replacement of one of the normal cone pigments by an altered protein with a different spectral sensitivity. Two common forms, protanomaly and deuteranomaly, together affect about 7% of males and represent, respectively, the replacement of the L or M cones by a pigment with some intermediate spectral sensitivity.

The genetics of color vision defects are well understood. The genes for the L and M pigments reside on the X chromosome in a head-to-tail arrangement (Figure 22–17A). The pigment proteins have very similar structures, differing in only 4% of their amino acids. People with normal color vision possess a single copy of the gene for the L pigment and from one to three—occasionally as many as five—nearly identical copies of the gene for the M pigment.

The proximity and similarity of these genes predisposes them to varied forms of recombination, leading either to the loss of a gene or to the formation of hybrid genes that account for the common forms of red–green defect (Figure 22–17B). Examination of these genes in dichromats reveals a loss of the L-pigment gene in protanopes and a loss of one or more M-pigment genes in deuteranopes. Anomalous trichromats have L-M or M-L hybrid genes that code for visual pigments with shifted spectral sensitivity; the extent of the shift

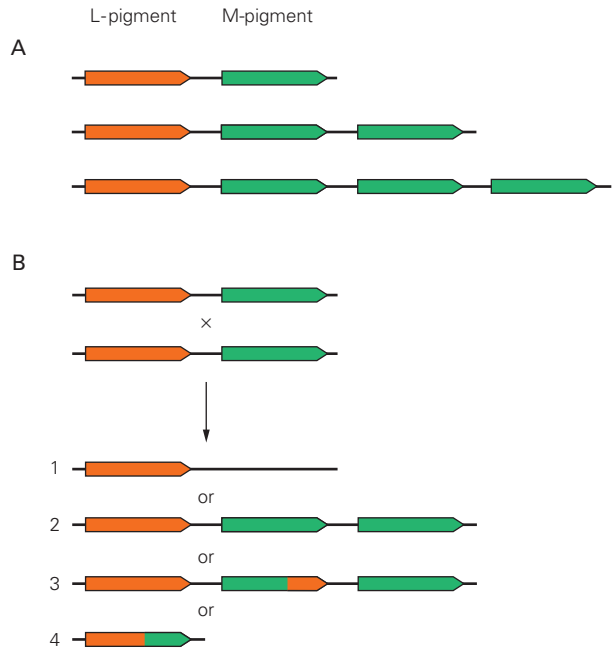

Figure 22–17 L- and M-pigment genes on the X chromosome.

A. The L- and M-pigment genes normally lie next to each other on the chromosome. The base of each arrow corresponds to the 5′ end of the gene, and the tip corresponds to the 3′ end. Males with normal color vision can have one, two, or three copies of the gene for the M pigment on each X chromosome. (Adapted, with permission, from Nathans, Thomas, and Hogness 1986. Copyright © 1986 AAAS.)

B. Recombinations of the L- and M-pigment genes can lead to the generation of a hybrid gene (3 and 4) or the loss of a gene (1), the patterns observed in colorblind men. Spurious recombination can also cause gene duplication (2), a pattern observed in some people with normal color vision. (Adapted from Streyer 1988. Used with permission from J. Nathans.)

depends on the point of recombination. In tritanopes, the loss of S-cone function arises from mutations in the S-pigment gene.

Rod and Cone Circuits Merge in the Inner Retina

For vision under low-light conditions, the mammalian retina has an ON bipolar cell that is exclusively connected to rods (see Figure 22–3B). By collecting inputs from up to 50 rods, this rod bipolar cell can pool the effects of dispersed single-photon absorptions in a small patch of retina. There is no corresponding OFF bipolar cell dedicated to rods.

Unlike all other bipolar cells, the rod bipolar cell does not contact ganglion cells directly but instead excites a dedicated neuron, the AII amacrine cell. This amacrine cell receives inputs from several rod bipolar cells and conveys its output to cone bipolar cells. It provides excitatory signals to ON bipolar cells through gap junctions as well as glycinergic inhibitory signals to OFF bipolar cells. These cone bipolar cells in turn excite ON and OFF ganglion cells, as described earlier. Thus, the rod signal is fed into the cone system after a detour that produces the appropriate signal polarities for the ON and OFF pathways. The purpose of the added interneurons may be to allow greater pooling of rod signals than of cone signals.

Rod signals also enter the cone system through two other pathways. Rods can drive neighboring cones directly through electrical junctions, and they make connections with an OFF bipolar cell that services primarily cones. Once the rod signal has reached the cone bipolars through these pathways, it can take advantage of the same intricate circuitry of the inner retina. Thus, the rod system of the mammalian retina may have been an evolutionary afterthought added to the cone circuits.

The Retina's Sensitivity Adapts to Changes in Illumination

Vision operates under many different lighting conditions. The intensity of the light coming from an object depends on the intensity of the ambient illumination and the fraction of this light reflected by the object's surface, called the *reflectance*. The range of intensities encountered in a day is enormous, with variation spanning 10 orders of magnitude, but most of this variation is useless for the purpose of guiding behavior.

The illumination intensity varies by about nine orders of magnitude, mostly because our planet turns about its axis once a day, while the object reflectance varies much less, by about one order of magnitude in a typical scene. But this reflectance is the interesting quantity for vision, for it characterizes objects and distinguishes them from the background. In fact, our visual system is remarkably good at calculating surface reflectances independently of ambient illumination (Figure 22–18).

With an overall increase in ambient illumination, all points in the visual scene become brighter by the same factor. If the eye could simply reduce its sensitivity by that same factor, the neural representation of the image would remain unchanged at the level of the ganglion cells and could be processed by the rest of the brain in the same way as before the change in illumination. Moreover, the retinal ganglion cells would only need to encode the 10-fold range of image intensities owing to the different object reflectances, instead of the 10-billion-fold range that includes variations in ambient illumination. Some of this adjustment in sensitivity is performed by the pupil, which contracts in bright light, reducing retinal illumination by up to a factor of 10. In addition, the retina itself performs an automatic gain control, called *light adaptation*, that approaches the ideal normalization we have imagined here.

Light Adaptation Is Apparent in Retinal Processing and Visual Perception

When flashes of light of different intensity are presented with a constant background illumination, the responses of a retinal ganglion cell fit a sigmoidal curve (Figure 22–19A). The weakest flashes elicit no response, a graded increase in flash intensity elicits graded responses, and the brightest flashes elicit saturation. When the background illumination is increased, the response curve maintains the same shape but is shifted to higher flash intensities. Compensating for the increase in background illumination, the ganglion cell is now less sensitive to light variations: In the presence of a higher background, a larger change is needed to cause the same response. This lateral shifting of the stimulus–response relationship is a hallmark of light adaptation in the retina.

The consequences of this gain change for human visual perception are readily apparent in psychophysical experiments. When human subjects are asked to detect a flash in a background field of constant illumination, detection on a brighter background necessitates a brighter flash (Figure 22–19B). Under the ideal gain-control mechanism discussed earlier, two stimuli would produce the same response if they caused the same fractional change from the background intensity. In that case, the threshold flash intensity should