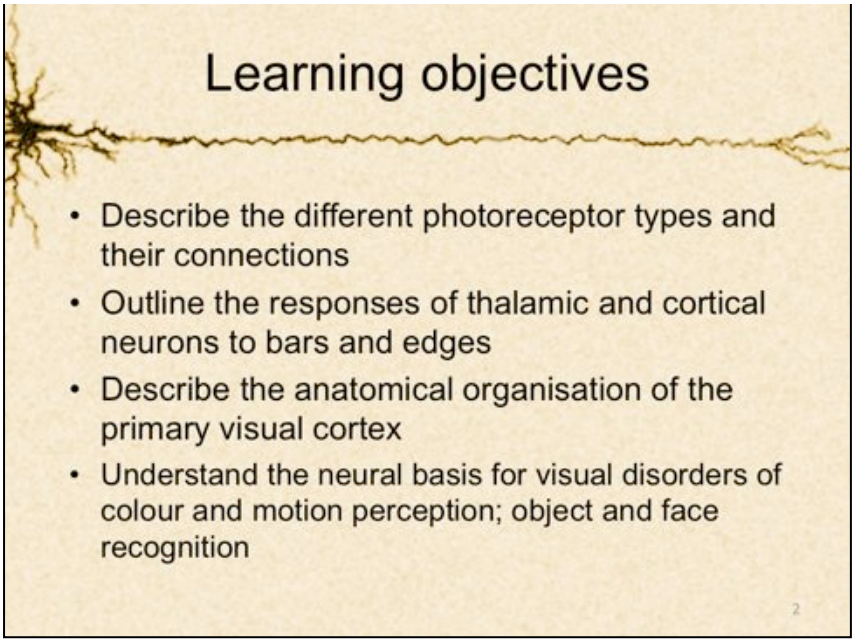
Mind, Brain & Behaviour 1
Behavioural Neuroscience

The human visual system


Learning objectives
• Describe the different photoreceptor types and their connections
• Outline the responses of thalamic and cortical neurons to bars and edges
• Describe the anatomical organisation of the primary visual cortex
• Understand the neural basis for visual disorders of colour and motion perception; object and face recognition
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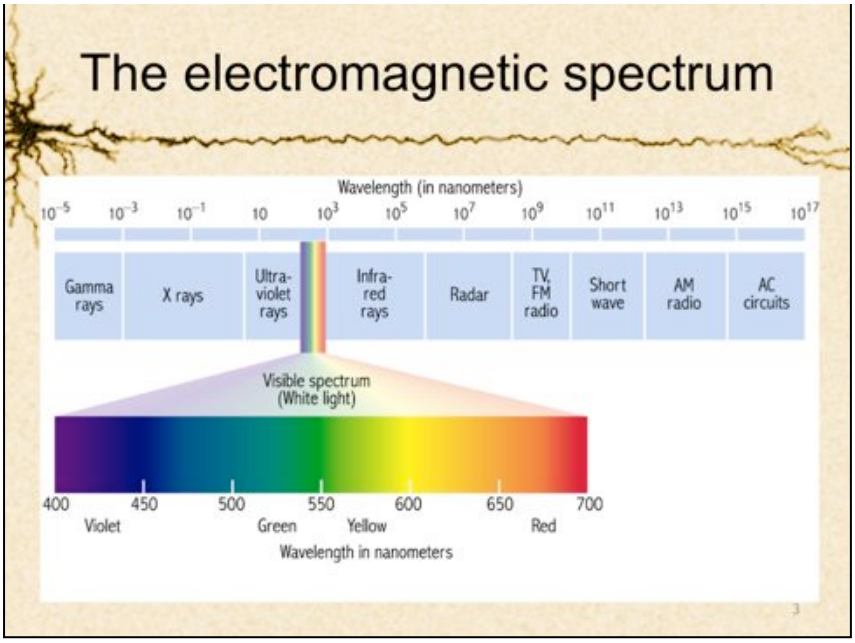


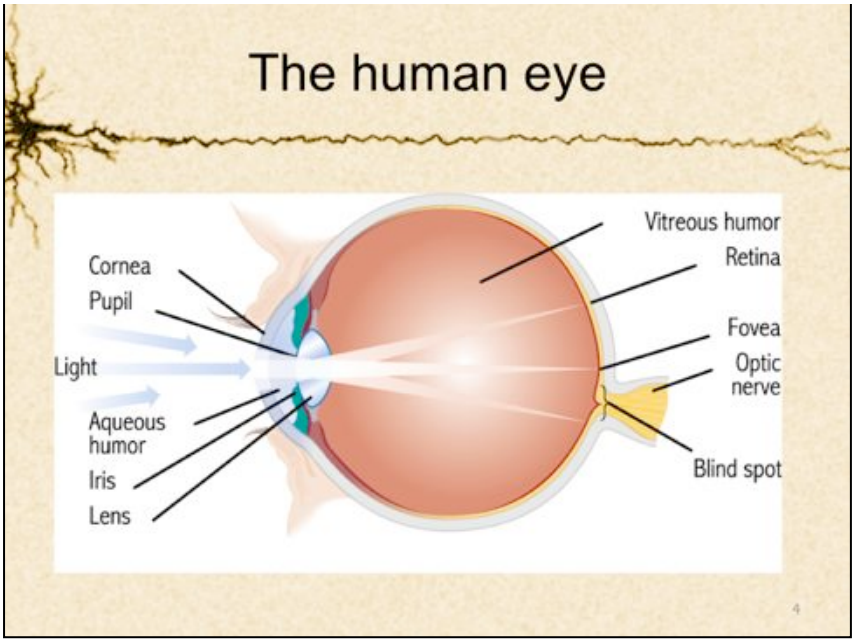
Our eyes detect the presence and pattern of light reflected off objects in the world. We are sensitive to a very narrow range of wavelengths in the **electromagnetic spectrum**, known as the **visible spectrum** (i.e., white light, or daylight). The visible spectrum extends from 380 nanometres (billionths of a metre) to 760 nm.

By contrast, honeybees can detect light within the ultraviolet range.

The range of wavelengths we can see is not qualitatively different from the rest of the electromagnetic spectrum; it's just the part of the continuum of electromagnetic radiation that we are sensitive to.

