IB Physiology

Dr H.R. Matthews
(hrm1@cam.ac.uk)

VISION

Six lectures

3, 5, 8, 10, 12, 15 Feb 1999

Lent term

Objectives

These lectures are intended to introduce you to the vertebrate visual system. After these lectures, the associated practicals, your supervisions, and your own study from textbooks you should understand:

- The physical and neuronal limitations to visual resolution and performance.
- The primary transduction events which take place within the photoreceptor cells, and the ways in which these signals are processed within the retina.
- The concept of the receptive field of a neuron, and the changes which take place in receptive field structure and stimulus preference as you ascend the visual pathway.
- The representation and processing of visual information within the cerebral cortex.
- The ways in which colour information is extracted and processed.
- How the visual system can respond over such a wide range of light intensity.

The visual system provides a good example of strategies adopted by the central nervous system when processing sensory information. Many students find it helpful to compare the principles which they meet in vision with those which they learn about when studying other sensory systems elsewhere in the course.

Textbooks


For more depth on specific topics


Detailed reviews of specific topics
Articles in *Trends in Neurosciences* vol. 9 no. 5 (May, 1986); Special issue: *Information processing in the retina.*


TOPICS

1: Image formation
   1.1: Structure of the eye
   1.2: Physiological optics
   1.3: Image quality
   1.4: Resolution

2: Retina
   2.1: Structure of the retina
   2.2: Retinal photoreceptors
   2.3 Phototransduction
   2.4: Signal transmission within the retina
   2.5: Processing in the mammalian retina

3: Visual pathway and cortex
   3.1: Visual pathway
   3.2: Primary visual cortex
   3.3: Cortical receptive field organization
   3.4: Ocular dominance
   3.5: Processing in higher cortical areas

4: Spatial analysis of the image
   4.1: The use of sinusoidal stimuli
   4.2: Spatial contrast sensitivity
   4.3: Spatial frequency filtering
   4.4: Spatial frequency channels

5: Colour vision
   5.1: Trichromacy
   5.2: Colour opponent processing
   5.3: Colour perception

6: Light and dark adaptation
   6.1: Intensity range of vision
   6.2: Light adaptation
   6.3: Photoreceptor adaptation
   6.4: Quantal fluctuations
   6.5: Dark adaptation
   6.6: Retinal reorganization during dark adaptation
1: IMAGE FORMATION

1.1: Structure of the eye

Incident light passes through the transparent cornea, and the aqueous humour in the anterior chamber. It then passes through the pupil to reach the lens. Finally it traverses the vitreous humour to reach the retina.

1.2: Physiological optics

When an image is formed by a thin converging lens, the positions of the object and image are related to the focal length $f$ by the lens equation:

$$\frac{1}{u} + \frac{1}{v} = \frac{1}{f} = D$$

The refractive power of the lens, $D$, is defined as the reciprocal of its focal length in metres. The unit of refractive power is the Dioptré.

The optics of the eye can be treated as a sequence of spherical surfaces separating media of different refractive index.

To a first approximation it is possible to sum the powers of individual surfaces in a complex optical system to obtain the total refractive power. This approximation works well only if the surfaces are relatively close together.
The optics of the eye form an inverted image of the visual world on the retina. As light passes through the eye to the retina, it encounters four refractive surfaces:

<table>
<thead>
<tr>
<th></th>
<th>Far vision</th>
<th>Near vision</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cornea</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Front surface</td>
<td>48.7 D</td>
<td>48.7 D</td>
</tr>
<tr>
<td>Rear surface</td>
<td>-5.9 D</td>
<td>-5.9 D</td>
</tr>
<tr>
<td>Lens</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Front surface</td>
<td>7.0 D</td>
<td>13.2 D</td>
</tr>
<tr>
<td>Rear surface</td>
<td>11.7 D</td>
<td>13.2 D</td>
</tr>
</tbody>
</table>

These surfaces provide a total refractive power of **60 Dioptre** when viewing a distant object. Most of the refractive power is due to the front surface of the cornea, because of the large difference in refractive index between air and the cornea. The rear surface of the cornea contributes slight negative power, because of a decrease in refractive index between cornea and aqueous humour.

The lens contributes only about a third of the total refractive power, but its power can be adjusted to focus the eye on objects at different distances. To focus on distant objects, the refractive power of the lens is decreased, while to focus on nearby objects the refractive power is increased. This process is known as **accommodation**.

The lens of the eye is deformable. It is suspended from the **ciliary muscle** by the fibres of the **suspensory ligament**. When the eye focuses on a distant object, the ciliary muscle is relaxed, and the diameter of the springy ciliary ring increases. This pulls on the suspensory ligament, flattening the lens, which decreases its optical power. When the eye accommodates to focus on a nearby object, the ciliary muscle contracts, thereby decreasing the diameter of the ciliary ring. This slackens the suspensory ligament, releasing the normal tension and allowing the lens to adopt a more rounded shape which increases its refractive power. The ciliary muscle is under **parasympathetic** control from the midbrain via the **ciliary ganglion**.
In the normal eye with accommodation fully relaxed, an object at infinity produces a sharp image on the retina. The eye is said to be **emmetropic**. However, if the retina is not located in the eye's focal plane, then the image will appear blurred. Such refractive errors can be subdivided into two classes, according to whether the focal plane is in front or behind the retina.

In **myopia**, the image of a point at infinity falls in front of the retina, so that the **far point** is closer than infinity. The subject therefore cannot image distant objects clearly, and is said to be "**short sighted**". Myopia can be corrected by using a **diverging lens** to "spread" the rays from infinity, so that they appear to come from the eye's inappropriately close "far point".

In **hypermetropia**, the image of a point at infinity falls behind the retina when the accommodation mechanism is fully relaxed. To obtain a sharp image of a distant object, the eye must therefore continuously accommodate. Hypermetropia can be corrected by using a **converging lens** to provide the extra power needed in order to image a distant object without continuous accommodation.

The range of accommodation decreases steadily with age, due to changes in the shape and elasticity of the lens, a process known as **presbyopia**. By the mid fifties, the amplitude of accommodation is zero, and nearby objects cannot be imaged clearly. Presbyopia can be corrected by using an additional **converging lens** to view nearby objects.

In **astigmatism** the eye exhibits a greater refractive power in one plane than another. This means that the image of a point is brought to a focus at different positions in the vertical and horizontal planes. Astigmatism can be corrected by using a **cylindrical lens** to alter the refractive power in one plane to make the two foci coincide.
1.3: Image quality

In addition to these purely refractive problems, degradation of the retinal image can take place through the processes of diffraction, chromatic aberration and spherical aberration.

**Diffraction** results from the wave nature of light. If plane waves are incident on a barrier with a small aperture, constructive interference between the wavelets from different points across the aperture causes the resultant beam of waves to spread or diffract. At a fixed wavelength diffraction is more pronounced for small apertures than for large ones.

The diffraction of light through a circular aperture forms a concentric diffraction pattern known as **Airey’s Rings**. The diameter of the central maximum, is directly proportional to the wavelength \( \lambda \), and inversely proportional to the diameter of the diffracting aperture \( D \). It also depends directly on the focal length \( f \) of the optical system, which corresponds to the distance of the aperture from the screen on which the diffraction pattern is produced. In the eye, the pupil acts as the limiting aperture, so diffraction poses a fundamental physical limit to the quality of the retinal image.

**Chromatic aberration** results from the variation of the refractive index of the cornea and lens as a function of wavelength. The refractive power of the eye is greater for blue than for red light, so blue rays are brought to a focus closer to the lens than red rays, yielding coloured fringes around retinal images. This is most pronounced at large pupil sizes, as the rays through the outer part of the lens are refracted most powerfully.

To combat this problem, the eye accommodates to produce an image which is sharply focused for yellow-green light, and mostly ignores the blue end of the spectrum for the resolution of fine detail. This is achieved by largely excluding blue-sensitive cone photoreceptors from the high resolution **fovea**, and by covering the fovea with a yellow filtering pigment, called the **macula lutea**, to exclude blue light.

**Spherical aberration** results from the use of optical surfaces which are spherical in profile. A lens with a spherical surface does not form a perfect point image from a point object. Rays from the outer portion of the lens are deflected more strongly than those passing through the centre. Therefore these marginal rays are brought to a focus closer to the lens than those from the centre, thus blurring the image.

