

Introduction to Neuroinformatics 1.0

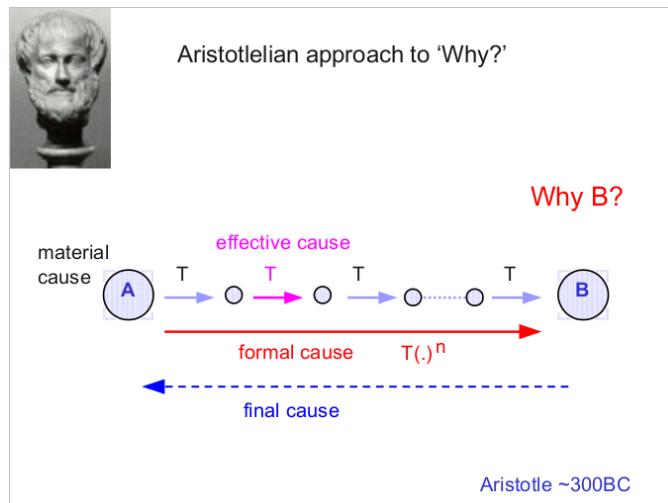
Summary of the lectures 2012

Benjamin Ellenberger

1 Lecture 1 - Neuroinformatics (Rodney Douglas)

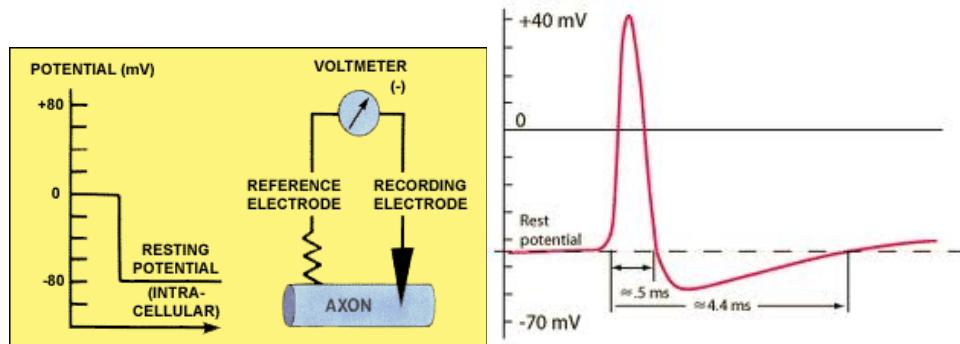
Introduction lecture, not much to learn yet.
Aristotelian approach to 'Why?'

- Material cause
- Effective cause
- Formal cause
- Final cause



2 LECTURE 2 - MEMBRANE POTENTIALS (RODNEY DOUGLAS) 2

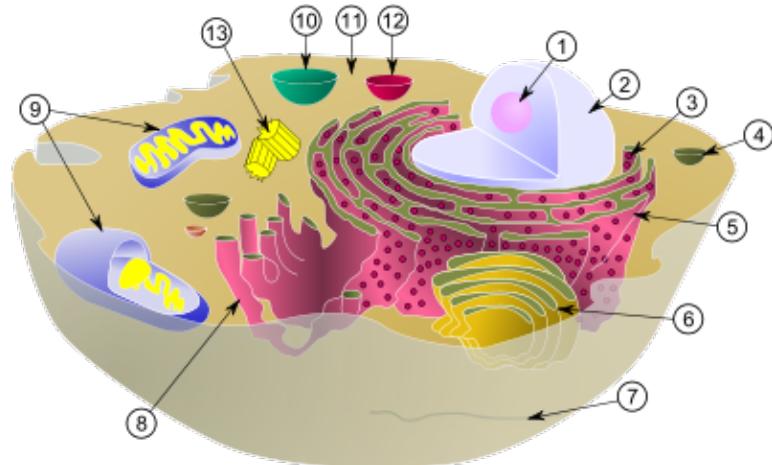
2 Lecture 2 - Membrane Potentials (Rodney Douglas)



The spike is the action potential caused by sodium (Na^{2+}) and changes in the membrane conductance. This shows that there is a voltage source in the membrane.