The **colour** of light is determined by three dimensions:

1) **Hue** – the wavelength of electromagnetic radiation
2) **Brightness** – the intensity of electromagnetic radiation
3) **Saturation** – the purity of electromagnetic radiation

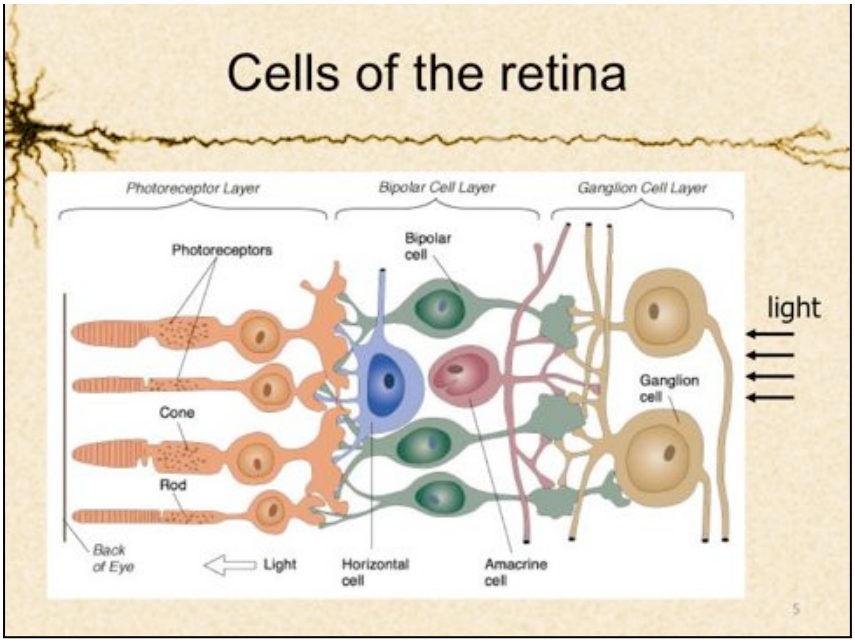


The eye contains the peripheral apparatus necessary for transducing (transferring) light into a neural signal.

Light enters the eye through the transparent outer layer known as the **cornea**. Immediately behind the cornea is the **lens**, which is made up of a number of transparent layers, much like an onion. The shape of the lens can be altered to help focus the image onto the back of the eye, which is lined by a light sensitive structure called the **retina**. The eyeball itself is filled with a clear gelatinous fluid called the **vitreous humour**.

Note that the light sensitive cells in the retina (the **rods** and **cones**) send their axons out of the eye from a common point, known as the **optic disk**. Because there are no photoreceptors at the optic disk, it causes a **blindspot** (i.e., the region of space from which an object is not visible). The axons that are bundled together at the optic disk are known collectively as the **optic nerve**.

Our primary concern in this lecture is with the structure and function of the light sensitive retina, and with the manner in which neural signals from the retina are elaborated by the rest of the brain to allow perception.



A closer view of a cross-section through the light sensitive retina reveals a series of layers, each containing specialised neurons, their axons and dendrites, and the photoreceptors (the retina is in fact part of the brain).

The light sensitive (**photosensitive**) cells are located at the **back** of the retina, so light must pass through each of the other layers to get to them. There are two types of these **photoreceptors**: **rods** and **cones**. The rods and cones contain **photopigments**. These pigments break down when exposed to light, and this breakdown process triggers a series of stages that leads to the neural impulses that are eventually conveyed to the brain by the optic nerve.

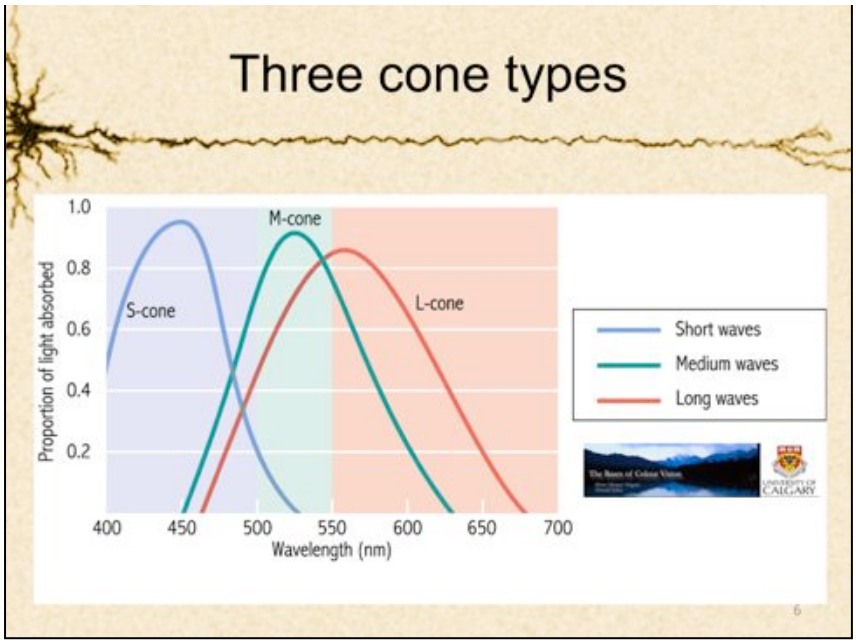
The human retina has about 120 million rods and about 6 million cones. Even though there are fewer cones, these are the most important for seeing fine detail, and they are most active in the daylight. Cones are concentrated in a region of the retina called the **fovea**, which is responsible for the central few degrees of our visual field. Different types of cones are also sensitive to different wavelengths of light, and so they are responsible for our ability to see colour. Rods do not discriminate between different wavelengths, and they cannot discriminate fine visual detail. But rods are much more sensitive to light than cones, and so rods are used in dimly illuminated environments (hence our failure to perceive colour or fine detail in semi-darkness).

The retina can be divided into three distinct layers:

1) Photoreceptor layer
2) Bipolar cell layer
3) Ganglion cell layer

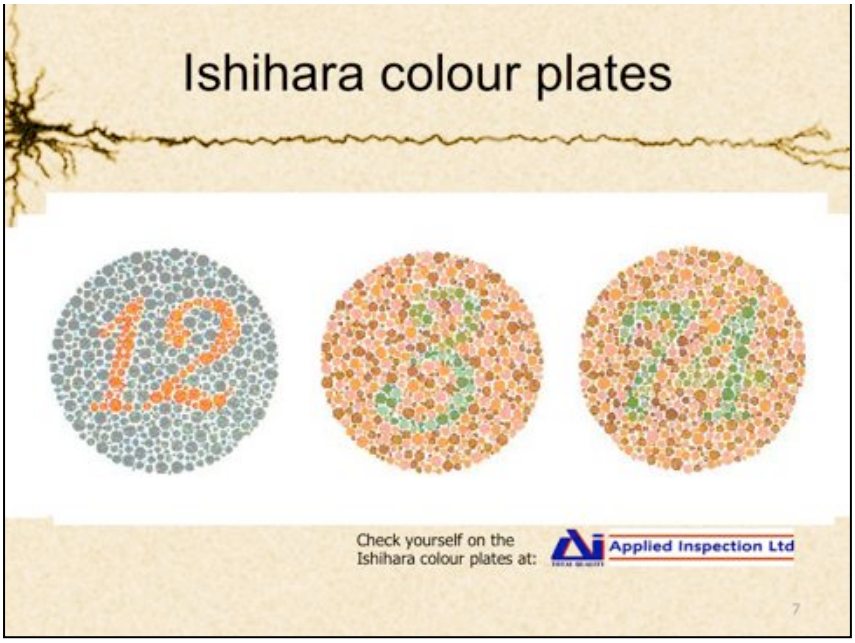
The rods and cones form synapses with **bipolar cells**, which in turn form synapses with **ganglion cells**. Ganglion cells send their axons through the optic nerve (the second cranial nerve), which conveys visual information to the brain. Two other cell types in the middle layer of the retina, **horizontal cells** and **amacrine cells**, serve the function of combining messages from several photoreceptors.