Spherical aberration is not an especially serious problem for the eye, as the cornea is not actually perfectly spherical. The outer portion of the cornea is slightly flatter than the centre, thereby reducing its refractive power and correcting for most of the aberration. Spherical aberration is most serious at large pupil sizes when the marginal rays contribute to the image.
1.4: Resolution

The combined effects of diffraction, chromatic aberration and spherical aberration prevent the eye from forming a perfectly sharp image. A perfect line object forms a broader distribution on the retina known as the line spread function (heavy trace). The central peak is not much worse than would be expected from diffraction by a 2mm pupil (light trace). To either side of this central peak is a broad skirt resulting from the effects of the other aberrations.

As the pupil becomes smaller, the resulting degradation of the image by diffraction increases. Conversely, when the pupil diameter increases, aberrations degrade the image to a progressively greater extent. So there is an optimal pupil size, of a little over 2mm, at which these two effects balance.

Pupil size is adjusted by two muscles. The sphincter muscle is under parasympathetic control, and serves to reduce the size of the pupil. The dilator muscle is under sympathetic control, and expands the pupil. Pupil size is adjusted according to the visual environment under the control of three reflexes mediated by the pretectal area of the midbrain.

The first two reflexes control pupil size according to the light intensity. An increase in light intensity results in a decrease in pupil size, either in the same eye (direct light response) or in the other eye (consensual light response). These pupillary light responses trade off between the quality of the retinal image and the quantity of light admitted. They can vary intensity over a 16-fold range, which is actually rather unimportant compared with other mechanisms of adaptation.

The third reflex is the convergence response. When the eyes are accommodated to view a nearby object, the pupil constricts. If light from a point object is brought to a focus just in front of the retina, the image formed on the retina itself is out of focus, yielding a blur circle. When the pupil is constricted the rays forming the outer part of the blur circle are obstructed, and its size decreases, increasing the depth of field.
Whether the eye can resolve two objects depends on the degree of overlap of their images on the retina. If the images of two point objects are well separated then they are easy to resolve. However, when they partly overlap, the task becomes more difficult. In principle, two images can be distinguished as long as a dip remains between the two peaks. Once overlap becomes so great that this dip vanishes, then the objects cannot be resolved.

However, for the visual system to detect the two images, the retinal photoreceptors must sample the image sufficiently densely to provide a faithful representation.

To adequately resolve the "coarse" image, the retina needs a photoreceptor at each peak and each trough. This is known as the **sampling criterion**, which requires that the image must be sampled at twice the highest spatial frequency of the image.

If the "fine" image is sampled with the "coarse" receptor array then it results in the same response as the "coarse" image. This is known as **aliasing**. To adequately sample the "fine" image, a proportionately finer receptor array is required, in order to satisfy the sampling criterion.
2: RETINA

2.1: Structure of the retina

The retina consists of a number of layers which transform the optical image into a neural representation. The photoreceptor layer, containing the rod and cone photoreceptors, faces the pigment epithelium at the back of the eyeball. Light therefore has to pass through the neural layers of the retina before it can form an image on the photoreceptor layer. The photoreceptors transmit their signals to bipolar cells in the outer synaptic layer, where horizontal cells mediate lateral interactions between photoreceptors and bipolar cells. In the inner synaptic layer bipolar cells synapse with ganglion cells, whose axons carry the output along the optic nerve to the brain. Amacrine cells mediate lateral interactions between ganglion and bipolar cells within the inner synaptic layer.

Rods and cones are not distributed uniformly. In the central retina, in the specialised region known as the fovea, there is a high cone density and a low rod density. Away from the fovea, the cone density falls, while the rod density rises to a peak in the parafoveal region. At the blind spot, where the optic nerve leaves the eye there are no photoreceptors. Finally the rod density falls towards the periphery of the retina.

The high cone density in the fovea samples the retinal image more densely than in the periphery. Each foveal cone sends its signals via 2-3 "private" ganglion cells, thereby preserving this high sampling resolution. In contrast, many rods converge on each ganglion cell, thereby degrading the detail of their representation of the image.

At the fovea the optical quality of the retinal image is enhanced by displacing interneurones and ganglion cells to either side, thereby thinning the neural layers of the retina through which the light must pass to reach the photoreceptors, and displacing retinal blood vessels. This stratagem reduces light scatter.

The high foveal cone density and high image quality mean that visual acuity is highest at the fovea, and falls off rapidly towards the periphery. Eye movements are therefore necessary to direct the fovea towards objects of interest: these are described in detail in your practical classes.
2.2: Retinal photoreceptors

Retinal photoreceptors are divided into two distinct classes, the rods and the cones, which differ in both structure and function. Both rods and cones are subdivided into two parts. The outer segment is specialised for phototransduction. The inner segment is concerned with metabolic functions, and contains mitochondria and the nucleus. At the synaptic terminal, the photoreceptor signal is transmitted to second order cells in the retina.

Both rod and cone outer segments are filled with a stack of transverse membranes. In cones, the stacked membranes are infoldings of the cell membrane, known as sacs. In rods, the stacked membranes are originally formed in a similar way at the base of the outer segment. As time progresses, these infoldings move along the outer segment and pinch off, to form individual disks which are separate from the surface membrane. In both rods and cones, the outer segment is connected to the inner segment by a modified cilium.

The stacked membranes of the disks and sacs contain photopigment molecules which absorb light, and initiate phototransduction. The rod pigment rhodopsin consists of two parts: a protein known as opsin, and embedded within it the retinal chromophore which absorbs the incident light. The opsin determines which wavelengths the chromophore can absorb, and then initiates the biochemical processes which lead to the light response. Cone pigments are similar to rhodopsin, but humans have three different cone opsins, which form pigments which absorb different wavelengths.

The chromophore is 11-cis-retinal, an aldehyde derived from Vitamin A. When incorporated into rhodopsin, retinal is covalently linked to the opsin at the aldehyde group by a Schiff base linkage. When retinal chromophore absorbs light the 11-cis bond is photoisomerised to yield all-trans retinal. Photoisomerisation initiates a series of changes which enable the opsin to interact with other molecules in the outer segment and initiate the light response.
All-trans retinal cannot be regenerated to 11-cis-retinal while it is attached to opsin, but must dissociate and pass from the photoreceptor to the pigment epithelium. All-trans retinal dissociates from opsin, and is reduced, to form all-trans retinol, which is transported to the pigment epithelium by an interstitial retinoid binding protein (IRBP). Within the pigment epithelium, an isomerase converts all-trans retinol to 11-cis retinol, which is then oxidised to 11-cis retinal. Finally 11-cis retinal travels back to the photoreceptors, to reassociate with opsin.

Rhodopsin preferentially absorbs blue-green light of wavelength 500nm (filled symbols). Rhodopsin must absorb light in order for a rod to respond, therefore the sensitivity of rods to light is also maximal for blue-green light, and follows the same action spectrum (open symbols).

2.3: Phototransduction

The process by which a photoreceptor generates an electrical response to light is known as phototransduction. In darkness a steady dark current flows in across the membrane of the outer segment, and out again across the inner segment. The current flowing across the outer segment membrane is carried predominantly by sodium ions, while the current flowing across the inner segment membrane is carried by potassium. The concentration gradients for sodium and potassium are maintained by a sodium-potassium pump in the inner segment.

The circulating current can be recorded by drawing the photoreceptor outer segment into a glass suction pipette, which constrains the current to flow through a current measuring device. A second whole-cell patch pipette can be used to measure the voltage across the cell membrane.
Flashes of light cause a graded suppression of the dark current. For a dim flash, the amount of current suppressed is small. As the flash becomes progressively brighter, more of the current is shut off, until finally for the very brightest flashes, the dark current is shut off altogether. These responses are quite slow: in this amphibian rod it takes about a second for the response to a dim flash to reach its peak. In darkness, the resting potential of the rod is around -30mV. This rather depolarized potential results from the steady dark current flowing in across the outer segment membrane. When the flash shuts off part of the inward current, the cell hyperpolarizes.