2 different types of neurons(prokaryotic, eukaryotic)

- Eukaryotic cells differ in cell body components(Mitochondria, endoplasmatic reticulum, Golgi apparatus...)

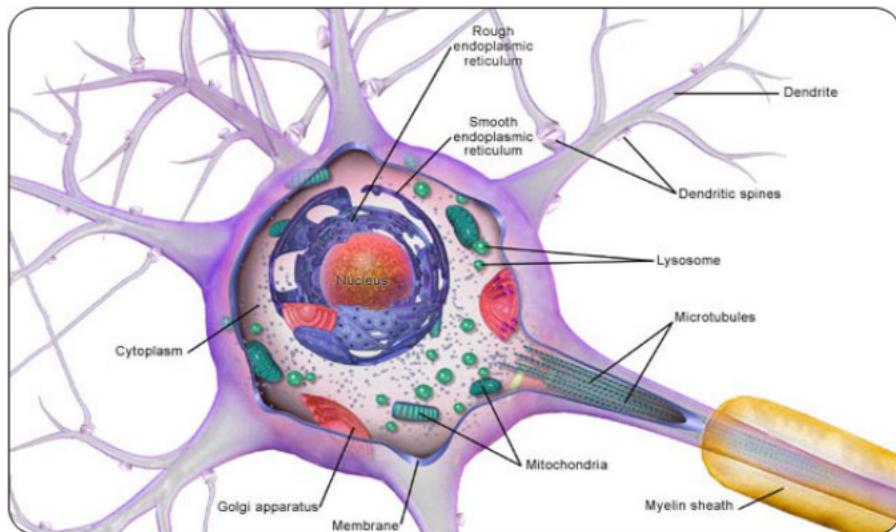


Typical animal (eukaryotic) cell subcellular components:

1. nucleolus
2. nucleus

2 LECTURE 2 - MEMBRANE POTENTIALS (RODNEY DOUGLAS) 3

3. ribosome
4. vesicle
5. rough endoplasmic reticulum
6. Golgi apparatus
7. Cytoskeleton
8. smooth endoplasmic reticulum
9. mitochondria
10. vacuole
11. cytoplasm
12. lysosome
13. centrioles within centrosome



