

(Human) Network Analysis

Lecture 2: **(Deep) Learning and processing in biological neurons and networks**

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In today's class, we will move away from artificial networks for machine learning to look at the structures that inspired them: networks of biological neurons learning about the world to guide our behaviour.

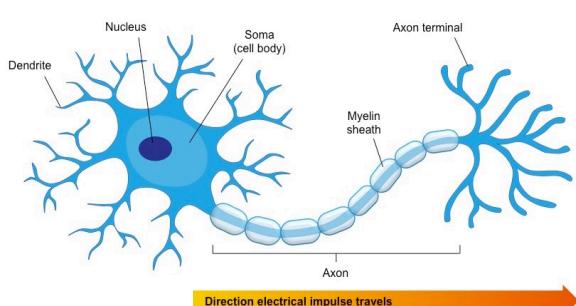
This is quite different from the content covered in our last class: it is the most important topic in biology for AI, but we know many of you have no background in biology. For those with CS backgrounds, who found the last class basic knowledge, the biological content in the next 45 minutes is probably the most challenging aspect of the course. So...

Why study biological networks?

- Inspiration for many AI networks, including DCNNs
- AI networks simplify processes involved for efficiency
- AI deep networks not yet as advanced as human brain
- Where the goal is to imitate human behaviour, following the neural mechanisms more closely may help
- Major link between AI and other sciences
- Links AI to biological sciences, not just math/CS
- Potential to link social sciences to biological sciences
- Leading model of neural computations

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A biological neuron



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A neuron is a specialised type of animal cell.

Most aspects work like other cells and are not important for performing computations.

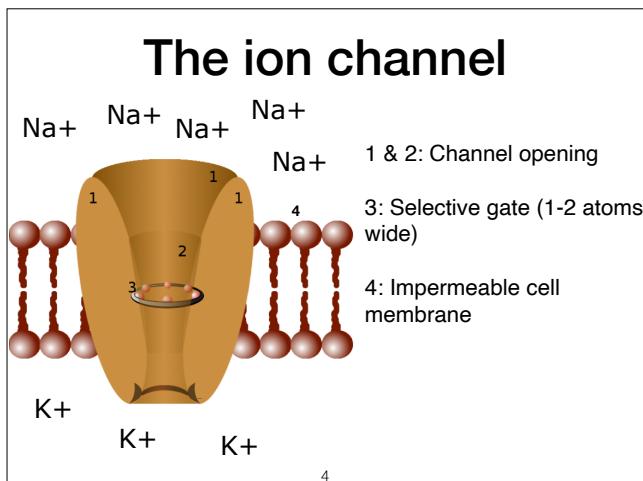
But the cell membrane in neurons is highly unusual. It is specialised for performing simple computations using electrical activity.

First, the dendrites integrating electrical signals coming in from other neurons than it is connected to.

These connections, or synapses, between dendrites and other neurons vary in strength, and change strength depending on past activity, so synaptic strengths are the biological equivalent of artificial neural network weights.

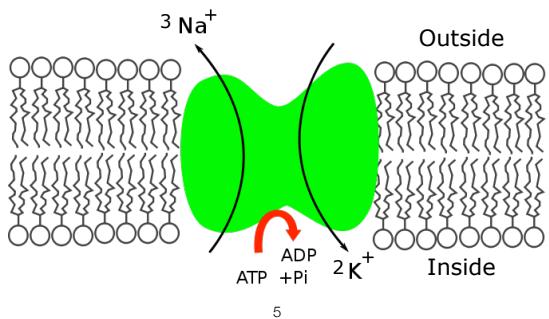
We will see that the biological equivalent of a convolutional filter is a tree of

dendrites working together to sample information from other neurons. If these combined incoming electrical signals are strong enough to pass a THRESHOLD, the neuron will then send an electrical impulse down the axon. On reaching the axon terminals, this is passed on to other cells, in the next LAYER of neurons.



The basic computational component of a biological neuron is a protein called an ion channel.
 -This can allow charged atoms, or ions, of sodium and potassium (natrium and kalium) to pass into and out of a neuron, but they are normally closed, blocking ion passage.
 -Ion channels are imbedded in a cell membrane, which surrounds the neuron.
 -This also does not allow ions to pass through.
 -As ions can't pass through the membrane if the ion channel is closed, the cell membrane has an imbalance of ions on inside and out: far more sodium ions outside the cell (top), a few more potassium ions inside (bottom).
 -This imbalance of electrically-charged ions causes a voltage difference across the cell membrane.
 -But the ion channel protein can change shape, allowing a particular type of ion to pass through: there are different ion channels for sodium and potassium ions.
 -When these ions cross the cell membrane, this changes the membrane voltage, or membrane potential.
 -Note that the ion channel is a passive mechanism, it does not pump the ions against their concentration gradient, it only opens to allow ions to diffuse down the concentration gradient or closes to prevent this.

The sodium/potassium pump: establishing the resting potential



Because the ion channel is passive, it only allows ions to move towards lower concentrations or to overcome voltage differences. This is like pulling a plug out of a bath, allowing the water to flow out as quickly as the hole allow.

-This is very fast, much faster than pumping ions around to change voltages. That would be like pumping water in to or out of the bath, much slower.

-But the ion imbalance must be maintained by an active mechanism (requiring energy). The bath must be filled before the plug hole can let water .

-This pump is another protein imbedded in the cell membrane

-Because of more sodium being transported out, the inside of the cell is negatively charged compared to the outside.

-In the cell membrane's rest state, there is imbalance of ions & charges across the cell membrane, making a voltage of -70 mV.

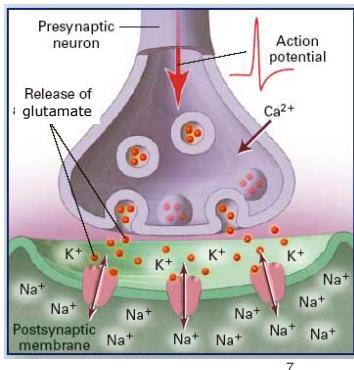
-This is known as a resting potential

So...