Since the disks in which rhodopsin is located are topologically separate from the outer segment membrane in which this conductance change takes place, an internal transmitter is required. Its identity was determined by using the patch clamp technique to apply different substances to the exposed cytoplasmic face of an inside-out patch of outer segment membrane. Cyclic GMP was shown to increase the ionic conductance of the outer segment membrane. The conductance decrease during the light response results from a decrease in the concentration of cyclic GMP, which acts as a negative transmitter.

Light initiates a cascade of reactions which leads to the destruction of cyclic GMP. Photoisomerised rhodopsin (Rh⁺) interacts with a GTP-binding protein, transducin, causing it to bind GTP in exchange for GDP. Transducin activates a phosphodiesterase (PDE), which hydrolyses cyclic GMP. The fall in cyclic GMP concentration allows the outer segment channels carrying the dark current to close, leading to the electrical response. The absorption of a photon by a single rhodopsin leads to the activation of many molecules of transducin, and the activation of one molecule of phosphodiesterase leads to the destruction of many molecules of cyclic GMP. These two amplification stages yield a measurable rod response to a single photon of light. Ultimately, the cascade deactivates, and more cyclic GMP is produced by guanylyl cyclase, which restores the original concentration of cyclic GMP and reopens the outer segment channels.

About 90% of the current across the outer segment membrane is carried by sodium ions, the remaining 10% being carried by calcium ions. Calcium is pumped out by a sodium-calcium exchange which uses the sodium and potassium gradients. When the circulating current is reduced during the light response, calcium entry is reduced, but the exchanger keeps extruding calcium, upsetting this balance and allowing the cytoplasmic concentration to fall. This plays an important role in the control of photoreceptor sensitivity.
Although phototransduction is broadly similar in rods and cones, there are some important functional differences. First, amphibian cone responses are almost five times faster than amphibian rod responses, allowing cones to follow more rapid changes in light intensity than rods. The responses of both types of mammalian photoreceptor are faster still, largely due to the higher mammalian body temperature. Second, cones are much less sensitive than rods. Therefore rods are used at very low light levels, whereas cones operate only at higher intensities.

2.4: Signal transmission within the retina

The interneurones responsible for the transmission of signals within the retina can be divided into two broad classes. First, the bipolar cells mediate transmission of information "straight through" the retina to the ganglion cells which send their axons along the optic nerve. Second, the horizontal cells and amacrine cells mediate lateral interactions at the level of the outer and inner synaptic layers. The responses change from graded potentials in the outer retina to action potentials in the inner retina.

The synapses from photoreceptors to second order cells form a synaptic triad which engulfs the bipolar and horizontal cell terminals to form an invaginating contact. The presynaptic active zone is modified into the synaptic ribbon, which may help to orient vesicles for synaptic release. Mammalian cones also form the flat contact which has no presynaptic ribbon. When the photoreceptor hyperpolarizes in response to light, there is a graded reduction in the release of its transmitter, \( l\)-glutamate.
Bipolar cells fall into two classes: hyperpolarizing bipolar cells and depolarizing bipolar cells. Hyperpolarizing bipolar cells respond to photoreceptor illumination with graded hyperpolarization. The photoreceptor response is transmitted to the hyperpolarizing bipolar cell via a sign preserving synapse, at which glutamate released from the photoreceptor terminal opens cation channels in the bipolar cell membrane. When glutamate release decreases in the light, these channels close, leading to hyperpolarization. Transmission to the depolarizing bipolar cell involves a sign-inverting synapse, at which glutamate decreases a cation conductance. It is therefore called a conductance decrease synapse. When glutamate release falls during the light response, the bipolar cell conductance increases, causing depolarization.

Bipolar cells in the amphibian retina synapse directly with ganglion cells. Ganglion cells also fall into two broad classes: on-centre and off-centre. The off-centre ganglion cell receives a graded hyperpolarizing synaptic potential from the hyperpolarizing bipolar cell via a sign-preserving synapse, which results in a decrease in the rate of firing action potentials. The on-centre ganglion cell receives a depolarizing synaptic potential from the depolarizing bipolar cell, via a sign-preserving, so its firing rate increases on illumination. These two classes of bipolar cell and ganglion cell provide separate on-centre and off-centre pathways within the retina, transmitting information about the presence or absence of light at a particular point on the retinal image.

The conductance decrease synapse to the depolarizing bipolar cell operates via an enzymatic cascade similar to that of the photoreceptor. Glutamate, released by the photoreceptor in darkness, binds to a receptor in the bipolar cell membrane, activating a G-protein, which, in turn activates a phosphodiesterase. The phosphodiesterase destroys cyclic GMP, which is required to keep cation channels open; therefore these channels close in darkness. However, when the photoreceptor hyperpolarizes in response to light, glutamate release decreases, leading to a fall in phosphodiesterase activity. Continual synthesis of cyclic GMP raises the cyclic GMP concentration, opening cation channels so that the bipolar cell depolarizes.

Horizontal cells produce antagonistic lateral interactions between photoreceptors and bipolar cells. If a photoreceptor is connected via a depolarizing bipolar cell to an on-centre ganglion cell, photoreceptor illumination depolarizes the bipolar cell, and increases ganglion cell firing. But if a neighbouring photoreceptor is illuminated, its signal passes via a sign preserving synapse to a horizontal cell, which hyperpolarizes also. A sign-inverting synapse from the horizontal cell depolarizes the central photoreceptor, hyperpolarizes the bipolar cell and decreases the firing rate of the ganglion cell. This centre-surround antagonism may be enhanced by the action of certain classes of amacrine cell in the inner synaptic layer.
The **receptive field** of a ganglion cell is the region of the retina which can influence its discharge. Ganglion receptive fields are circularly symmetric. When the receptive field centre of an **on-centre** ganglion cell is illuminated, its discharge rate increases; whereas illumination of the surrounding annulus decreases firing. If both centre and surround are illuminated simultaneously, an intermediate response results. Consequently ganglion cells respond relatively poorly to spatially uniform illumination. An **off-centre** ganglion cell is inhibited by illumination of the receptive field centre and stimulated by illumination of the surround.

Centre-surround antagonism is a form of **lateral inhibition**, which serves to emphasise local spatial differences in the retinal image. Suppose that an array of receptors is stimulated with a step of light. The receptors at the left hand end will all give the same response to the stimulus, because each receptor is directly excited by light, and also receives a double dose of inhibition from the two receptors on either side. However, the receptor just to the left of the step is itself excited by light, but only receives a single dose of inhibition, as the receptor on its right is not being stimulated. Therefore its response is larger than that of its neighbours to the left. Similarly, the receptor just to the right of the step is not only not directly excited, but also receives inhibition from its neighbour on the left. So its response will be less than that of its rightwards neighbours, which receive neither excitation nor inhibition. This process enhances the response to the step.
2.5: Processing in the mammalian retina:

The processing of signals within the mammalian retina differs somewhat from the amphibian retina described above. The main differences relate to the nature of the direct pathway through the retina mediated by the bipolar cells. These pathways can be subdivided into those primarily responsible for transmitting cone signals, and those responsible for transmitting rod signals.

Mammalian cones make sign-inverting synapses with depolarizing bipolar cells at invaginating ribbon synapses, and sign-preserving synapses with hyperpolarizing bipolar cells at flat contacts. In mammals the ganglion cell receptive field centre may be driven by both depolarizing and hyperpolarizing bipolar cells, using sign-preserving or sign-inverting synapses. This push-pull action may improve signal transmission in the retina.

In the mammalian retina, rods signals can follow two possible routes. First, in the cat, retina rods are electrically coupled via gap junctions to the cone terminals, so that rod signals can gain access to the push-pull cone bipolar pathway to the ganglion cells. Second, there is a specific class of rod bipolar cell which connects only to rods, and hyperpolarizes in response to light. This does not synapse directly with ganglion cells, but instead sends its output via an inhibitory synapse to the AII amacrine, or rod amacrine, which sends its signals via the terminals of the cone bipolar cells, finally reaching both off- and on-centre ganglion cells. Which of these is actually used depends critically on the prevailing level of illumination.