Note: Photoreceptors and bipolar cells do not produce action potentials. Rather, they release neurotransmitters that increase or decrease the firing rate of action potentials generated by the ganglion cells.



As already outlined, there are three types of cones, each containing a photopigment that is sensitive to a different range of wavelengths within the visible spectrum.

1) **Short-wavelength (S) cones** – peak sensitivity at 440 nm (blue light)
2) **Medium-wavelength (M) cones** – peak sensitivity at 530 nm (green light)
3) **Long - wavelength (L) cones** – peak sensitivity at 560 nm (red light)



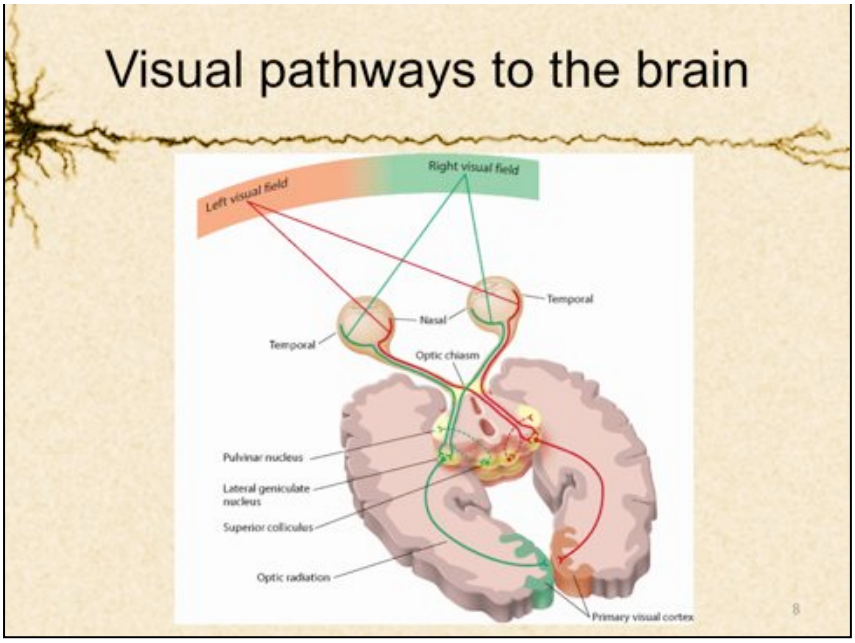
Different types of **colour blindness**, a genetic condition, arise from anomalies in the pigments of one or more cone types in the retina. The two most common forms of colour blindness are more common in males than females because the responsible gene is located on the X chromosome. Males have just one X chromosome and so the defective gene is expressed. Females have a pair of X chromosomes, one of which is likely to have a normal gene that can mask the expression of the defective one.

In fact most people with colour blindness are not literally 'blind' to colour. They still see the world in colour, but they are deficient in discriminating between certain hues. The most common kind of colour deficiency is one in which a person is poor at discriminating red and green (**'red-green deficient'** – this affects around 10% of males and about 1% of females).

People who are colour deficient have anomalies in the **photo-pigments** of one or more of the three cone-types (S, M or L).

The **Ishihara Colour Plates** are used to test anomalies of colour perception. The disk on the left of the slide contains a digit that can be seen by both normals and those with colour deficiencies. The central and right disks contain digits that can be seen by normals; individuals with red-green deficiency may see an incorrect digit; others with anomalous colour vision may not see any digits at all.

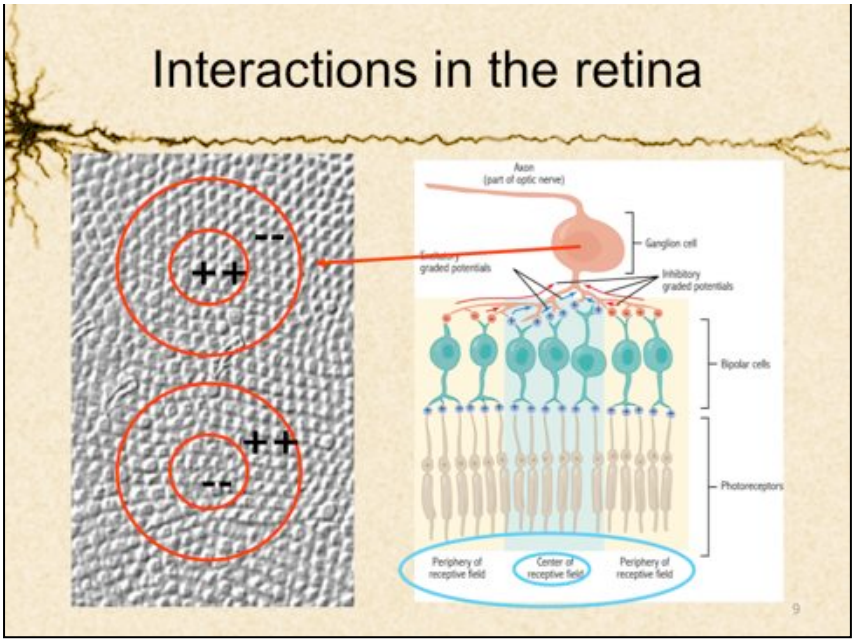
Colour deficiency is not uncommon, especially in males, and is no cause for alarm. Other than an unusual taste in coordinating the colours of their clothes, there are few (if any) everyday problems for most people with anomalous colour vision.