- The cell membranes of neurons are specialised for electrical computations.
- Ions are atoms with electrical charges
- Cell membranes do not allow ions through
- Sodium/potassium pumps establish an imbalance of ions across the membrane
 - Much more sodium outside
 - A little more potassium inside
- Opening ion channels allows these ions to pass through, in the opposite direction, towards balance

If you are having trouble with understanding this, there is a great introductory video here:
<https://www.youtube.com/watch?v=oa6rvUJlg7o>
This explains the basics really slowly.
Then come back to this class.

Opening the ion channel



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So opening sodium ion channels causes sodium ions to enter the cell through the cell membrane, changing the membrane voltage or membrane potential.

-This may lead the cell to activate (or 'fire'), and the amount of this activation is what is simulated by the activation values in the feature maps of an artificial convolutional neural network.

-For our purposes, there are two important ways an ion channel can open.

-First, it can open because a neurotransmitter binds to the ion channel. In that case, the ion channel is a 'receptor' for the neurotransmitter.

-When the neurotransmitter binds, the ion channel protein changes shape, opening the ion channel for ions to cross the membrane.

-Another neuron firing causes the neurotransmitter release into a synapse, the gap between two linked neurons.

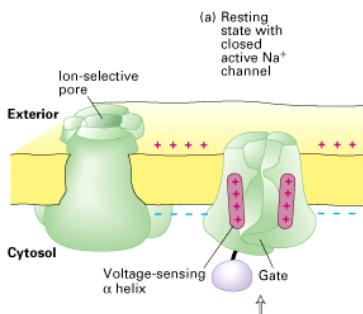
-So the neurotransmitter is a signal released by activation of one layer of a neural network (the presynaptic neuron) and causes activation of the next layer of the neural network (the postsynaptic neuron)

-Here we will look at excitation of the postsynaptic neuron by binding of glutamate, and inhibition by binding of gamma-amino butyric acid (GABA).

-Using both excitation and inhibition allows connections with both positive and negative weights, as convolutional filters have.

-Other neurotransmission works by similar mechanisms, but glutamate and GABA are by far the most common.

Opening the ion channel



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The second way an ion channel can open is due to a change in membrane voltage. This is called a voltage-gated ion channel.

-Here, the voltage across the membrane has been changed by other ion channels opening.

-This change in membrane voltage changes the shape of the voltage-gated ion channel, opening it and allowing more ions to pass and the membrane voltage to change more.

-After a lot of ions have passed and the voltage has changed a lot, the ion channel is closed in a different way: the outer segment moves into the channel's gate.

-Then, over a longer time, pumping of sodium and potassium returns the

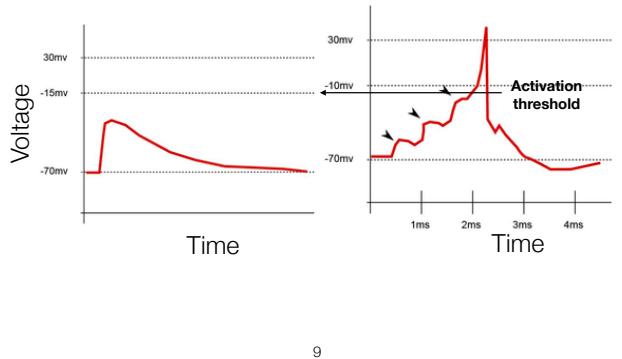
membrane voltage to its resting potential, the

The voltage-gated ion channel is particularly important because opening the ion channel also changes the membrane voltage.

-So changing membrane voltage can lead to further changes in membrane voltage.

-In this way, the voltage-gated ion channel acts very much like an electronic transistor, that is an electrical switch that opens and closes because of an electrical input. Miniaturised transistor circuits are the basis of all computer processors.

Excitatory post-synaptic potentials (EPSPs)



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-When glutamate binds to a post-synaptic receptor, it causes the receptor's ion channel to open, and sodium to enter the cell.

-This causes a change in the membrane voltage, called an excitatory post-synaptic potential.

-The ions become more balanced across the cell membrane. The size of the voltage difference across the cell membrane decreases, so we say the cell membrane is depolarised.

-But over time, this depolarisation decreases as sodium and potassium are pumped around to return the membrane to its resting potential.

-So this one neurotransmitter molecule binding causes no activity on the postsynaptic neuron, because it doesn't reach a threshold that activates the voltage-gated ion channels. Then, the neuron doesn't pass this event on, and it has no further effect.

-However, if several neurotransmitters molecules bind over a short period, this can reach the **threshold voltage** for the voltage-gated sodium channels.

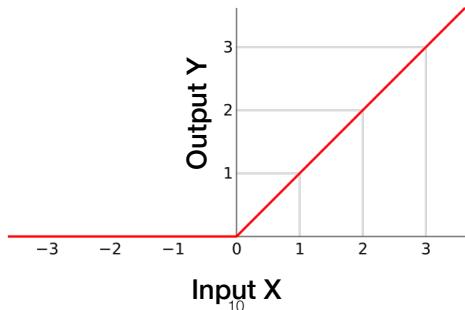
-This causes a much more extreme depolarization, a spike of voltage or action potential. This will be passed down the axon to the next layer of neurons.

-So this threshold for activation of voltage-gated sodium channels is the biological equivalent of the threshold/rectification operation we saw in artificial networks.

The threshold/rectification operation

an activation function using a rectified linear unit (ReLU)

$$Y = f(X) = \max(0, X)$$

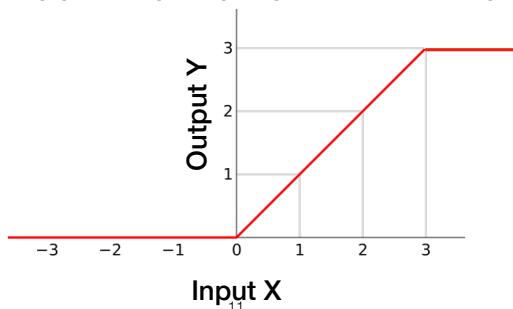


- Until we reach a certain threshold voltage, there will be no response.
- This threshold is not zero in biological neurons, it's around -15mV because an imbalance of ions is maintained at rest to speed up neuron responses.
- Then the firing rate (output) of the neuron will increase as the strength of the inputs increases.