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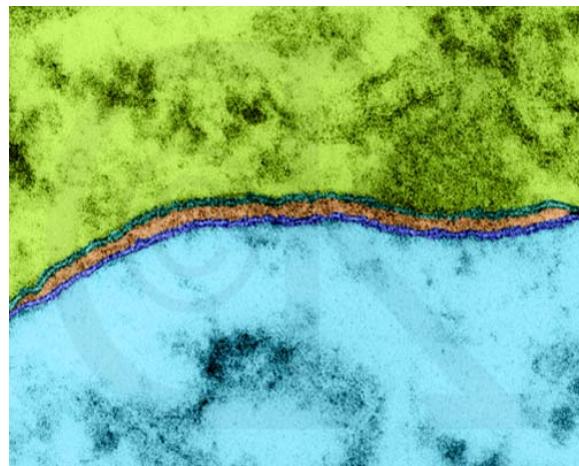
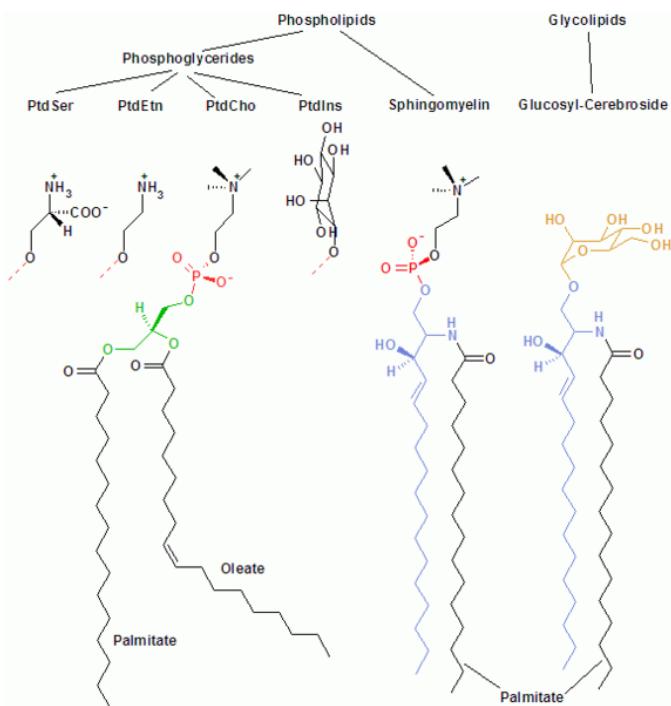
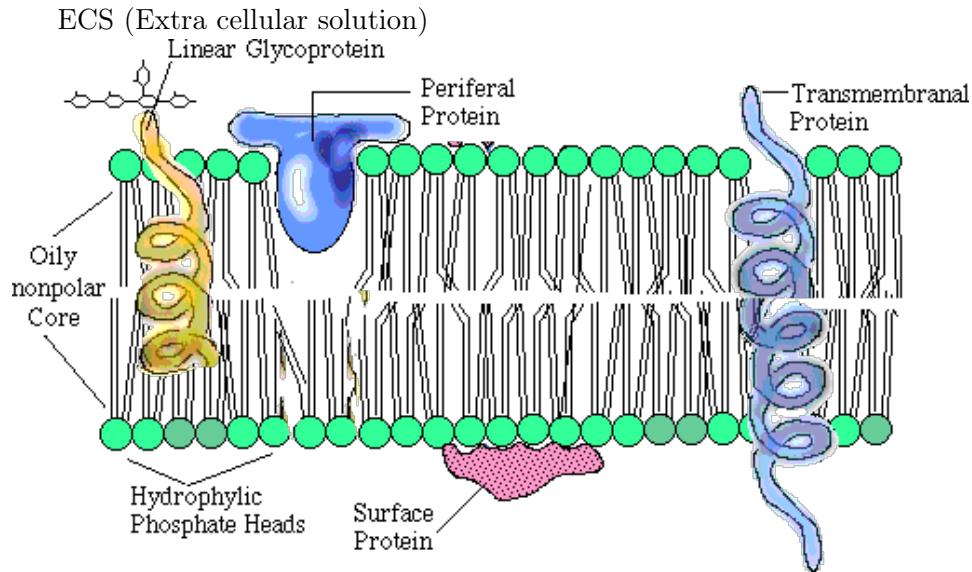


Figure 1: The membrane bilayer



The bilayer is built out of those molecules whereas the charged sides align (such as the one with N^+H_3 and the uncharged ends align such as the long CH chain. The uncharged ends are hydrophobic).



ICS (Intra cellular solution)

Transmembranal proteins play an important role as they have charged ends(phosphate heads) and uncharged bodies and therefore fit inside of the membrane as it aligns with the uncharged inner part of the membrane and with the outer charged parts.