After leaving the eye the axons of retinal ganglion cells are bundled together to form the **optic nerves** (one for each eye). These project posteriorly and medially toward the **optic chiasm**. Here, roughly half the axons from the retina of each eye cross over to the opposite side of the brain. Axons from the temporal half of the retina of the right eye remain on the same side, but axons from the nasal half cross over to the left hemisphere. Similarly, axons from the temporal half of the retina of the left eye remain on the same side and axons from the nasal half cross over. This arrangement means that visual information from the right visual field is conveyed exclusively to visual areas in the left hemisphere, whereas visual information from the left visual field is conveyed exclusively to the right hemisphere.

Beyond the optic chiasm, axons in the optic tracts continue posteriorly until they form synapses with neurons in a part of the thalamus called the **lateral geniculate nucleus** (**LGN**; there is one in each hemisphere). Neurons in the LGN send their axons posteriorly where they form synapses with neurons in the **primary visual cortex**. Note that about 90% of LGN axons terminate in the primary visual cortex. The remaining 10% project to other areas, including the **superior colliculus** (part of the midbrain) and **pulvinar nucleus of the thalamus**.

Note once again that the primary visual cortex in each hemisphere represents visual information from the contralateral half of the visual field (NOT from the contralateral eye).

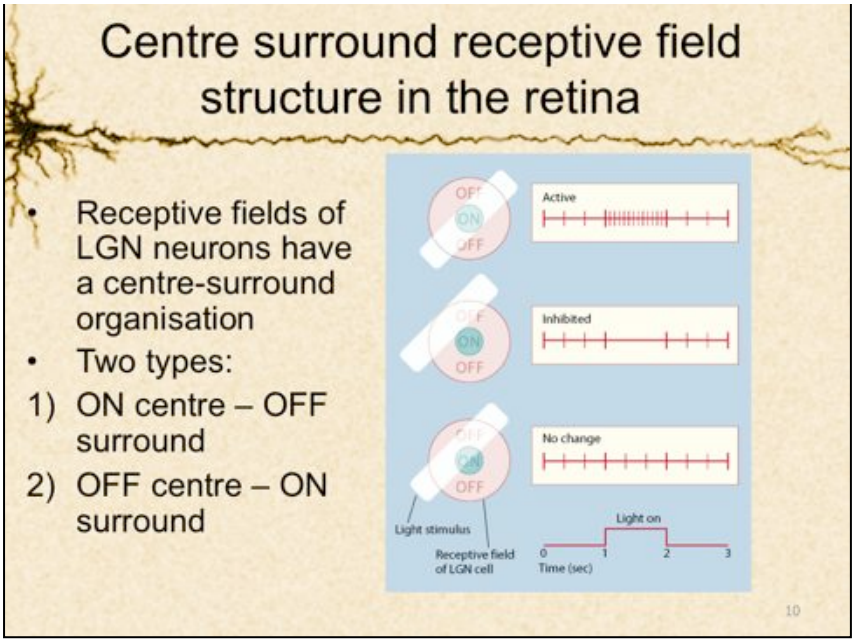


To appreciate how neurons in the brain encode visual information from the eye, it is necessary to understand how the photoreceptors, bipolar cells and ganglion cells interact in the retina.

There is a convergence of information from the photoreceptors, through the bipolar cells to the ganglion cells. The responses of many photoreceptors are combined to influence the response of a single ganglion cell.

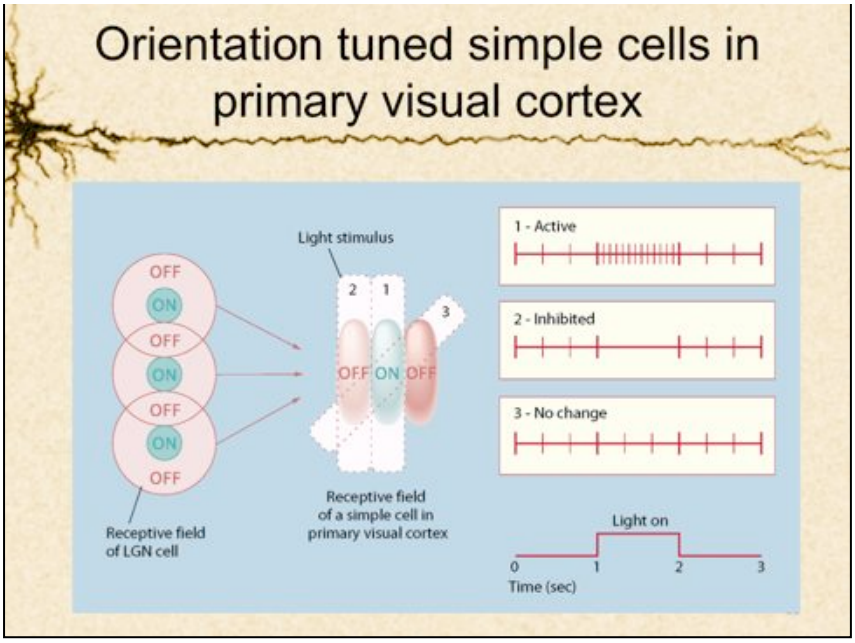
Each ganglion cell responds to signals from one small cluster of photoreceptors, which in turn are excited by light from one small region of the visual field (this is known as the cell's **receptive field**). The photoreceptors exert an excitatory influence on the bipolar cells with which they form connections. Adjacent bipolar cells can then have either an excitatory or inhibitory influence on a single ganglion cell.

Receptive fields of ganglion cells tend to have a **centre-surround organisation** (like a two-dimensional doughnut). ON cells are excited by light in the centre of their receptive field, and inhibited by light in their surrounding field. In contrast, OFF cells are inhibited by light in the centre of their receptive field, and excited by light in their surrounding field. These two simple types of receptive field provide a powerful mechanism for signalling edges and borders in the visual world, which in turn forms the basis for higher level visual recognition.

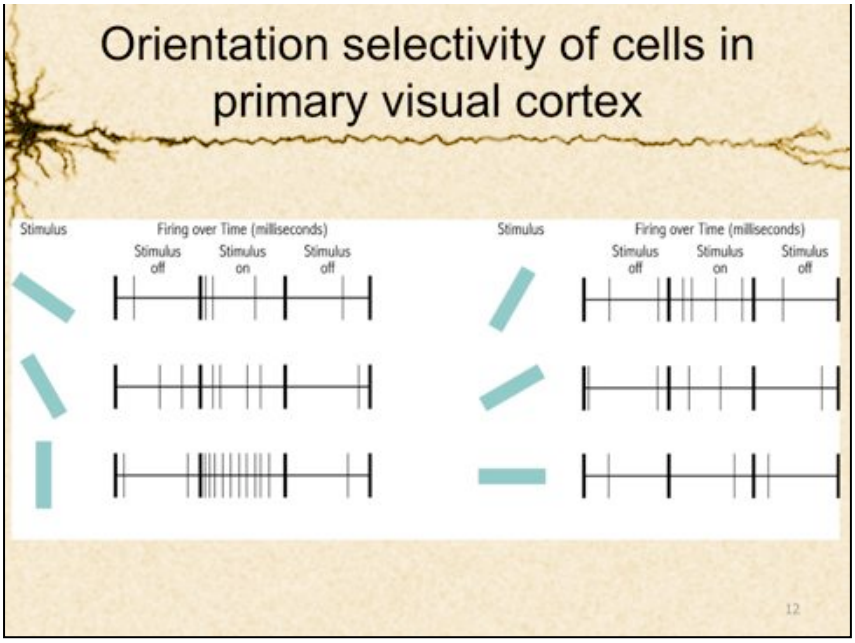


Neurons in the lateral geniculate nucleus, with which retinal ganglion cells form synapses, also have receptive fields with a centre-surround organisation.