The threshold/rectification operation

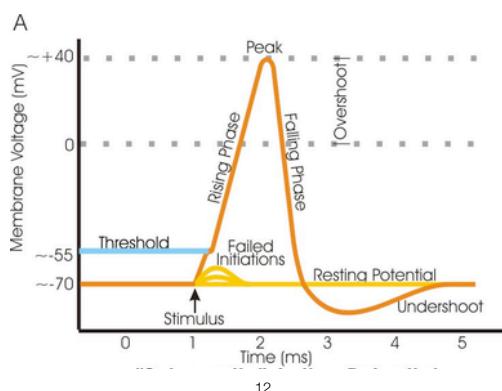
an activation function using a rectified linear unit (ReLU)

$$Y = f(X) = \min(\max(0, X), \text{MaxActivation})$$



And at some point this output will reach a maximum firing rate

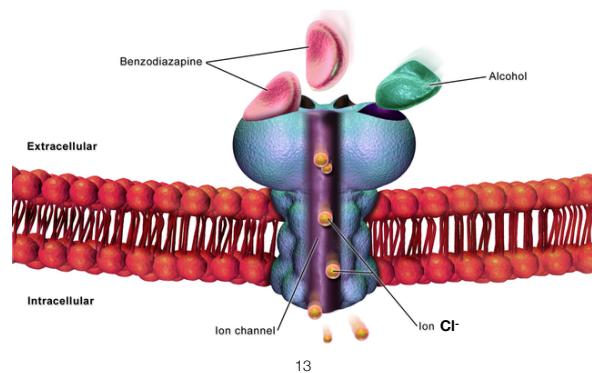
Action potentials



- The reason for this maximum firing rate is that an action potential takes time, although not very much time.
- At the peak, Na^+ concentration is almost equal across the cell membrane
 - The high voltage then causes the Na^+ channels to close. This is a different closed state than before, as the channel protein is a different shape.
 - Here, the Na^+ channel cannot open again until the cell is repolarised.
 - The initial repolarisation (falling phase) relies on K^+ channels opening, which repolarises faster than pumping, but the Na^+ concentration is too high inside for further diffusion, and the K^+

concentration is too low inside. So opening the Na^+ channels again will do nothing. The Na^+/K^+ pump needs to return the cell to resting potential.

Inhibitory post-synaptic potentials (IPSPs)



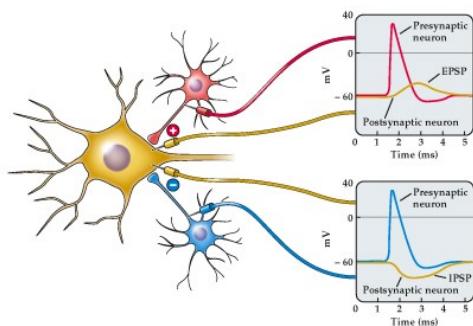
Activity in a presynaptic neuron can also inhibit the postsynaptic cell firing, by causing the membrane to polarise further, making the membrane voltage more negative. We call this state **hyperpolarized**: the membrane potential difference is greater, specifically **more negative** than it was before.

-The GABA receptor is also an ion channel, but for negative chloride (Cl^-) ions. When GABA binds, Cl^- enters and the cell membrane potential becomes more negative than the resting potential.

-This pushes the cell further away from activating.

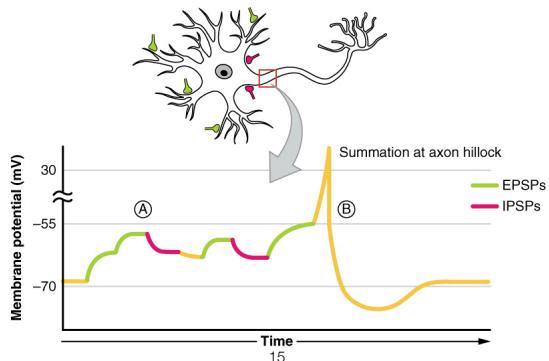
-Alcohol and benzodiazepine drugs like valium also bind to the GABA

Inhibitory post-synaptic potentials (IPSPs)



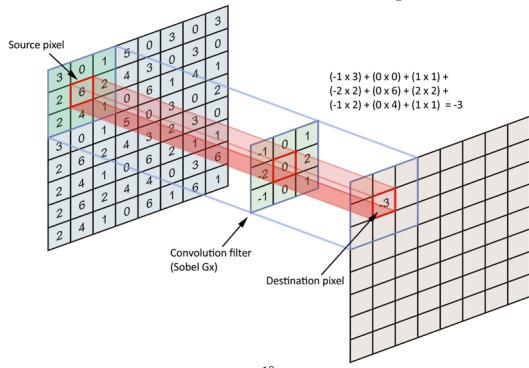
So we have both positive and negative inputs onto the same postsynaptic neuron

Inhibitory post-synaptic potentials (IPSPs)



These can be from a range of different places, and from a number of different neurons. However, there is a limited spatial distribution of inputs because the dendritic tree has a limited size. Which may sound very familiar...