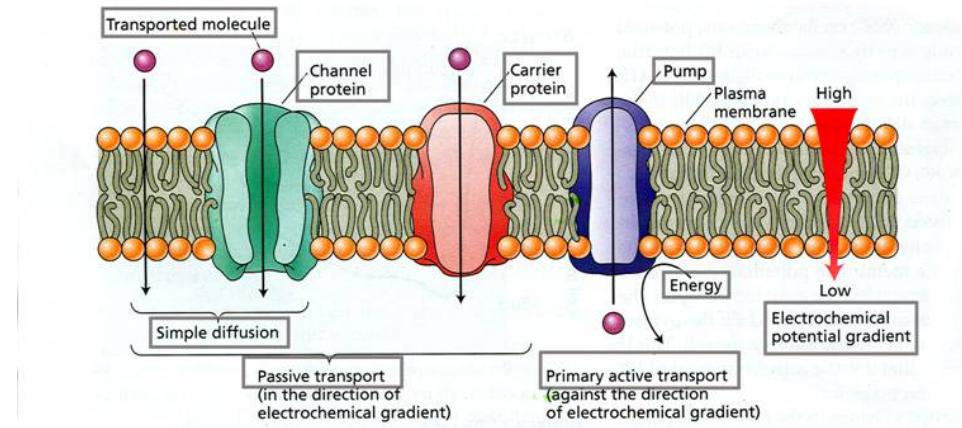
2 basic mechanisms for the membrane potential:

- Gibbs-Donnan equilibrium (Causes asymmetry across membrane)
- Active mechanisms(Na^+/K^+ -Pump from ECF to ICF)

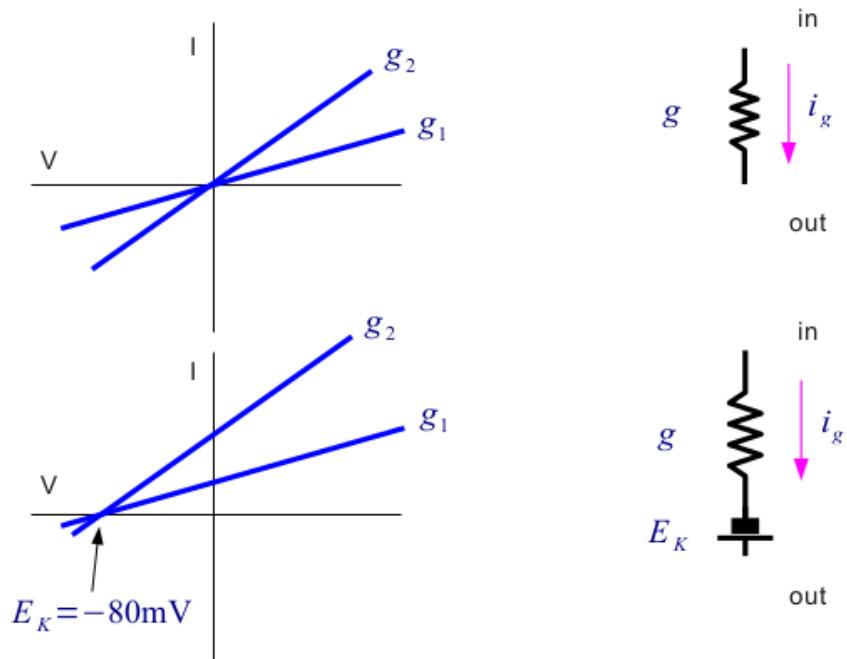
2.1 Concentrations across the membrane

	ICF	ECF
Na^+ (Sodium)	-	+
K^+ (Potassium)	+	-
Cl^- (Chlorine)	+	-
Ca^{2+} (Calcium)	x	(+)
Large Anions(not passing membrane)	+	(-)

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There is a concentration gradient top-down from high concentration to low concentration. The pump uses ATP to build up the gradient if it is released.



The function of conduction is approximative linear and reaches $E_k = -80mV$ at the point of no current.