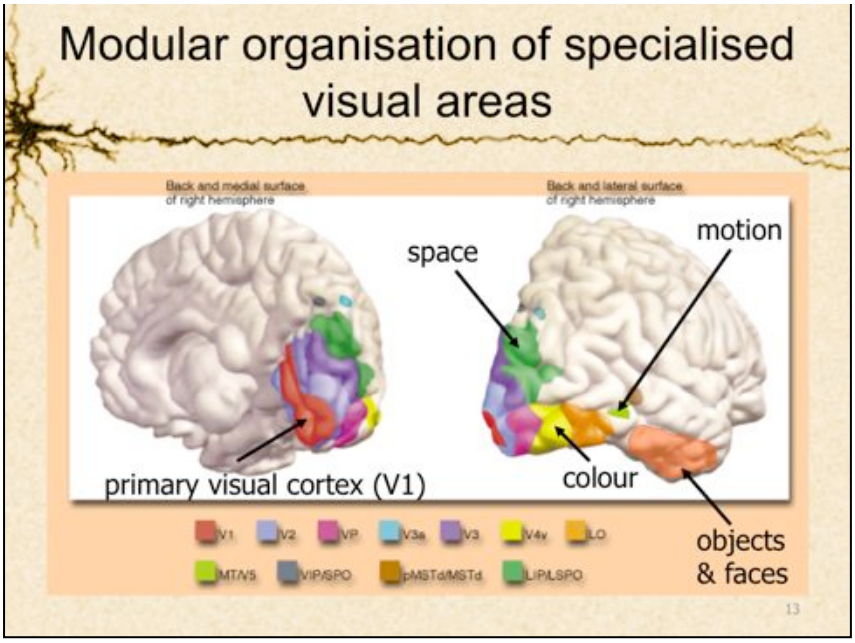
A small bar of light passing over the ON region of an LGN cell's receptive field will increase the firing rate of the neuron; a bar that passes over the OFF region alone will decrease the firing rate; and a bar that only partially passes over the ON region will not alter the neuron's rate of firing relative to its level of spontaneous activity.



Neurons in the primary visual cortex known as **simple cells** receive inputs from several LGN neurons with adjacent receptive fields. Such simple cells signal not only the presence of a bar or edge, but also its **orientation**. A simple cell is either excited or inhibited by a bar that follows the orientation of the ON and OFF regions of its receptive field; its rate of firing remains unchanged for a bar that is oriented across both ON and OFF regions.



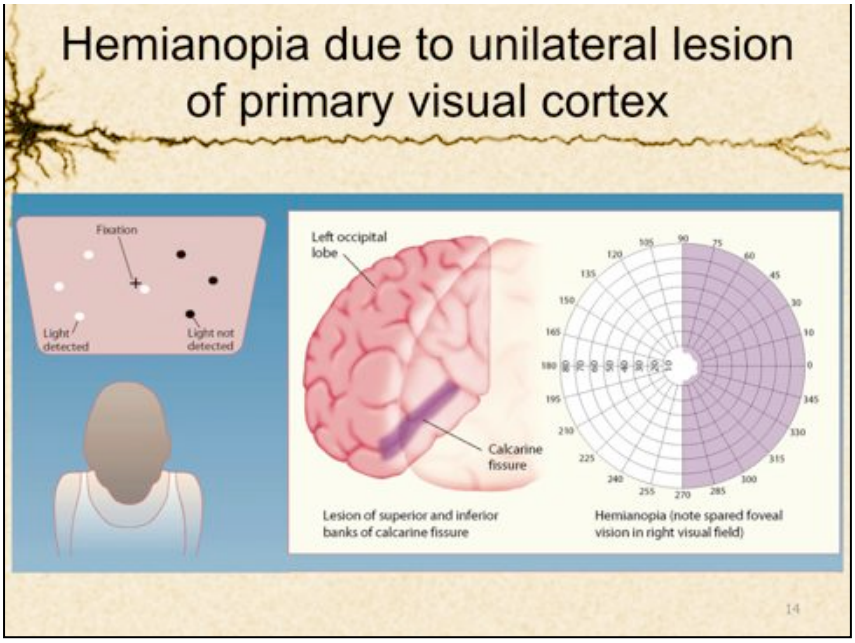
Simple cells in the primary visual cortex are said to have an **orientation preference**, which is to say that their receptive field organisation is such that each cell will fire preferentially to bars and edges of one particular orientation (e.g., vertical, horizontal, 45° clockwise from vertical, etc.).



The primary visual cortex is divided into several **functional modules** or subregions, each of which contains neurons that have specialised properties for extracting specific information from the visual input.

We shall consider just a few of these areas in this lecture:

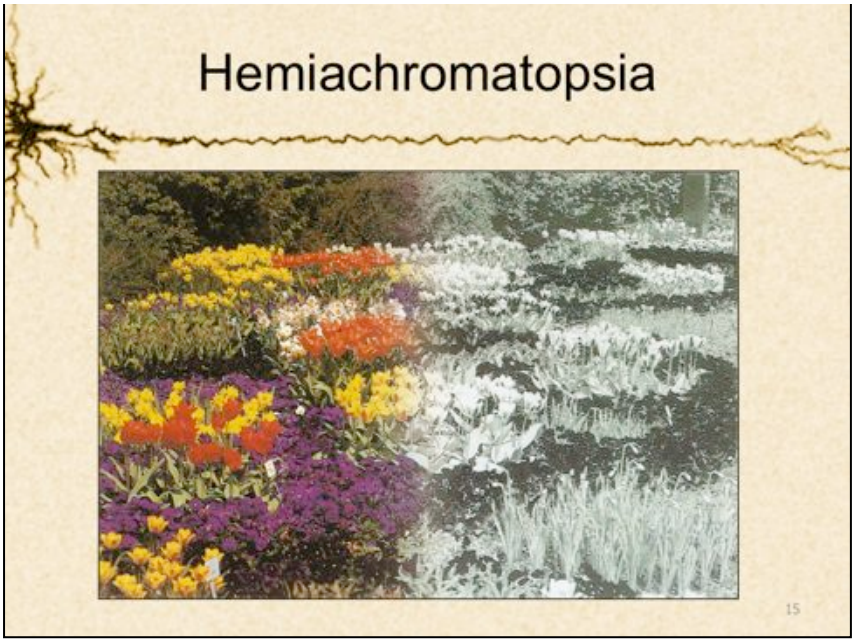
1) Primary visual cortex (also known as the first visual area, or V1)
2) Area V4, which has neurons that are sensitive to the colour of visual inputs
3) Area MT, which is responsive to moving visual stimuli
4) Inferior temporal cortex, which contains neurons that are selectively responsive to complex objects and faces