Rod signals are not processed independently in the retina, but are gathered together from a large number of rods covering a substantial area of retina: convergence. Similarly, each rod can send its signals to a large number of ganglion cells: divergence. The high degree of convergence and divergence in the rod pathway makes it unsuitable for resolving fine detail. Instead, it serves to collect light from a large region of the image, thereby assisting vision at low light intensities. Convergence and divergence take place to a much lesser extent in the cone system. At the fovea the degree of cone convergence and divergence is very small, making it ideally specialised for high spatial resolution.
In mammals, retinal ganglion cells can be subdivided into a number of classes. In the cat, which has been especially well studied, there are three classes of ganglion cell: the X cells, Y cells and W cells.

**X cells** have medium-sized cell bodies and more slowly conducting axons. They comprise about 80% of the total ganglion cells. The response of an X cell to light is relatively **sustained**, giving a reasonably steady elevation or depression of the cell's firing rate. X cells have small receptive fields, and therefore subserve high acuity vision and the resolution of fine spatial detail.

**Y cells** have large cell bodies and large rapidly-conducting axons. They comprise about 10% of the total ganglion cell population in the cat retina. Y cells give a **transient** response to light, firing only a few spikes at the onset or offset of the stimulus, then falling silent again. Y cells have larger receptive fields, and serve to analyse crude form; their transient responses makes them especially suitable for the analysis of movement.

**W cells** have small cell bodies and large dendritic arborizations. They project almost exclusively to the superior **colliculus** and **pretectal nuclei**, where they are involved in the control of eye movements and the pupil diameter light reflexes.

The receptive field sizes of different ganglion cell classes vary quite substantially as a function of position on the retina. X and Y cell receptive fields are smallest in the centre of the visual field, and become progressively larger towards the periphery. This is yet another manifestation of the coarser visual grain at the periphery, the most spatially acute vision being reserved for the fovea, where the X cell receptive fields are very small.

In the primate retina, ganglion cells are subdivided into **parvocellular** and **magnocellular** classes, known as **P cells** and **M cells**, which may broadly correspond to the X and Y ganglion cells of the cat retina.
3: VISUAL PATHWAY AND CORTEX

3.1: Visual pathway

The ganglion cell axons pass up the optic nerve from each eye and join at the optic chiasm. The optic chiasm performs a partial decussation of the fibres running in the two optic nerves, which are reorganized so that each half of the brain receives input from the contralateral visual hemifield.

The overlap between the visual fields is used to make comparisons between the images seen by the two eyes for the binocular judgement of relative distance. Such comparisons are only possible in species such as primates with forward-facing eyes and a large degree of binocular overlap. In species with side-facing eyes, such as the rabbit, binocular overlap is minimal.

Suppose the two eyes view objects, A, B and C, two of which are at the same distance, while the third is closer. Suppose that the subject has fixated on object A so that its image falls on each fovea. The image of B falls at the same relative position to that of A in each eye. However, the image of C falls closer to the image of A in the left eye's view than in the right eye's view, because the angle between the ray from C and the axis of the left eye is smaller than the angle between C and the axis of the right eye. The difference between these two angles is known as the disparity. For points A and B the disparity is zero as they are at the same distance, while the non-zero disparity between A and C indicates that they are at different distances. So by comparing the view of the left eye with the view of the right eye, it is possible to determine the distances of point C from the fixation plane.

Following the optic chiasm, the ganglion cell axons proceed up the left and right optic tracts to the lateral geniculate nucleus (LGN). Within each of the six layers of the LGN there is a systematic representation of the contralateral half of the visual field. This representation is distorted as the fovea sends a disproportionately larger number of ganglion cell axons to the lateral geniculate and thus commands a larger proportion of the map of visual space. The inputs from the two eyes are segregated within the LGN. Afferent axons from the ipsilateral eye synapse in layers 2, 3 and 5, whereas axons from the contralateral eye synapse in layers 1, 4 and 6.
The magnocellular layers 1 and 2 contain relatively large cells, while the parvocellular layers 3-6 contain smaller cells. These act as specific targets for the two main subclasses of retinal ganglion cells. The X cells, comprising about 80% of the total, project to the four parvocellular layers of the LGN. The Y cells, comprising about 10% project to the two magnocellular layers. Receptive fields within the LGN are circularly symmetric, but there is an enhancement of the degree of center-surround antagonism in comparison to ganglion cells.

The W cells project to the superior colliculus, where they participate in the control of eye and head movements and in pupillary light reflexes.

### 3.2: Primary visual cortex

The projection cells of the various layers of the LGN send their axons to the primary visual cortex via the optic radiation. The visual cortex is located in the occipital lobe. The primary visual cortex, located in area 17 and also known as the striate cortex, is the target for the projection cells from the LGN. Areas 18 and 19 are involved in higher order visual processing, elaborating specific aspects of the stimulus.

The primary visual cortex receives a map of the contralateral half of the visual field. Disproportionately larger areas of cortex are devoted to the representation of the centre of the visual field, therefore the image at the fovea is not only sampled in greater detail but also represented and processed in great detail, thereby preserving the high foveal acuity. This representation is known as a retinotopic map of the visual field.

The visual cortex, in general with other areas of neocortex is a six-layered structure. The layers are numbered from layer 1 at the surface of the cortex, to layer 6 just above the underlying white matter. Different layers within the cortex are specialised in terms of their inputs and outputs.
The main input from the LGN terminates in layer 4c. Outputs leave the visual cortex from superficial and deeper layers. **Layers 2 and 3** project to higher visual cortical areas. **Layer 5** projects to the superior colliculus, which therefore receives both direct retinal input, and cortical output. **Layer 6** projects back to the LGN, where it may play a role in visual arousal.

Afferent information from the parvocellular and magnocellular layers of the LGN is segregated within layer 4. The parvocellular afferents largely terminate in layer 4c-beta. There are also more minor parvocellular terminations in layers 4a and 6. In contrast, the magnocellular afferents terminate within layer 4c-alpha. From layer 4c-alpha this magnocellular information passes to layer 4b, which then projects to area 19, the **medial temporal cortex**.

### 3.3: Cortical receptive field organization

The response properties of cortical neurons can be determined by using an extracellular electrode inserted into the visual cortex of an experimental animal to record the patterns of action potential discharge in response to stimuli projected onto a screen.

Most cells in visual cortex respond only poorly to small spots of light. Instead, they respond best to elongated bars of light of a particular orientation. Cortical neurons are therefore said to exhibit **orientation tuning**. There are two exceptions to orientation tuning in the primary visual cortex. The first is that cells in layer 4c which receive primary geniculate input have circularly symmetric receptive fields which are not orientation tuned. The second concerns cells in peg-like regions of cortex called **blobs**, which are described in more detail below.

The orientation tuned cells of primary visual cortex are divided into two main categories: **simple cells** and **complex cells**.

**Simple cells** have orientation tuned receptive fields which can be mapped into distinct excitatory and inhibitory regions, using a small spot of light. The best stimulus for a simple cell can be predicted from the map of its receptive field, which thus corresponds to a precise position on the retinal image. The number and sign of regions in the receptive field can vary between different simple cells. There is an overall balance between excitatory and inhibitory regions, so simple cells are not influenced significantly by diffuse illumination which covers the entire receptive field.
One way in which simple cell receptive fields might be constructed is by the convergence of a number of LGN afferents onto a single cortical cell. If the circularly symmetric centre-surround receptive fields of the LGN cells were lined up in a row, then this would result in receptive field with a central excitatory region and two inhibitory flanks. However this scheme is not entirely satisfactory, and the precise way in which the simple cell receptive field originates is not known.

Complex cells also respond to lines of a specific orientation but do not require that this line be located at a precise position within the receptive field. A complex cell therefore responds vigorously to a bar of correct orientation moving slowly across its receptive field. Some complex cells also prefer the stimulus to move in a particular direction across the receptive field. The receptive field of a complex cell cannot be mapped using small spots of light. If you attempt to do so, you just get a confused jumble, rather than the orderly pattern of excitatory and inhibitory regions of the simple cell.

One way in which this behaviour might be produced is by connecting together the outputs from a series of simple cells with appropriately placed receptive fields. Unfortunately, this hierarchical model is almost certainly not correct as complex cells are known to receive primary geniculate input as well as the output from simple cells.