2.2 Fick's Law of diffusion

$$J_{diff} = -D \frac{\delta [C]}{\delta x} \quad (\text{Concentration gradient})$$

ICF → ECF

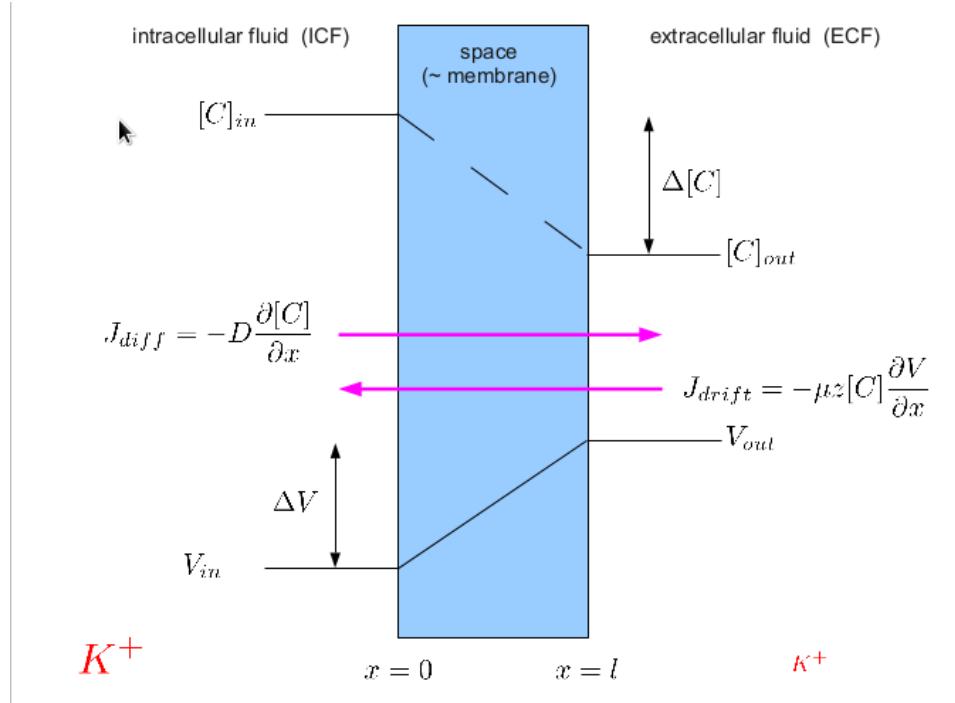
- J Diffusion flux ($\frac{\text{molecules}}{\text{sec} \cdot \text{cm}^2}$)
- D Diffusion coefficient ($\frac{\text{cm}^2}{\text{sec}}$)
- [C] Concentration of species ($\frac{\text{molecules}}{\text{cm}^3}$)

2.3 Ohm's law of drift

$$J_{drift} = \delta_{el} E = -\mu z [C] \frac{\delta V}{\delta x} \quad (\text{Voltage gradient})$$

ICF ← ECF

- J Drift flux ($\frac{\text{molecules}}{\text{sec} \cdot \text{cm}^2}$)
- δ_{el} Electrical conductivity ($\frac{\text{molecules}}{\text{V} \cdot \text{sec} \cdot \text{cm}}$)
- $E = -\frac{\delta V}{\delta x}$ Electric field ($\frac{\text{V}}{\text{cm}}$)
- V Electrical potential
- μ Mobility of species ($\frac{\text{cm}^2}{\text{V} \cdot \text{sec}}$)
- z Valence of ion (dimensionless)
- [C] Concentration of species ($\frac{\text{molecules}}{\text{cm}^3}$)



2.4 Einstein's relation between diffusion and mobility

Diffusion and drift are additive

$$D = \frac{k \cdot T}{q} \mu$$

- k Boltzmann's constant ($1.38 \times 10^{-23} \frac{Joule}{K}$)
- T Absolute Temperature (K)
- q Charge of the molecule (Coulombs)

2.5 Nernst-Planck equation

Substitutes part of Einstein's formula $J = J_{drift} + J_{diff} = -\mu z[C] \frac{\delta V}{\delta x} - D \frac{\delta [C]}{\delta x}$