The primary visual cortex (also known as **visual area 1**, or **V1**) is the first cortical region to receive axons from visual cells in the lateral geniculate nucleus. Area V1 in each hemisphere contains a retinotopic map of the contralateral half of the visual field.

Damage to area V1 can occur after a stroke affecting one of the major arteries at the back of the brain, the **posterior cerebral artery**. Following rupture or occlusion, the neurons supplied by the posterior cerebral artery are starved of oxygen and glucose, and so are irreversibly damaged. If area V1 is affected, the patient will become blind to all visual stimuli arising to the contralateral side of their present point of fixation, e.g., damage to V1 in the right hemisphere will cause blindness in the left visual field. This disorder is called a **hemianopia** (loss of vision for one side).

Patients with a hemianopia are normally aware of their visual loss and will take active steps to compensate for the problem, e.g., by directing exploratory head and eye movements toward the affected side in an effort to bring unseen parts of the environment into the normally sighted half of their visual field.

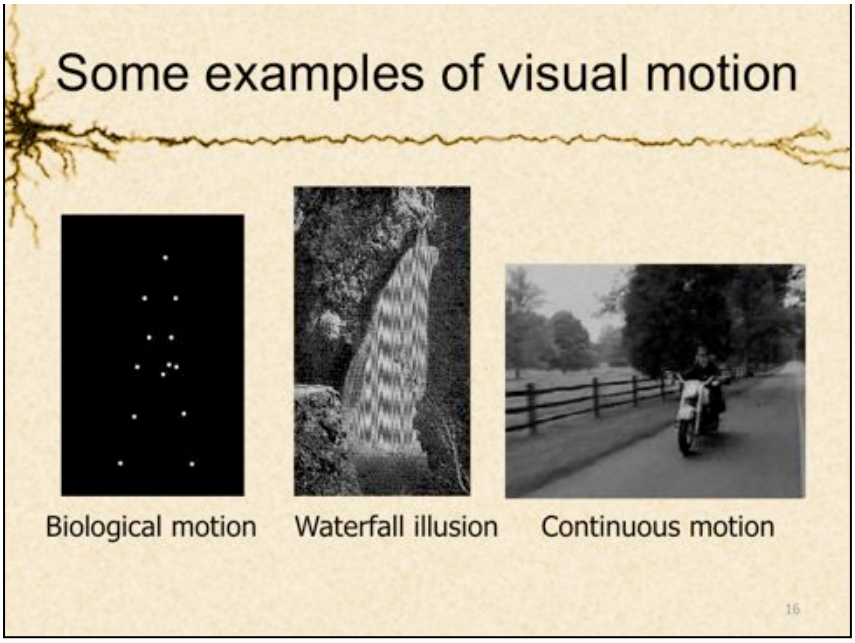


Patients with damage that involves an area of visual cortex called V4 are no longer able to perceive colour in the contralateral visual field. Interestingly, these individuals continue to see forms and movement, so they are not completely blind. Instead, one half of the world looks like a black and white movie, while the other is in technicolour!

The loss of colour vision that accompanies damage to visual area V4 is called **achromatopsia** (vision without colour). The loss of colour vision in isolation, without any corresponding impairment of other visual abilities, suggests that area V4 is specialised for processing the wavelength of visual stimuli. This has been supported by brain imaging studies in normal individuals that have shown that area V4 is selectively active when participants view brightly coloured stimuli, compared with a baseline stimulus that is matched for shape and luminance but that is **achromatic** (without colour).

[Note that achromatopsia should not be confused with deficiencies of colour vision arising from anomalies in the photo-pigments of cones in the retina (‘colour blindness’ – see Slide 8 from this lecture)].

Patients with pure achromatopsia are very rare. Why? Because the strokes that cause brain damage typically affect large areas of cerebral tissue, and almost never damage just one small functional area in isolation. Area V4 is adjacent to several other visual areas that are involved in processing the shape, and in fact V4 neurons themselves are also sensitive to orientation. Therefore in virtually all cases of achromatopsia careful testing often reveals other subtle visual losses in the region of space in which colour vision has been lost. But the fact remains that a small lesion in area V4 impairs colour vision more than any other visual attribute, and confirms the important role of area V4 in normal colour vision.

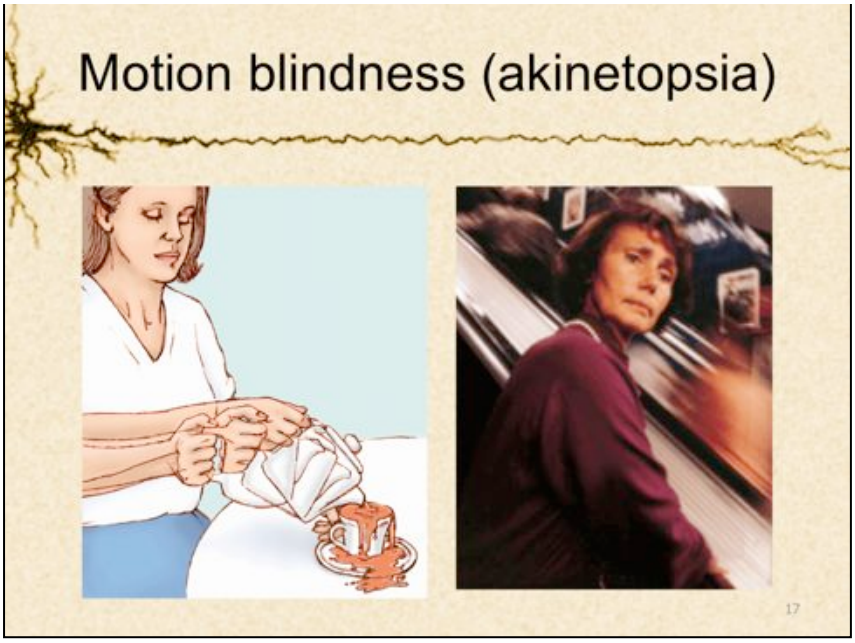


Colour is just one of the visual attributes we take for granted in our perception of the visual world. Another is our ability to perceive visual motion. Psychologists have spent more than 100 years trying to understand the mechanisms underlying motion perception.