The filter/convolve operation

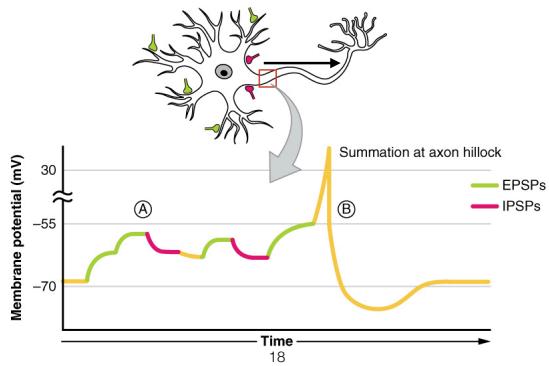


Because convolutional network filters imitate this structure: they have positive and negative filters, which are multiplied by the activity on a spatially limited group of presynaptic units to determine the activity of the postsynaptic unit

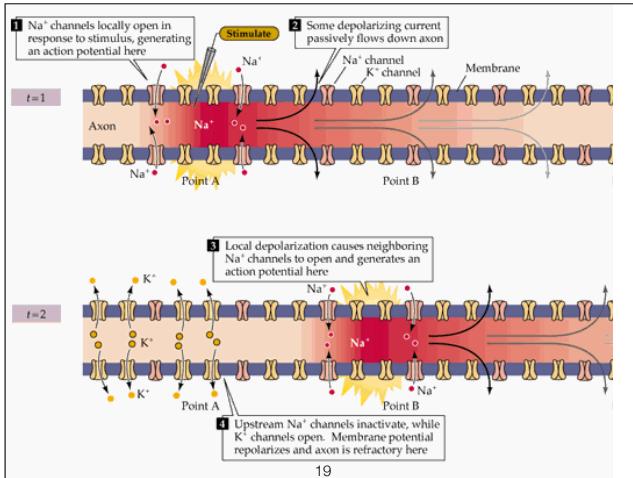
So...

- Neurotransmitters (e.g. glutamate and GABA) released from a pre-synaptic cell can excite (depolarise) or inhibit (hyperpolarise) activity in the post-synaptic neuron
- This relies on ligand-gated (i.e. neurotransmitter activated) ion channels
- If membrane polarisation reaches a threshold, voltage-gated ion channels open
 - Results from many excitatory inputs
 - Strongly depolarises (fires) the neuron: Action potential

Action potentials

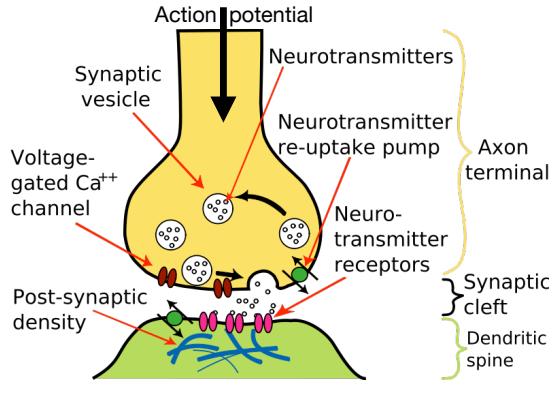


Once an action potential has been triggered, it travels from the input end of the neuron, the postsynaptic dendrites, along the length of the the neuron's main fibre, the axon, to provide inputs to the next layer, which is often in a different brain area some distance away.



- This process also relies on voltage-gated sodium channels.
- The depolarisation at Point A spreads to neighbouring locations to push their membrane potential above threshold. This causes voltage gated ion channels at this neighbouring location to open, leading to Na^+ entry and depolarisation at point B. This opens further voltage gated ion channels even further along (further right), causing Na^+ enter here.
- So the depolarisation spreads down the axon like a wire.
- Note this can only go in one direction. After the action potential wave has passed, the membrane will be depolarised and there won't be a suitable concentration difference in Na^+ ions across the membrane. Also the ion channels will be closed by their outer segment, rather then having their gate close by the resting membrane potential.

Neurotransmitter release



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- On reaching the presynaptic terminal for the synapse to the next layer of processing, the action potential leads to neurotransmitter release.
- At rest, neurotransmitters are stored ready for use, in membrane bubbles called vesicles.
- The arriving action potential causes voltage gated calcium ions (Ca^{++}) channels to open, allowing calcium ions to flow into the cell.
- Ca^{++} binds to proteins in the axon terminal to change their shape, bringing the vesicles to the synaptic surface and releasing the neurotransmitter to activate the next postsynaptic neuron.

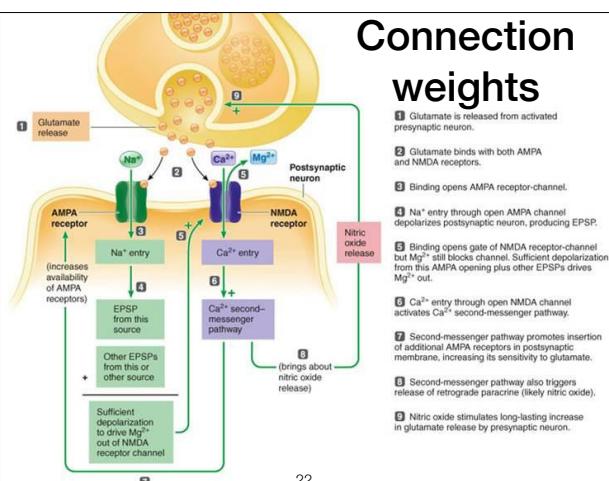
Weights in biological neurons

- Connection weights in biological systems don't depend on backpropagation of error
 - We are not trained by a supervised process
 - There is no 'correct' response in how we learn about the world
 - However, there can be adaptive and maladaptive responses/behaviours
- Instead, we learn mainly by unsupervised processes
 - To recognise patterns of activity we have seen before
 - To learn the statistics of the world we live in
- Long-term potentiation
 - Lasting enhancement of synaptic connections by co-activation of presynaptic and postsynaptic neurons
 - Hebb's postulate: 'Cells that fire together, wire together'

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- The Skinner box is an apparatus used in animal experiments to deliver rewards (food) and punishments (electric shocks through the floor) when an animal performs (or fails to perform) certain behaviours. This trains the animal and changes its behaviour.
- Conditioned responses like these those learned from rewards and punishments are unlikely to work through backpropagation-like mechanisms affecting the whole network.
- They are more likely to work at a single decision stage, where the final decision is associated with a reward or fear response. This is enough to modify behaviour.