Divided by Avogadro to obtain molar form, J scaled by zF to obtain molar charge

$$I = J \cdot zF = -(\mu z^2 F [C] \frac{\delta V}{\delta x} + u z RT \frac{\delta [C]}{\delta x})$$

at $37^\circ C \rightarrow -60mV$

- R Universal gas constant ($8.3144 \frac{J}{mol \cdot K}$)
- F Faraday constant ($96500 \frac{C}{mol}$)
- u Molar mobility ($\frac{cm^2}{V \cdot sec \cdot mol}$)

2.6 Goldman-Hodgkin-Katz equation (GHK-Voltage equation)

$$\Delta V = \frac{RT}{F} \ln \left(\frac{P_K[K^+]_{out} + P_{Na}[Na^+]_{out} + P_{Cl}[Cl^-]_{in}}{P_K[K^+]_{in} + P_{Na}[Na^+]_{in} + P_{Cl}[Cl^-]_{out}} \right)$$

This is only for static situations but gives us a sense of how it is actually working. Basic assumptions:

- Ion flux obeys Nernst/Planck equation
- Ions move across membrane independently (no interaction)
- Electric field in the membrane is constant $E = -\frac{\delta V}{\delta x} = -\frac{\Delta V}{l}$

2.7 Permeability

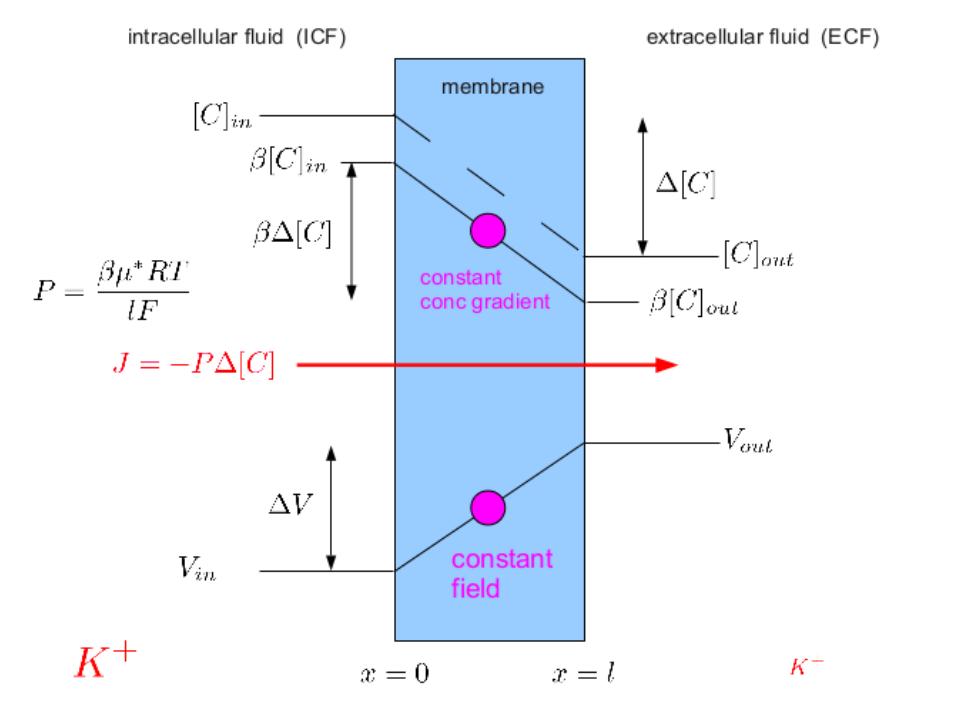
$$J = -P\Delta[C]$$

$$D^* = \mu^* \frac{kT}{q} = \mu^* \frac{RT}{F}$$

$$P = \frac{\beta D^*}{l} = \frac{\beta \mu^* RT}{lF}$$

- β Water-membrane partition coefficient for ion i
- μ^* Mobility of ion i within the membrane
- D^* Diffusion coefficient within membrane