We can use motion to help us to discriminate form and structure. Consider the example of **biological motion**, in which point light sources placed at the joints of the limbs are sufficient to permit us to perceive a person walking.

The visual neurons that are sensitive to visual motion may gradually adapt when exposed to a continuously moving stimulus, so that when the motion ceases a **motion aftereffect** occurs (as in the classic **waterfall illusion**). The effect does not occur due to adaptation of cells in the retina, since adapting one eye to the moving stimulus and then using the other eye to view a stationary surface still yields a motion aftereffect.

Some simple manipulations can trick the motion system into interpreting displacement of a single visual image in space as **continuous motion**.

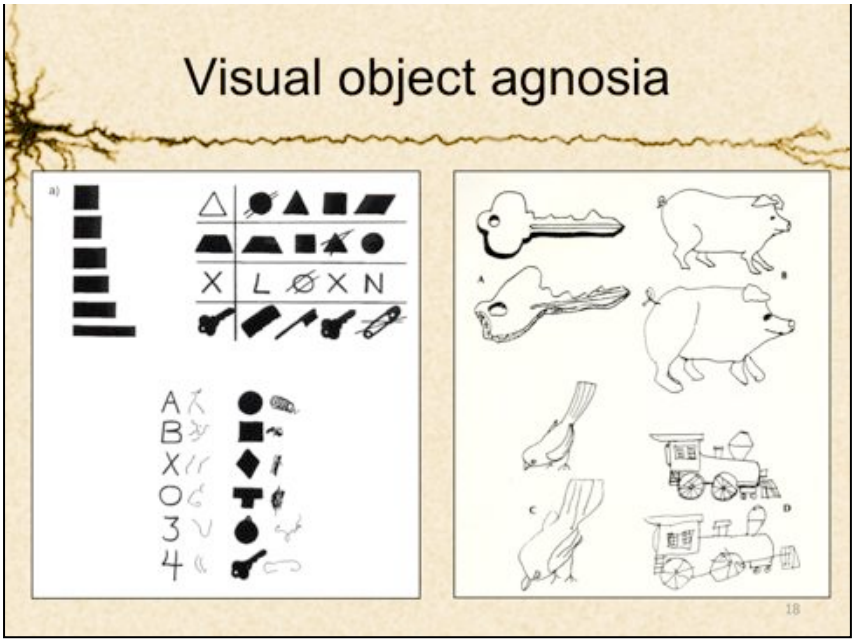


What would it be like to lose our ability to perceive motion, but retain our ability to perceive shapes and colours?

Even though it is exceedingly rare, there have been a few patients reported who have lost their ability to perceive visual motion. The condition, known as **akinetopsia**, occurs when area MT is damaged in both hemispheres, but in which regions responsible for colour and form vision remain intact.

In a famous report, Joseph Zihl and his colleagues in Munich reported the case of a woman, whose initials are LM, who lost her ability to perceive visual motion after damage of area MT bilaterally. To her, moving visual stimuli appeared as a succession of 'snapshots', similar to what we perceive when viewing the world under stroboscopic lighting, as is often found in nightclubs. When pouring a cup of tea LM would see the liquid frozen in mid-air, like a glacier. The tea would rise in her cup but she would fail to notice its progression until it had overflowed onto the table. LM became reluctant to cross the street. As she noted 'When I'm looking at the car first, it seems far away. But then when I want to cross the road, suddenly the car seems very near'.

Intriguingly, a recent study found that LM is able to perceive **structure from motion** in biological motion displays. She can recognise people depicted solely by moving points of light even though she cannot perceive the movements themselves. This findings suggests that area MT is not involved in perceiving structure from motion. Other patients with damage in a medial part of the occipital lobe have been reported that show the opposite pattern: they can perceive movement but cannot see structure from motion. This dissociation between the pattern of impairment shown by LM and that shown by the patients with medial occipital damage imply that perception of motion and perception of structure from motion involve different regions of the visual association cortex.



We have already seen that certain brain lesions can selectively impair colour and motion perception. In keeping with the modular organisation of the visual system, lesions elsewhere in the visual association cortex can impair the ability to recognise familiar objects, but leave motion and colour vision intact.

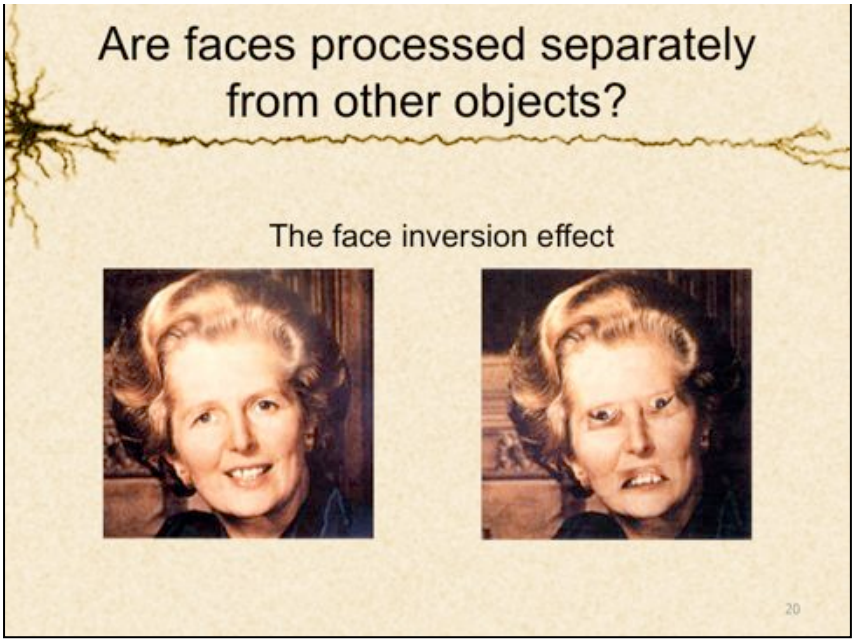
Selective loss of the ability to recognise familiar objects through the modality of vision is called **visual object agnosia**. Crucially, patients with this condition are still able to recognise objects through other sensory modalities, such as hearing and touch, indicating that they have not lost information about what the objects are called, what they mean and what they are used for. For example, a patient with visual object agnosia who cannot recognise a bunch of keys visually will immediately recognise them by the sound of them jangling, or by how they feel when held.