Connection weights



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So, how does firing together lead to wiring together?

-Postsynaptic neural activity modifies synapse structure at both the presynaptic and postsynaptic terminal through a variety of mechanisms.

-These increase presynaptic neurotransmitter concentration and release, and the density of (active) postsynaptic receptors for the neurotransmitter.

Here are some example mechanisms.

AT END: Another important mechanism is an increase in the transcription of receptor genes in the cell nucleus, as cell polarisation affects the activity of gene transcription enzymes.

-Mechanisms like this seem very important in learning and memory, and seem to occur at all synapses.

-Several common drugs interfere with these processes. Alcohol and benzodiazepines like valium reduce spiking activity by activating GABA receptors and reducing the probability of depolarisation. This reduces memory formation,

so these sedative drugs have a tendency to cause amnesia.
-Ketamine blocks the NMDA glutamate receptor, a vital step in strengthening weights. Ketamine is therefore very effective at inducing amnesia.

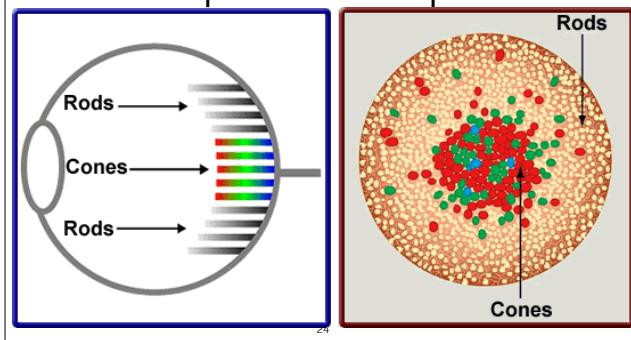
The story so far

- Network layers: layers of neurons at different levels of synapses
- Feature map: ?
- Filter: integration of EPSPs and IPSPs across the dendrite tree
- Threshold: activation of voltage-gated Na^+ channels by above-threshold depolarisation by EPSPs
- Pool: ?
- Normalise: ?
- Learning mechanism:
 - Unsupervised: 'cells that fire together, wire together' (Hebbian learning)
 - Activity-dependent changes in synapse structure/strength (weights)

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AT END: So the weights on filters result primarily from learning.
-However, some filters appear hard-wired in early stages of vision. These seem to arise from evolution rather than learning, suggesting they provide a useful structure that subsequent layers can build on.
-In artificial deep networks for vision, the filters are the first level and are often hard-wired edge detectors.
-Understanding these early filters gives an idea of which feature maps arise in biological vision, and how they are used by subsequent stages.
-Understanding this is important because the pooling and normalisation stages depend on the structure of these feature maps.
-So, let's look at some biological feature maps, and how those pool and normalise information.
-All of these structures and operations don't rely on a single neuron, but a larger group of neurons. So now we will look at larger structures containing large numbers of neurons.

Input feature maps: The photoreceptors



The eye has four different sensors for light: three types of colour-sensitive cones sensor and one type of rod sensor.

-Rods don't carry colour information, but respond quickly and under low light conditions. Their output also gives good information about fast events like moving objects.

-Together, these form the input image to the visual processing network, and are essentially already four feature maps.

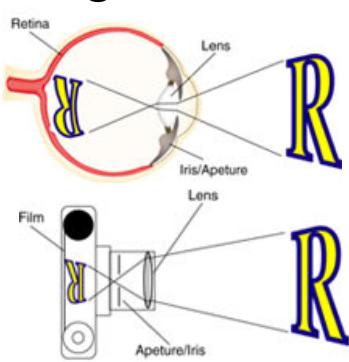
-Unlike in a camera that usually provides the input to an artificial network, these light sensors are not evenly distributed across the image.

-In central vision ('where we are looking') we only find cones, while further out we find mostly rods.

-The overall density of photoreceptors drops off with the distance from central vision. So vision strongly over-emphasises the centre, where we are looking.

-This greatly reduces the computational load on the brain, while still giving high detail in central vision.

Image inversion

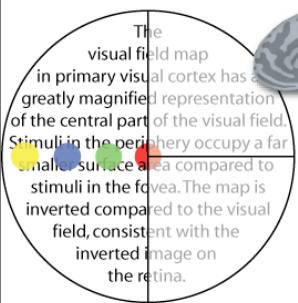


The brain's representation of visual space is also flipped up-down and left-right.

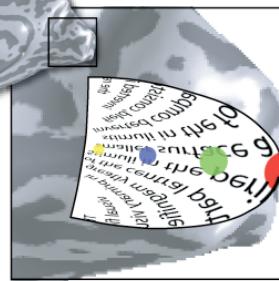
-This begins in the retinal projection of the image because rays of light are passing through a small hole at the pupil, much like an image is inverted in a camera sensor.

Cortical visual field representation

Visual field



Visual field representation in the brain (V1)



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This over-representation of central vision and this image inversion continue into the cortex, where the visual image is 'mapped' onto the cortical surface.

-You may think that this strange representation of vision would cause problems for perception.

-However, the location of information on the retina or cortex is not important when we think of the visual cortex as a set of feature maps, neural representations of image transformations, rather than thinking of this as an image. As I mentioned last time, the activity in a feature map can be visualised into an image, but it is very misleading to think of a network layer's activity as an image.

Cortical map representations

- Spatially map the structure of the sensory organ onto the cortex
 - Maintain spatial relationships
 - Allow analysis of spatially-restricted patterns
- Magnify the representation of important parts of sensory space
 - Central vision, hands and face, vocal auditory frequencies
 - Balances detailed analysis with computational load
- We can change what we sample in detail
 - Move our hands and eyes
 - Attention increases processing of attended parts
- Artificial networks for vision process the whole image in detail
 - Normally, a human has decided where to point the camera

-But it is very important that neighbouring locations are represented next to each other so that the spatial extent of a filter represents a continuous piece of the input image.