A further stimulus preference shown by cortical cells is the preference for oriented lines of a particular length. Such cells are said to be end-stopped. It was once believed that such end-stopped receptive fields came about by the convergence of two complex cells by means of excitatory and inhibitory synapses. This led to their being called hypercomplex cells, a term which is still much used by textbooks! However, this explanation cannot be correct, as both simple and complex cells can prefer end-stopped stimuli.
When viewing real images these cells will convey different types of information. As an example, consider the response to a bright rectangle on a dark background.

**Ganglion cells** and **LGN afferents** will respond best when their receptive field centres are located either just inside or outside the bright boundary. They will not, however, give any information about the nature of this boundary. **Simple cells** will respond to the boundary if they are tuned to the correct orientation. They therefore indicate a boundary of a particular orientation and position. **Complex cells** will respond to the boundary without requiring that it be at a particular position. They therefore abstract the presence of an edge of a particular orientation. **End-stopped cells** will respond to the corners of the rectangle, indicating the presence of an oriented edge which ended within the receptive field.

Orientation-tuned cells are arranged systematically within the cortex. This can be demonstrated by examining the preferred orientations of the cells encountered during a long electrode penetration through the visual cortex. When the electrode passes perpendicular to the cortical surface, all the cells show the same orientation preference. But when the electrode penetrates the cortex obliquely, there is an orderly progression of preferred orientation along the electrode track. These observations indicate that cells of similar orientation tuning are organised in **orientation columns** perpendicular to the cortical surface. Adjacent columns have only slightly differing orientation preferences.

### 3.4: Ocular dominance

Within the area of binocular overlap each point on the object forms an image on both retinas. These signals first interact at the cortex. If one eye is injected with radiolabelled proline the label is taken up by ganglion cells and transported along their axons to the LGN, where it is taken up by second order cells and transported to the visual cortex, revealing the pattern of afferent termination within layer 4c. It is distributed in labelled patches which receive input from the injected eye, separated by unlabelled regions which receive input from the other eye. These regions of cortex which receive input from one or the other eye are called **ocular dominance columns**.
Within layer 4c, the degree of ocular dominance is complete, but becomes more balanced between the two eyes in the upper and lower layers. Most of these neurones which receive inputs from both eyes, have receptive fields located at corresponding positions on the two retina. But some respond instead to stimuli located at slightly different positions on the two retinae, to function as disparity detectors for detecting objects nearer or further than the plane of fixation. They are used in stereopsis, the binocular judgement of relative depth.

Idealised orientation columns and ocular dominance columns can be thought of as a series of slabs, intersecting at right angles. This columnar organisation is superimposed on the underlying retinotopic map. A particular location in the retinal image is completely analysed for orientation in one complete cycle of preferred orientations, and for stereopsis in a pair of ocular dominance columns. This hypercolumn occupies about 1 square millimetre of cortical surface. The actual layout of orientation columns and ocular dominance columns is less tidy, appearing as two sets of intersecting stripes.

Superimposed on the orientation columns and ocular dominance columns are patches known as blobs which stain for the enzyme cytochrome oxidase.

The unstained regions between are known as interblobs. Each hypercolumn contains two blobs, one for each ocular dominance column. The cells within the blobs are not orientation-tuned, but instead have circularly symmetric receptive fields which show centre-surround antagonism. They respond selectively to particular wavelengths, and are involved in the processing of colour.

3.5: Processing in higher cortical areas

The striate cortex or V1 receives the primary input from the LGN, and is located in area 17. It contains a single retinotopic map of the contralateral visual field. Adjacent to it is area 18, the prestriate cortex. It contains four separate maps of the visual field, known as V2, V3, V3A and V4. Area 19, the medial temporal area contains a single map of the visual field, known as V5, or MT.

If the cortex is stained for cytochrome oxidase, V2 is seen to contain a series of stripes, perpendicular to the boundary between areas 17 and 18. These can be subdivided into thick stripes and thin stripes, with unstained interstripes in between. These represent an orderly pattern of projection from V1 to V2 which are divided into three parallel processing streams.
The first stream runs via the blobs in V1, which receive their input from the parvocellular layers of the LGN, and thus from the sustained X retinal ganglion cells. The blobs send their output to the thin cytochrome oxidase stripes of V2. The thin stripes in V2 then project to V4, which is involved in the detailed processing of colour information. This **blob-thin stripe-V4 stream** is a colour selective pathway, which is not selective for orientation. The spatial resolution of this processing stream is comparatively low, so it is not well suited for the perception of fine detail, but may instead be involved in colour vision.

The second stream runs via the interblob regions of V1. Cells in the interblobs also receive input from the parvocellular layers of the LGN, and comprise the orientation-tuned simple and complex cells. Many of these cells are also binocularly driven and disparity selective. There are also a number of colour-sensitive orientation tuned cells within the interblobs, but these appear to be responsible for the detection of colour boundaries, rather than for colour perception itself. The interblobs send their output to the interstripe regions of V2, which in turn projects to V4. This high spatial resolution **interblob-interstripe stream** is believed to be used for analysing the shapes of objects.

The third stream originates from the magnocellular layers of the LGN which receive input from the transient Y ganglion cells. The magnocellular layers project to layer 4c-alpha of V1. Here, the signal is relayed to layer 4b, and then projects to the thick cytochrome oxidase stripes in V2. These thick stripes then project to the medial temporal area. There is also a direct projection from layer 4b to MT. Many cells within this stream respond specifically to a particular direction of movement within their receptive field. The **magnocellular-4b-thick stripe-MT stream** is principally concerned with the analysis of motion.

The separate processing streams thus handle different attributes of the visual image. Within each parallel stream there is a hierarchical sequence of processing involving a series of different visual areas. Ultimately the information from these streams must be integrated together within higher **inferotemporal** and **parietal** cortical areas for conscious perception.

The way in which these different aspects of the image are combined together for perception is known as the **binding problem**. Certain **elementary properties**, such as brightness, colour and line orientation, can cause boundaries to be automatically detected without conscious thought. This rapid **pre-attentive process** may operate on **feature maps** which encode a particular attribute of the image. Other more subtle properties which depend upon the combination of information from several of these categories require conscious attention for their discrimination. This slower **attentive process** may operate on a **master map** encoding the differences between the individual feature maps, and direct the **spotlight of attention** toward particular regions of interest for further detailed analysis.
4: SPATIAL ANALYSIS OF THE IMAGE

4.1: The use of sinusoidal stimuli

To investigate the ability of a human observer to analyse the spatial structure of an image we need a simple, readily characterised visual stimulus. One stimulus which has been widely used for this purpose is the sine wave grating, in which the intensity varies sinusoidally as a function of position.

The spatial period of such a grating is defined as the distance in which the intensity goes through a full sinusoidal cycle. For measurements on a human observer it is often expressed as the visual angle subtended by a single cycle of the stimulus. The spatial frequency of the grating is the reciprocal of this period, often expressed in terms of cycles per degree of visual angle.

This sinusoidal variation is superimposed on a background light of steady mean intensity. When the sine wave is added to this steady mean level, it results in maximum and minimum intensities corresponding to the peaks and troughs of the sine wave. The mean intensity and the amplitude of the sine wave can be defined in terms of these maximum and minimum intensities:

\[
\text{Mean} = \frac{I_{\text{max}} + I_{\text{min}}}{2} \quad \quad \text{Amplitude} = \frac{I_{\text{max}} - I_{\text{min}}}{2}
\]

The spatial contrast of the stimulus is defined as the amplitude divided by the mean intensity:

\[
\text{Contrast} = \frac{\text{Amplitude}}{\text{Mean}} = \frac{I_{\text{max}} - I_{\text{min}}}{I_{\text{max}} + I_{\text{min}}}
\]

Thus a contrast of zero would correspond to a sine wave of zero amplitude resulting in no modulation from the mean intensity with distance, while a contrast of one would correspond to complete modulation, from zero to twice the mean intensity.