Visual object agnosia typically occurs after unilateral or bilateral damage of the inferior part of the temporal cortex. Functional brain imaging studies have revealed that the region of the temporal cortex that is damaged in visual object agnosia is selectively active in normal participants when they view pictures of familiar objects, compared with a baseline condition consisting of meaningless fragments or nonsense shapes.

Many patients with visual object agnosia have quite basic problems with visual recognition. They may not even be able to discriminate between two different shaped rectangles or find a simple geometric shape amongst displays containing a set of different shapes. Other patients may perform normally on these simple tasks, and may even be able to copy or draw quite complex visual objects, and yet not know what they are.

Perhaps one of the most common 'objects' we are required to recognise on a daily basis is the human face. In many respects faces are unique. They convey crucial information about a person's identity, their gender, their current emotions, and even perhaps their intentions.

We often recognise a face long before we can retrieve the person's name, and occasionally we can recognise people's faces without knowing where we know them from. We are also prone to see faces in otherwise apparently random textures or clusters of objects (e.g., in the paintings of fruit by the 16th century Italian artists Giuseppe Arcimbaldo).

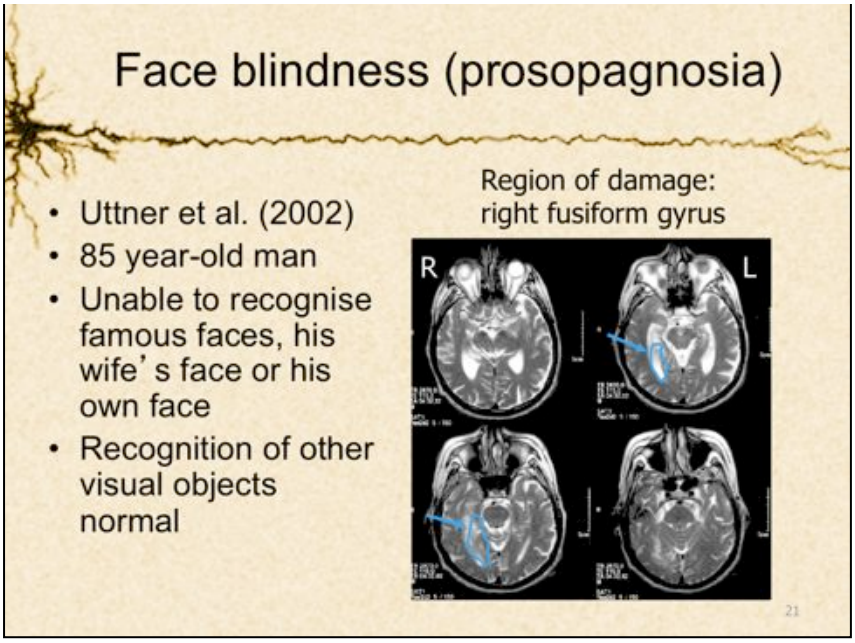


What evidence is there that faces are processed any differently to other objects?

There are some simple illustrations of how face processing might be different from the processing of other object classes. For instance, people are readily able to learn to recognise pictures of faces they have never seen before, but they have difficulty learning to recognise pictures of faces when they are turned upside-down. By contrast, learning to recognise pictures of other objects (e.g., houses) is not much different whether they are shown upright or upside-down. This is called the **face inversion effect**.

Another example of how face processing is special comes from the **Thatcher Illusion**. We fail to notice even quite striking visual anomalies when a face is shown upside-down.

It has been suggested that the brain has specialised visual areas devoted to processing the subtle differences in configuration of the eyes, eyebrows, mouth, nose, chin and so on – all the features that go together to make a particular face unique. When a face is inverted these processes can no longer operate because the configuration between parts is altered (e.g., the eyebrows are now below the eyes rather than above them).

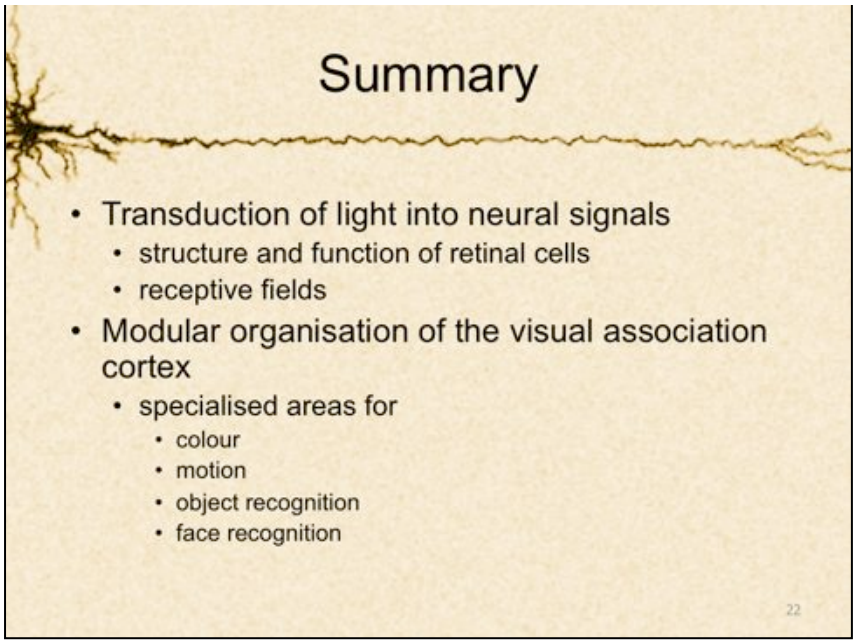


Several studies have suggested that a region of the inferior part of the temporal lobe called the **fusiform gyrus** is particularly important for face recognition.

Human patients with damage of the fusiform gyrus, either bilaterally or in the right hemisphere alone, may have a selective impairment in recognising familiar faces, even though their ability to recognise other objects may still be relatively good. A problem in recognising familiar faces is called **prosopagnosia**.

Note that as is the case with visual object agnosia, in prosopagnosia the sufferer does not have a problem with person knowledge – an individual who is not recognised by his or her face may be recognised by voice or some other distinguishing visual feature, such as glasses or a necklace.

In severe cases of prosopagnosia the patient will not even recognise siblings or spouses, and may even fail to recognise their own reflection in a mirror.


Summary
• Transduction of light into neural signals
• structure and function of retinal cells
• receptive fields
• Modular organisation of the visual association cortex
• specialised areas for
• colour
• motion
• object recognition
• face recognition
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