-This is particularly important when we consider that the dendrites of the next neuron will sample from this layer, and have a limited extent too. This seems to be the main reason why the brain maintains spatial relationships at each level of processing.

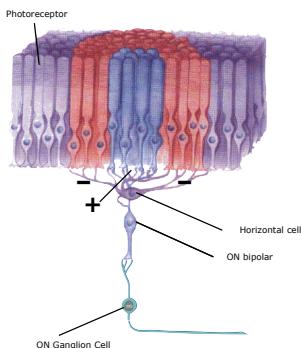
-So we process central vision in more detail. Processing the whole image input in similar detail would be computationally intensive for the brain.

-Artificial sensory systems generally don't use such distorted inputs, aiming instead to process everything in great detail.

-The main advantage is that the cameras that provide the to artificial networks don't need to move to sample the important parts of the image in detail, like our eyes do. Normally, the inputs are static images, so this isn't an option anyway.

-It will take considerable advances in artificial deep networks before they can process input images immediately, decide where the important details are, and move the sensors to sample those details.

Spatial comparison filters: surround suppression



0	-1	-1	-1	-1	-1	0
-1	-1	-1	-1	-1	-1	-1
-1	-1	7	7	-1	-1	
-1	-1	7	7	-1	-1	
-1	-1	-1	-1	-1	-1	
0	-1	-1	-1	-1	-1	0

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Now let's look at the first filtering stages in the eye: how is incoming image first transformed?

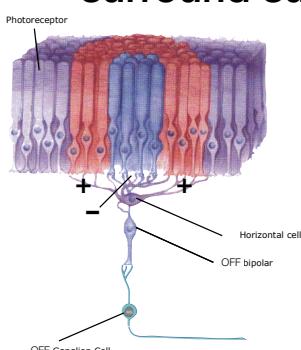
-The retina analyses the relationships between nearby locations, comparing the responses of a group of neighbouring photoreceptor cells, all linked in to its tree of dendrites.

-Those in the centre of the tree produce EPSPs, while those at the edges of the tree produce IPSPs. So activation at the edges inhibits responses to activation at the centre.

-As a result, a point of light at the centre produces a stronger response than a field of light covering both the centre and the surround. CLICK-Expressed as a convolutional filter, it might look something like this. If we have a bar of equal intensity (+1) running through the centre of the filter, we get a positive response (+20). A point of light will at the centre only will produce a larger positive response (+28). A field of light everywhere will produce zero response.

-Just like a convolutional filter, there are overlapping copies of this cell throughout the retina. So a photoreceptor that falls in the negative zone of one horizontal cell filter, also falls in the positive zone of another.

Spatial comparison filters: surround suppression



0	1	1	1	1	1	0
1	1	1	1	1	1	1
1	1	-7	-7	1	1	
1	1	-7	-7	1	1	
1	1	1	1	1	1	1
0	1	1	1	1	1	0

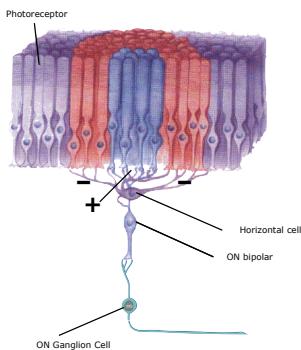
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-A complementary filter is operating in parallel, responding to darkness at the centre and suppressed by darkness in the surround.

-This is called an 'off' response, a response to no light. This might sound unnecessary, but features are often darker than the background, like the text on this page.

-The corresponding convolutional filter might look like this, if we express darkness as negative, so darkness -1 multiplied by filter value -7 gives +7 activation. Darkness -1 multiplied by filter +1 gives -1 activation.

Spatial comparison filters: surround suppression

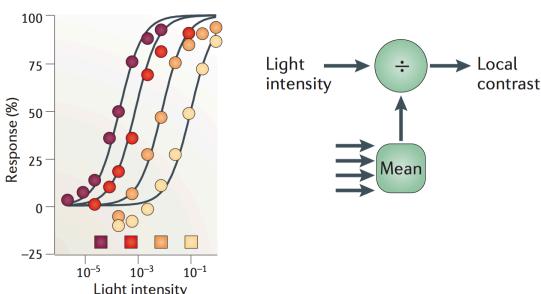


0	-1	-1	-1	-1	-1	0
-1	-1	-1	-1	-1	-1	-1
-1	-1	7	7	-1	-1	
-1	-1	7	7	-1	-1	
-1	-1	-1	-1	-1	-1	
0	-1	-1	-1	-1	-1	0

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- This analysis effectively converts the amount of light at each location to the amount of contrast: how much the amount of light changes around this location.
- Contrast is necessary in this description because a full field of light or darkness produces no response.
- By transforming the representation of brightness in the rods and cones into a representation of contrast, most of the image can be discarded, the parts that contain no change.
- But the changes in the image are maintained. This keeps all the useful information, but greatly compresses it for transmission down the small optic nerve to the brain.
- More generally, the visual system responds to changes rather than constant inputs. These spatially-specific 'changes' can be thought of as 'features'.
- An artificial convolutional network filter is also detecting contrast when the mean of the convolutional filter is zero. -Any area of the input feature map that has no change in activation (i.e. no contrast) will activate all the weights in the filter by the same amount, making zero activation regardless of the level of activation of the input feature map.
- So activation of an output feature map reflects contrast or structure in the input, specifically structure that is consistent with the structure of the filter weights.