4.2: Spatial contrast sensitivity

The contrast sensitivity of a human observer is defined as the reciprocal of the threshold contrast at which the grating is only just visible:

\[
\text{Contrast sensitivity} = \frac{1}{\text{Threshold contrast}}
\]

The contrast sensitivity can vary between one and infinity. A contrast sensitivity of one is clearly extremely poor: it means that the observer can only detect the grating at maximum modulation. Human contrast sensitivity falls off at both high and low spatial frequencies. Above about 50 cycles/deg the subject cannot detect the stimulus even at full modulation.
Why might this high spatial frequency fall off take place? One possibility might be the blurring effect of the optics of the eye as described by the line spread function. This blurring effect can be analysed for gratings by measuring the ratio of the image contrast to the object contrast as a function of spatial frequency to yield the modulation transfer function. The modulation transfer function of the eye declines with increasing spatial frequency, falling more rapidly in the real eye due to the additional blurring caused by aberrations. This corresponds to blurring of the bars of the grating by the line spread function.

This optical demodulation can be compared with the human contrast sensitivity function by assuming that the contrast sensitivity of the retina and brain are independent of spatial frequency. However, the neural contrast sensitivity function falls more abruptly than this prediction at both high and low spatial frequencies. These observations demonstrate that the contrast sensitivity of the retina and brain does depend on spatial frequency, and that it is this neural limit which determines the human contrast sensitivity function. To appreciate how and why it does so, it is necessary to examine the interactions of such sine wave stimuli with the receptive fields of neurons in the visual system.

4.3: Spatial frequency filtering

Suppose that a peak of a sine wave grating is aligned with the central excitatory region of the circularly symmetric receptive field of an on-centre X ganglion cell. If the spatial frequency of the grating is low, the peak strays into the flanking inhibitory region of the receptive field, and the response is reduced. If instead the spatial frequency of the grating is high, light and dark regions fall in both excitatory and inhibitory regions, and the response is greatly reduced. However, if the spatial frequency is "just right", the peak fills the central excitatory region, and the troughs fall in the inhibitory region, yielding a maximum response. The low frequency decline in contrast sensitivity results from lateral inhibition, while the high frequency fall off stems from the finite size of the receptive field centre.

Cortical simple cells respond optimally to a sine wave grating when a peak fills the excitatory region of the receptive field and the neighbouring troughs fill the inhibitory flanks. But if the spatial frequency is too low or too high then the response is reduced.
Responses to gratings change systematically at different levels in the visual pathway. The centre-surround receptive fields of retinal ganglion cells yield broadly-tuned responses to gratings of different spatial frequency. The peak spatial frequency depends on the size of the receptive field centre. At the lateral geniculate nucleus additional lateral inhibition sharpens the spatial frequency tuning curve, enhancing the low-frequency fall in sensitivity. In the cortex, this sharpening continues, mediated by the multiple excitatory and inhibitory regions of the simple cell receptive field, so cortical simple cells will respond to gratings within a narrow range of spatial frequency of the correct orientation.

The behavioural contrast sensitivity curve is the envelope of the narrowly-tuned spatial contrast response curves of many cortical cells with different receptive field sizes. This is illustrated here for two simple cells in cat visual cortex. Note that the contrast sensitivity peaks at a lower frequency in this nocturnal animal than in the human.

4.4: Spatial frequency channels

A group of cortical cells with overlapping receptive fields of different sizes function as distinct spatial frequency channels. The visual system may carry out a local Fourier analysis in each small region of the image, breaking it down into a number of sinusoidal components.

A simple example of Fourier synthesis is the construction of a square wave grating from sinusoidal components. First we need a sine wave of the same fundamental frequency. To this are added higher odd harmonics, with the correct amplitude and phase. Perfect reconstruction of the square wave requires an infinite number of harmonics.

This relationship between square and sine waves can be used to demonstrate spatial frequency channels by comparing the contrast sensitivity functions for the two stimuli. At low spatial frequencies the square wave grating is more easily detected, because the visual system detects the higher harmonics, to which it is more sensitive.
Further evidence for the existence of spatial frequency channels comes from selective adaptation. Suppose that an observer views a grating of constant spatial frequency for an extended period. Thereafter contrast sensitivity is reduced for spatial frequencies close to that of the adapting grating; the prolonged exposure to the adapting grating has desensitised the spatial frequency channels which can respond to it, but has left the others relatively unscathed. These spatial frequency channels correspond to populations of neurones in the visual pathway which respond to particular ranges of spatial frequency.
5: COLOUR VISION

The ultimate purpose of colour vision is to allow the detection of objects which reflect light of different spectral composition from the backgrounds upon which they are superimposed. Ideally the perceived colours of objects should not depend on the conditions of illumination but instead estimate the spectral reflectance of objects, irrespective of the spectral composition of the illuminating light. Human colour vision can respond to a range of wavelengths, varying from 400 nm at the blue end of the spectrum, through 500 nm in the green, to 700 nm in the far red.

5.1: Trichromacy

Human colour vision is based on the absorption of light by three different cone pigments, which preferentially absorb at short, medium and long wavelengths. Each cone has only one type of pigment, resulting in blue, green and red cones. The response is initiated by the absorption of light by the photopigment, which determines the action spectrum of the cone. Human colour vision is therefore trichromatic. In the intact eye the spectral responses will be affected by the yellow pigmentation of the human lens. The pigment absorbance curves are quite broad leading to significant overlap, especially between the red and green pigments. Thus each wavelength will stimulate more than one class of cone. However, the waveform of the cone response to light does not depend on the wavelength: a property known as spectral univariance.

The possession of more than one cone class allows discrimination of objects which differ in colour but not intensity. Suppose that a monochromat, with only one cone class, views an object reflecting a single wavelength superimposed on a background of equal intensity and a different wavelength. If the object causes the same cone response as the background then it will be invisible. But a dichromat, who possesses two classes of cone, can discriminate them as this object preferentially stimulates short wavelength cones, whereas the background preferentially stimulates long wavelength cones.

A trichromat can discriminate objects which would be invisible to a dichromat. Suppose that the object comprises both short and long wavelengths, while the background is a single wavelength in between. A dichromat cannot detect the object. However a trichromat with a third class of short wavelength cone can discriminate them, as only the object stimulates the short wavelength cones.
Colour discrimination is achieved by comparing the degree of stimulation of the different cone classes. Consider a dichromat with medium and long wavelength cones. At short wavelengths only the M cone is stimulated, whereas at long wavelengths only the L cone is stimulated. But in the region of overlap in between, both classes of cone are stimulated. If the responses of the two classes of cone are plotted against each other, each wavelength and intensity of stimulation will generate a single point. The distance of the point from the origin represents brightness, while the angle made by the vector joining it to the origin indicates colour. As the wavelength becomes progressively longer, the vector pivots from vertical, representing green, towards horizontal, representing red. In between, both cone types are stimulated, giving rise to yellow. The colour can be represented by the intersection between the vector and the line joining the 100% stimulation points on the two axes, known as the colour line for the dichromat. The overlap between the pigment curves is vital for this graded response to wavelength.

In a trichromat this concept can be extended to three dimensions, corresponding to the response of each cone class as it is stimulated by a light of constant intensity and changing wavelength. If brightness is ignored, the colour of the stimulus can be represented by projecting the response curve onto the equilateral triangle cutting off equal distances on each of the three axes.

The curve within this colour triangle represents the possible responses that can be obtained by stimulation with light of a single wavelength. As the wavelength becomes longer the response point moves from the blue vertex of the triangle along the heavy curve to the red vertex. There is an unattainable region at the green vertex of the colour triangle, where all three classes are being excited. As these pure spectral colours are diluted with an increasing amount of white the response point moves towards the centre of the triangle.
5.2: Colour opponent processing

Cone responses thus combine information about brightness and colour. These are transformed by the visual system into independent **colour opponent channels**. In the **red-green** opponent channel, the signal is the difference between the responses of the medium and long wavelength cones. The **blue-yellow** opponent channel is formed from the difference between the responses of the short wavelength cones, and the sum of the medium and long wavelength cone responses. By summing together the responses of the medium and long wavelength cones, a signal representing brightness, or **luminance** is produced. These opponent channels produce a steep variation of response with wavelength in the regions of pigment curve overlap, giving high sensitivity to changes in wavelength.

![Colour opponent channel diagram](image)

These colour opponent channels correspond to a transformation of co-ordinates in the colour triangle. The point at the centre represents white, when all three cone classes are stimulated equally. Crossing this point are two colour opponent axes. The horizontal axis represents the differential excitation of red versus green cones; the vertical axis represents the differential excitation of blue cones versus red and green cones.