3 LECTURE 3 - PASSIVE (CABLE) MEMBRANE PROPERTIES (RODNEY DOUGLAS)



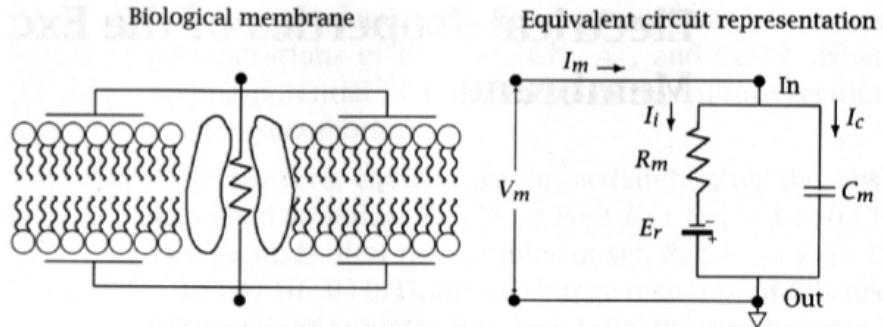
3 Lecture 3 - Passive (Cable) Membrane Properties (Rodney Douglas)

- J_{diff} and J_{drift} are in an equilibrium during the resting potential
- Resting potential is due to K^+ concentration
- GHK-equation ΔV = Difference in Potential across the membrane
- if $P(Permeability) = 1 \rightarrow Nernst - equation$

3 LECTURE 3 - PASSIVE (CABLE) MEMBRANE PROPERTIES (RODNEY DOUGLAS11

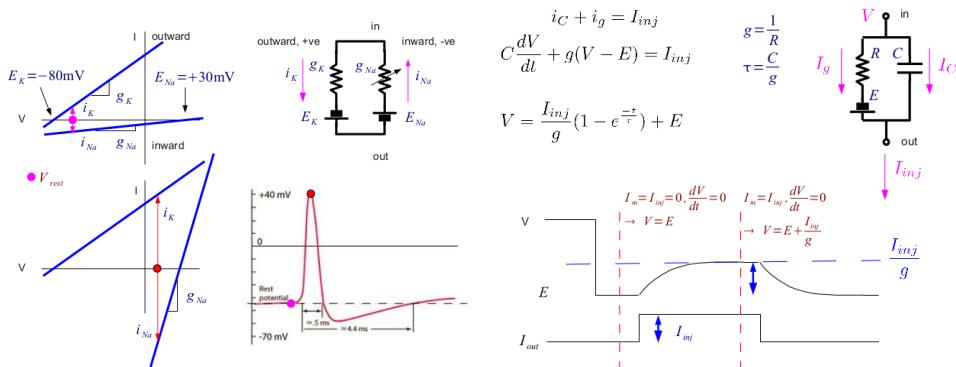
3.1 Ohmic model of biological membrane

Ohmic model



$$C_m \frac{dV_m}{dt} + \frac{V_m - E_r}{R_m} = I_m$$

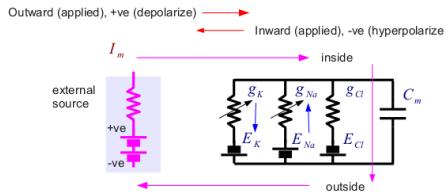
- The conductor indicates the temporal dynamicity
Conductance $G = \frac{\text{Current } I}{\text{Voltage } U} = \frac{1}{\text{Resistance } R}$
- $I_c + I_o = I_m$
- If $\frac{\delta V_m}{\delta t} = 0 \rightarrow$ membrane is at rest
- Changing the permeability of the membrane results in changeable conductance g in membrane \rightarrow over time.