Surround suppression and normalisation

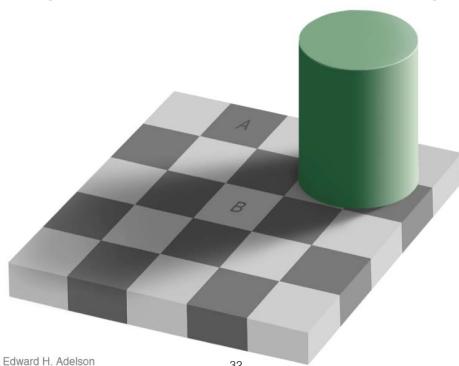


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- As a result of this surround suppression, the response to light intensity inside the positive centre area is normalised by the light intensity outside the receptive field.
- The light level in the surround is used to normalise the light level in the receptive field, yielding local contrast.
- Immediately, nearby post-synaptic cells start inhibiting each other too, so that they are all inhibited more when they are all responding more.
- These operations, surround suppression of the inputs and lateral inhibition of the outputs, closely resembles the normalisation operation of deep networks.
- However, this mean light level used here is probably not taken over the whole image: there is a limitation to the distance that the neuron can be connected to.
- Furthermore, the mean light level is probably restricted to a single feature map.
- It's unclear whether these differences are

better or worse than global normalisation. They allow more complex patterns of normalisation, which might be useful if an image has light parts and dark parts, but are limited in spatial extent.

Perception of likely object brightness, not retinal light

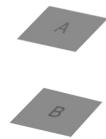


Edward H. Adelson

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Our perception of brightness is therefore strongly affected by the brightness of the immediately surrounding area. The main benefit of this is that we perceive the object's likely real-world colour regardless of how it is illuminated

Perception of likely object brightness, not retinal light



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Removing the context makes this clear, but it is very hard to see in the original image

The receptive field

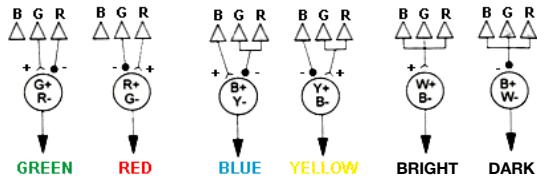
- The part of the input IMAGE that produces a positive response in a biological neuron is the 'receptive field'
 - The surrounding area inhibiting that response is the 'suppressive surround'
- In artificial networks, 'receptive field' often means the spatial extent of a convolutional filter
 - Often in its INPUT FEATURE MAP
 - This is a programmed parameter
 - It's harder to determine the spread of the inputs in the input image
- In biological neurons, it's really hard to determine the spatial spread of a neurons input
 - This is sometimes called the 'connective field'

-The area producing a positive response is called the receptive field of the cell, while the area suppressing that response is called the suppressive surround.

-In an artificial neural network, the term 'receptive field' is often used for the spatial extent of a filter. Properly, this should refer to its spatial extent in the input image, though it is often used to mean the extent in the previous feature map.

-In biological systems, 'receptive field' always refers to the extent in the retinal image, partly because it's really hard to determine the spatial spread of a cell's inputs. We'll look at some methods to do this in the next class.

Colour map comparison filters: colour opponency



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Together with this spatial comparison, there is also a comparison over the different colour cones.

-This forms four further feature maps, one each for greenness, redness, blueness, and yellowness.

-This is very distinct from the cone activations, though this is not obvious for most students. For example, white light contains all colours and so it activates all cones strongly. But that light isn't coloured. So the activation of a single cones does not tell us the light's colour, the relative activations of different cones must be compared to determine colour.

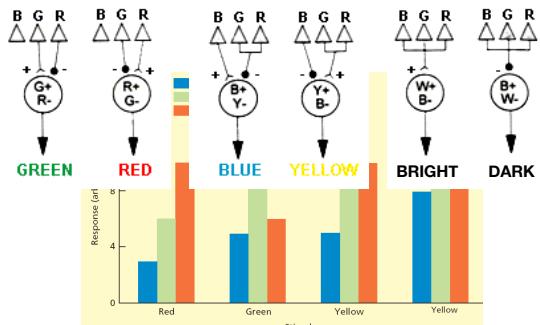
-There are relatively few blue cones, so redness and greenness are each determined by the difference between their intensity and the intensity of the other. Where both red and green are present at equal intensity, for example in white light, there is no response in these channels.

-Blueness is determined by the difference between the intensity of blue cone activation and the intensity of other cone's activations.

-Yellow light activates both green and red cones, but not blue. So yellowness is the average of red and green cone activations minus blue cone activation. Again, white light will produce no response.

-To detect brightness and darkness, all three cones activate or inhibit the response.

Colour map comparison filters: colour opponency



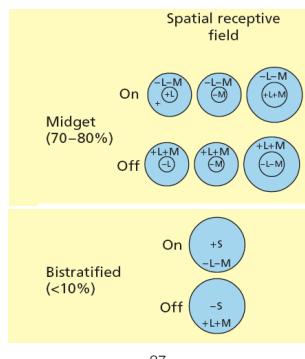
This is approximately optimal for representing colour information
-Beyond the photoreceptor, we have three opponent channels for the **ratios** of R vs G, Y vs B and light vs dark

-Using these ratios, each individual colour representation gives an accurate description of the colour of light falling on the retina, not its intensity.

-To get this colour-based description, there would always need to be a stage to **COMPARE** the ratio of activation of the three cone types

-Doing the comparison early (in the retina) produces that representation as early as possible, so it can be used for all later processing.

Colour and position comparison together



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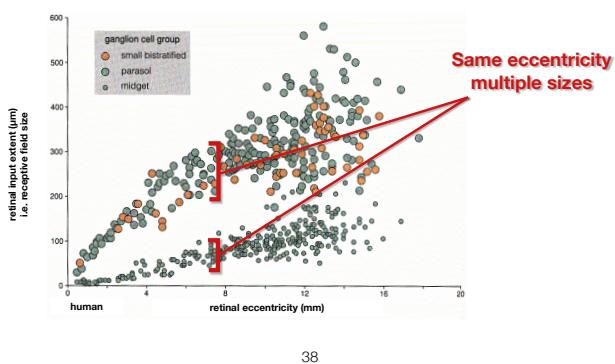
So far, we have treated the spatial comparisons and the colour comparisons separately, but actually they are together, so the filters cross both colour and space: they cover a limited spatial extent differently for different feature maps.

-In the case of the bistratified retinal ganglion cell, there is no suppressive surround around the activating centre, but these filters do have a limited spatial spread.