These colour opponent channels are set up within the primate retina. The majority of primate retinal ganglion cells display centre-surround antagonism, but the spectral sensitivity of the centre differs from that of the surround. Most ganglion cells fall into red-green and blue-yellow antagonistic classes. For example, a given cell might be excited by red light to the receptive field centre, and inhibited by green light to the surround. These colour-encoded ganglion cells are known as **single opponent** cells, as the antagonism takes between different regions of the receptive field, driven by different cone mechanisms. The single opponent cells correspond to the **P cells** in the primate. The **broad band** class of retinal ganglion cell are driven by both red and green cones. They exhibit centre surround antagonism without a chromatic component, thus encoding luminance, and correspond to the **M cells**. A small number of **non-concentric** ganglion cells do not exhibit centre-surround antagonism. Their receptive fields may be driven by one or several cone classes, without antagonism.
These single opponent cells provide signals which are ambiguous for colour and brightness. For example, a red-green single opponent cell cannot discriminate between large and small red spots and a small white spot. This problem is resolved by double opponent receptive fields, in which chromatic antagonism takes place not only between centre and surround but also within each region. For example, red light might excite the centre and inhibit the surround, whereas green light would inhibit the centre and excite the surround. This approach resolves the ambiguity of single opponent signals.

Double opponent cells are found in the cytochrome oxidase blobs of the striate cortex. Their receptive fields might be constructed by antagonistically connecting together single opponent cells of opposite colour preference at appropriate positions on the retina. They project via the thin stripes to V4 in area 18. The cells in V4 exhibit a very narrow degree of spectral tuning, each responding only to a narrow band of wavelengths. So V4 no longer represents the opponent channels seen lower down the pathway, but deals instead with individual colours or hues. These properties give rise to the phenomenon of colour constancy.

5.3: Colour perception

A trichromat can always match a test light of arbitrary colour by appropriately adjusting the intensities of three primary colours: a Rayleigh match. These primaries must be reasonably well spaced in wavelength, so as to preferentially stimulate each of the classes of cone. In the simplest form of match it is simply necessary to adjust the relative intensities of our three primaries on one side of the screen in order to match the test light, \( L \), on the other. However, sometimes it may be necessary to add one of the three primaries to the same side of the screen as the test light, because the test light may not sufficiently stimulate one of the cone classes.

Lack of an individual cone pigment in protanopia (red), deuteranopia (green), or tritanopia (blue) results in dichromatic colour vision. Less extreme is the possession of an abnormal cone pigment, with a modified peak wavelength of absorption. For example, in protanomaly, the red cone pigment is shifted towards yellow. This means that more red is needed in colour matches than for a normal observer in order to sufficiently stimulate this abnormal pigment with a shorter than normal peak wavelength. In deuteranomaly, the green cone pigment is shifted towards yellow,
with an equivalent need for extra green in the colour match. In contrast to these red-green anomalies, congenital abnormality of the blue mechanism, or tritanomaly is very rare.

<table>
<thead>
<tr>
<th></th>
<th>Red</th>
<th>Green</th>
<th>Blue</th>
<th>Rod</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>Rod monochromat</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>+</td>
<td>Low acuity</td>
</tr>
<tr>
<td>Protanopia</td>
<td>0</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>Deuteranopia</td>
<td>+</td>
<td>0</td>
<td>+</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>Tritanopia</td>
<td>+</td>
<td>+</td>
<td>0</td>
<td>+</td>
<td>Rare, usually acquired</td>
</tr>
<tr>
<td>Protanomaly</td>
<td>Shifted</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>Male linked, genetic</td>
</tr>
<tr>
<td>Deuteranomaly</td>
<td>+</td>
<td>Shifted</td>
<td>+</td>
<td>+</td>
<td>Male linked, genetic</td>
</tr>
<tr>
<td>Tritanomaly</td>
<td>+</td>
<td>+</td>
<td>Shifted</td>
<td>+</td>
<td>Very rare</td>
</tr>
</tbody>
</table>

Such defects of colour vision are especially common for the red and green pigments, the genes for which are located close together on the X chromosome, giving rise to male-linked “colour blindness”. This can arise from deletion of the MW or LW gene by unequal intergenic recombination, or the creation of a hybrid gene by intragenic recombination.

Colour opponent cells account for colour opponency within the boundaries of an object. However, double opponent cells can also enhance colour contrasts across the boundaries of objects. For example, shining a green light into the surround of a double opponent cell with a red centre and a green surround will induce a response which would be interpreted as the presence of red in the receptive field centre. Such comparisons serve to correct for the variations in the spectral composition of the illuminating light and thereby aid a more accurate assessment of reflectance. They are believed to give rise to the phenomenon of colour constancy.
6: LIGHT AND DARK ADAPTATION

6.1: Intensity range of vision

The visual system has to operate over an enormous intensity range. At visual threshold, a human observer can detect the absorption of only a few photons of light. But the intensity can be made some $10^{13}$ times brighter before vision ceases and retinal damage results.

The first factor which permits such an enormous operating range is the use of both rods and cones in a duplex retina. Consequently, each type of receptor has to operate over a more restricted range. The rods are extremely sensitive so that they can respond to light of very low intensity within the scotopic range. Scotopic sensitivity is highest in the parafoveal region, which has the highest rod density. As the intensity of retinal illumination increases, the less sensitive cones start to respond too within the mesopic range. Finally, the intensity becomes too high for the rods, whose responses saturate, so within the photopic range only the cones contribute to vision.

At any moment the eye only receives a much smaller range of intensities, because objects normally reflect light from some other light source in proportion to the reflectance of the object. So the eye only needs to function over a narrower intensity range of only $10^2$-$10^3$ under any particular illumination. The visual system is not concerned with absolute intensities, but instead with the differing reflectances of objects, according to whether they are at the top or the bottom of the current operating intensity range. This sliding scale of brightness results from automatic changes in the sensitivity of the rod and cone pathways known as adaptation. Adaptation can be subdivided into two types. Field adaptation, also known as light adaptation, is the rapid and reversible change in sensitivity which takes place when the steady intensity is altered. Bleaching adaptation is the profound decrease in sensitivity induced by very bright light, which recovers only slowly in the process of dark adaptation.

6.2: Light adaptation

Field adaptation can be investigated by an increment-threshold experiment. The sensitivity of the rod system alone is tested using a stimulus consisting of a green test spot, which preferentially stimulates rods, superimposed on an orange background, which preferentially adapts the medium and long wavelength cones. The subject fixates on the eccentrically-placed cross, so that the stimulus falls on the parafoveal region where the rod density is highest. The experiment consists of determining the threshold, test spot intensity as a function of steady background intensity.
Over a wide range of intensities the log of the threshold intensity, ΔI, increases linearly with the log of the background intensity, I, the slope of one indicating that the threshold is directly proportional to the intensity of the background. At very low intensities the threshold is independent of background intensity. This absolute threshold in darkness is set by an internal signal similar to background light, known as the dark light.

If the dark light, I₀, is added to the actual background intensity, it allows the form of the entire curve to be explained. This relationship is known as Weber's law. Within the Weber range of intensities, ΔI/I is constant; this ratio is the threshold contrast.

\[
\frac{\Delta I}{I + I_0} = k; \quad \text{If} \quad I \gg I_0 \quad \text{then} \quad \frac{\Delta I}{I} \approx k
\]

The constant threshold contrast corresponds to the sliding scale of intensity described above. When the background becomes very bright, the rod system saturates, which results in a steep increase in threshold with increasing background intensity. Under normal conditions, when the rod system is not artificially isolated by this stimulus, the less sensitive cone system takes over well before rod saturation, and exhibits its own Weber law adaptation.

### 6.3: Photoreceptor adaptation

Rod photoreceptors themselves adapt to light. The left hand panel shows the responses of a mammalian rod to flashes of light of progressively increasing intensity, presented either in darkness or during steady light. During background light the response to a given flash is smaller than in darkness: the rod has adapted to the background according to Weber's law.

As well as becoming less sensitive during steady light, the responses of rods also become faster, as shown in the right hand panel. Thus photoreceptors are able to respond to more rapid changes in bright light than in dim light. Similar light adaptation also takes place in cone photoreceptors.
Photoreceptor light adaptation involves modulation of the transduction mechanism which controls the concentration of cyclic GMP. The absorption of light triggers an enzymatic cascade which culminates in the destruction of cyclic GMP and the resultant closing of channels in the outer segment membrane. These channels normally let both Ca$^{2+}$ and Na$^+$ enter the outer segment. The Ca$^{2+}$ which enters is pumped out again via the sodium-calcium exchanger. When Ca$^{2+}$ influx decreases in the light, this efflux continues for a while, so the cytoplasmic Ca$^{2+}$ concentration falls. This fall in Ca$^{2+}$ concentration causes photoreceptor light adaptation; the most telling evidence is that when it is prevented, light adaptation is abolished also. Ca$^{2+}$ is believed to cause adaptation by acting on the transduction cascade in several ways. First, Ca$^{2+}$ inhibits guanylyl cyclase, which produces cyclic GMP. In darkness, when the Ca$^{2+}$ concentration is relatively high, guanylyl cyclase is partially inhibited. But in the light, when the Ca$^{2+}$ concentration falls, this inhibition is partly removed, allowing more rapid cyclic GMP production and earlier recovery of the light response. Second Ca$^{2+}$ is believed to prolong the activation of photoisomerized rhodopsin, so that its activation switches off more rapidly when the Ca$^{2+}$ concentration falls during illumination. It also affects the cyclic GMP activated channel, and the activation of rhodopsin by light. The increased turnover of cyclic GMP by the phosphodiesterase is also believed to contribute to light adaptation, by allowing changes in cyclic GMP concentration to follow changes in phosphodiesterase activity more closely.

However, the light adaptation of individual rods takes place at intensities which are high in comparison to the Weber-law adaptation of the whole rod system, acting only to prevent premature saturation of the rods in the mesopic range. So the adaptation of the whole rod system which takes place at lower intensities must be due to some other mechanism, as it begins at intensities so low that individual rods only occasionally receive photons.

However, the outputs of individual rods are summed together within the retina through convergence. It is this summed output which provides not only the visual signal, but also the signal for adaptation. Rods are connected together to form adaptation pools, which adaptation of the rod system to take place within the retina, somewhere between the receptors and the ganglion cells. This is demonstrated by the curve shifting of individual ganglion cells as the steady background intensity increases. The precise mechanism by which this gain control in ganglion cells takes place is not known, but it appears to involves changes in the way in which the rod signals are summed together.
6.4: Quantal fluctuation

Light is not continuous, but is composed of individual photons, or quanta. The figure shows the response of a single amphibian rod to a series of dim steady lights of increasing intensity. When the light is extremely dim, the rod only occasionally absorbs a photon, yielding distinct responses. As the light becomes brighter, these individual events merge together, to give a response which displays quantal fluctuations because of the random arrival of individual photons. As the light becomes even brighter, the size of the fluctuations decreases because the larger the number of photons, the smaller the fluctuation in comparison with the mean:

\[
\text{fluctuation} \propto \sqrt{I}; \quad \text{mean} \propto I; \quad \therefore \quad \frac{\text{fluctuation}}{\text{mean}} = \frac{\sqrt{I}}{I} = \frac{1}{\sqrt{I}}
\]

During dim steady light, any stimulus must be detected as being distinct from these random fluctuations. Their role in limiting the sensitivity of the human visual system can be revealed by repeating the increment-threshold experiment with a small stimulus presented for a short time which will thus deliver only a few quanta to a small number of rods. designed to maximise the effect of quantal fluctuations.

At very low intensities, the sensitivity is limited by the dark light, as before. At higher intensities, the curve rises according to Weber's law, and ultimately saturates. But at intermediate intensities, the curve rises more shallowly than Weber's law, with a slope of 0.5 which corresponds to a square root relation between the threshold and the background intensity. This indicates that the response must be bigger than the fluctuation in order to be detected. Even in darkness the rod system is not free of fluctuations, as rhodopsin can spontaneously isomerize due to thermal agitation. This results in one spontaneous isomerization every couple of minutes in any given rod. These spontaneous quantal events are believed to give rise to the dark light, which sets the absolute threshold for rod vision.
6.5: Dark adaptation

If a human subject views a very bright light and then returns to darkness, visual sensitivity is greatly decreased. This decrease in sensitivity does not depend simply on the intensity of the bright light, but instead on the total amount of photopigment bleached during the light exposure, and is therefore known as bleaching adaptation. On the return to darkness, sensitivity recovers slowly in the process of dark adaptation.

If a large white stimulus is used to test sensitivity, both rods and cones are stimulated and sensitivity recovers in two stages. If instead a small red stimulus is used to stimulate only the foveal cones, then only the first component of the recovery is seen. In a rod monochromat, who completely lacks retinal cones, the first component of recovery is absent. So the first component is due to the rapid recovery of cones, and the second due to the slower recovery of the rods; the division between these two phases is known as the rod-cone break.

During dark adaptation the spectral sensitivity of vision progressively changes in the Purkinje shift. Before the rod-cone break, vision relies on middle and long wavelength cones and the threshold is lowest at about 550 nm. But later when the rods take over, the wavelength of peak sensitivity changes to the peak wavelength of rhodopsin at about 500 nm.

Following exposure to intense light bleached rhodopsin is gradually regenerated. By the time of the rod-cone break, even though the regeneration of rhodopsin is more than 90% complete, the rod system is still desensitised by several hundred fold: threshold rises approximately exponentially with the fraction of pigment bleached.
The elevation of threshold after bleaching affects the rod system in much the same way as an increase in the dark-light. Bleaching directly desensitises photoreceptors. This bleaching desensitisation is believed to involve persistent excitation of the phototransduction mechanism, which reduces cytoplasmic Ca²⁺ concentration as in light adaptation.

Bleaching also results in an increased rate of spontaneous quantal events in each rod. Such post-bleach noise has been seen in amphibian rods after bleaching a few percent of the rhodopsin, and will limit the detection of the responses to dim flashes as described above.

6.6: Retinal reorganization during dark adaptation

During dark adaptation, there is a progressive change in the neural pathways followed by the photoreceptor signals within the mammalian retina. In the photopic range, only the cones function and the rods are fully saturated. The cone signals pass through the cone circuit via the on and off cone bipolars. In the mesopic range, both rods and cones are in use, and both rod and cone signals pass through the cone pathway. The rod signals gain access to the cone circuit via their electrical coupling to the cone terminals. In the scotopic range, at the end of dark adaptation, the rods are disconnected from the cone pathway by the uncoupling of their gap junctions to the cone terminals and the rod circuit takes over, running via the rod bipolar to the AII amacrine cell, and finally via the terminals of the cone bipolars to the ganglion cells. The synaptic actions of the AII amacrine are turned on at the end of dark adaptation by the action of the A18 amacrine, which releases the neurotransmitter dopamine. In mammals this rod bipolar pathway lacks the antagonistic horizontal cell surround of the cone pathway. So the ganglion cell receptive fields in the fully dark-adapted retina also lack a surround, having only the receptive field centre.
Changing illumination has profound consequences for the spatial acuity of the visual system. At low intensities, acuity is low over the entire retina, as the high-convergence rod pathway is used. But as the light intensity increases, the foveal acuity improves dramatically as its tightly-packed densely sampled cones are brought into play. So high spatial resolution requires light intensities sufficient to adequately stimulate the cone system.

Changing illumination also profoundly affects the temporal properties of vision, which can be assessed using the critical fusion frequency: the frequency above which a flickering light is perceived as steady. At low intensities vision depends on the rods, which are best stimulated by blue-green light. Rod responses are, however, quite slow, giving rise to a low flicker fusion frequency which never exceeds 15 Hz. At higher intensities the cones take over, and exhibit far better temporal resolution. At high intensities at which the cone system responds most rapidly, the flicker fusion frequency approaches 60 Hz. If at low intensities a long wavelength stimulus is used which barely stimulates rods, only the contribution of the cone system is seen. These observations show that as the mean intensity increases, the visual system becomes progressively better at following fast changes.