-The midget ganglion cells have small receptive fields and small suppressive surrounds, with different colour inputs to the receptive field and the surround.

-Some of these also respond to any colour in the centre receptive field and also the surround. Because blue cones (S-cones) are relatively low density, these are not used here. This forms the light-dark opponent filter pair.

Receptive field extent and spatial frequency



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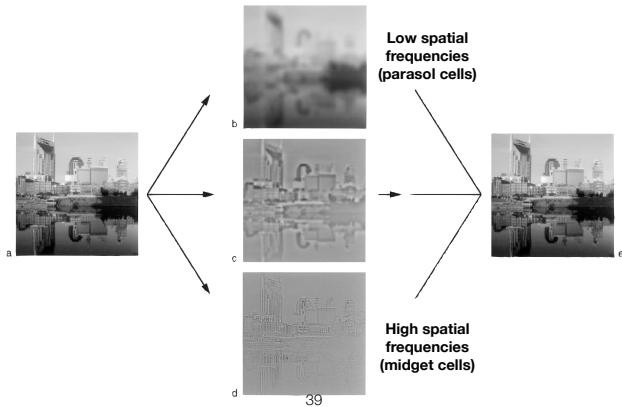
As we go from central to peripheral vision, the spatial extent of these integrating filters, the horizontal cells and retinal ganglion cells, increases.

-So unlike in artificial deep networks, filters are not the same over the whole visual field

-Even at the same retinal location, we see two distinct classes of retinal ganglion cells integrating spatial information over different extents, the midget and parasol cells.

-And even with these cell populations, there is a range of spatial extents.

Spatial frequency components of an image



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Using these differently sized receptive fields, effectively different filter sizes, the retina can break down any image into different components at different spatial frequencies, different spatial scales.

-These have different properties. At low frequencies, we miss changes that are at high frequency, so we can see big blocks of light and dark with no edges

-At high frequencies, we can only see the edges, with no lower-frequency changes in color because these would stimulate both the center and the surround.

-But if we keep these different parts, we can add them back together to recreate the original image

-So these representations of different spatial frequencies also form different feature maps, even at the first stages of processing.

-This is quite different from the process in artificial deep networks, where each layer is convolved with filters of a single spatial size.

-In artificial networks, larger-scale features can only be detected by filters at later stages, after intermediate stages of spatial integration and pooling.

Retinal feature maps

- In the input image from the photoreceptor:
 - Red light intensity, green light intensity, blue light intensity (cones), overall light intensity (rods)
- By the retinal ganglion cell:
 - Redness, greenness, blueness, yellowness, lightness, darkness
 - Many spatial frequency ranges
 - Many combinations (not all)

So the filters in the retina perform a range of comparison operations on their inputs

Normalisation and pooling

- Normalisation
 - Inhibition of activity by average nearby activity
- Pooling
 - No distinct problem of computational load
 - But progressive layers do have smaller representations, and more 'feature maps'
 - No distinct pooling stage: just use fewer neurons to sample previous layer

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Shared weights?

- Weights at each synapse develop independently from other neurons
- No fixed filter extent at the retina
 - Receptive field (filter) sizes vary with eccentricity
 - Multiple receptive field sizes at same location
- However, if a filter structure develops at one part of the feature map to compute something useful, similar filter structures are likely to develop elsewhere.

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Remember that a major benefit of shared weights is that convolution is a fast matrix operation, but relies on a single filter.

This limitation does not apply if filters are all made from distinct biological elements.

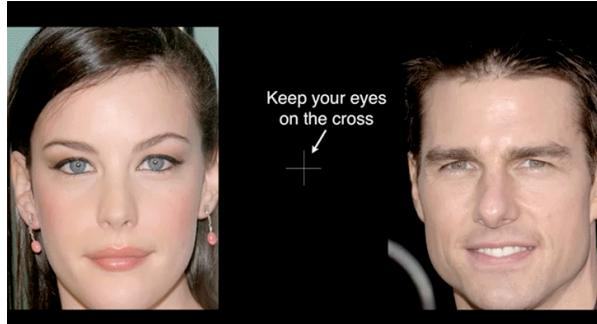
There is no convolution operation, and no single processor core that needs to process the operations of all units.

Another benefit of shared weights was that the target for machine learning is a very small number of filters if the same filter applies across the whole feature map.

Again, this is not important if filters are all made from distinct biological elements.

Hebbian learning, unlike back propagation, operates on every synapse independently.

Differences in perception across the visual input



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-Indeed, differences in object recognition abilities across the visual image are clear in perception
-Try identifying these faces using your peripheral vision only
-If we move our gaze between the central cross and the faces, we can see how faces appear with and without high spatial frequencies
-This shows that even at higher levels like object recognition, the brain's filters for visual information are not the same over the whole visual image

Deep learning in biological neurons and networks

- Shares most operations with artificial networks (except pooling), but typically with some differences
- Filter/convolve:
 - Dendritic tree synapses with many neurons
 - Different synapses have different strengths (weights)
 - Filters cross multiple feature maps, even at earliest stages
- Threshold:
 - Activation of voltage-gated ion channels
 - Following summation on EPSPs and IPSPs
 - Leads to action potentials
- Normalise:
 - Inhibition of activity by local mean activity
- Learning
 - Hebbian learning: 'cells that fire together wire together'
 - Strengthens responses to patterns of activity previously causing with responses

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Major differences to artificial deep networks

- Feature maps do not represent the whole image with the same detail: strong bias towards sensitive/important parts
 - Sensitive areas (hands, eyes) can move to sample task-relevant information
- Filters have a limited spatial extent, but not a fixed extent
 - Multiple spatial scales analysed at the same brain area 'layer'
 - Image layout is maintained in later stages, allowing analysis of spatial relationships at many levels
- Filter weights not shared across each layer, but similar filters likely to emerge in many places
- Maximum response is always part of the threshold
- Normalise of activity by local (not global) mean activity
- Learning is generally unsupervised

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