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3 LECTURE 3 - PASSIVE (CABLE) MEMBRANE PROPERTIES (RODNEY DOUGLAS12

3.2 Electrical nature of the membrane



$$I_m = I_C + I_K + I_{Na} + I_{Cl}$$

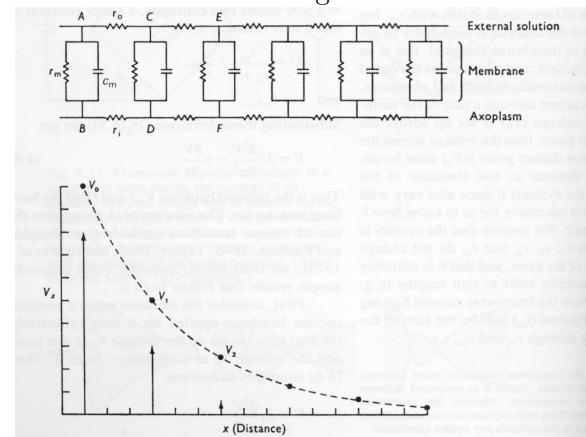
$$C_m \frac{dV}{dt} + g_K(V - E_K) + g_{Na}(V - E_{Na}) + g_{Cl}(V - E_{Cl})$$

when $I_n = 0, \frac{dV}{dt} = 0$

$$V_{rest} := V = \frac{g_K E_K + g_{Na} E_{Na} + g_{Cl} E_{Cl}}{g_K + g_{Na} + g_{Cl}}$$

- Typical values for electrical properties of membranes:
- Capacitance (Cell of $20\mu m$ diameter: $c_m = C_m \cdot A = 1 \frac{\mu F}{cm^2} \cdot 10^{-5} \approx 10 pF$
Resistance: $r_m = \frac{R_m}{A} = \frac{10 k\Omega \cdot cm^2}{10^{-5}} \approx 1000 M\Omega$
Time constant ($\mu F \cdot M\Omega = sec$): $\tau_m = c_m r_m = \frac{R_m}{A} \cdot C_m A = R_m C_m \approx 10^{-6} \mu F \cdot 10^4 \Omega = 10 msec$

- Membrane behaves like a resistance network
Current leaks out through membrane resistance



Voltage leakage is rising exponentially. 4 decay constants leads to $\approx 0V$

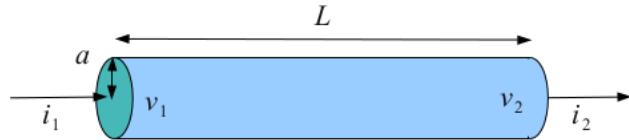
3 LECTURE 3 - PASSIVE (CABLE) MEMBRANE PROPERTIES (RODNEY DOUGLAS13

3.3 The cable equation

The membrane potential along a neuronal cable is expressed as a function of a single longitudinal spatial coordinate x and time, $V(x, t)$.

Current flows within a neuron due to voltage gradients.

Currents flowing in the direction of increasing x are defined to be positive.



specific membrane parameters (independent of geometry)

$$R_m = 10 \text{ k} \Omega \cdot \text{cm}^2, \quad R_a = 100 \Omega \cdot \text{cm}, \quad C_m = 1 \mu\text{F}/\text{cm}^2$$

$$\text{axial resistance} \quad r_a = \frac{R_a L}{\pi a^2}$$

$$\text{membrane resistance} \quad r_m = \frac{R_m}{2 \pi a L}$$

$$\text{membrane capacitance} \quad c_m = C_m 2 \pi a L$$

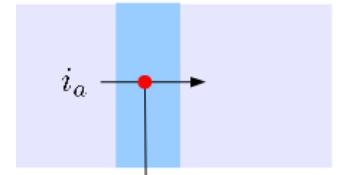
r_a, r_m, C_m are cylinder (geometry) dependent parameters

$\tau = \text{time constant}$

$\lambda = \text{space constant for } R_m \text{ and } R_a \text{ of this cable.}$

$$\frac{\partial v}{\partial x} = -r_a i_a$$

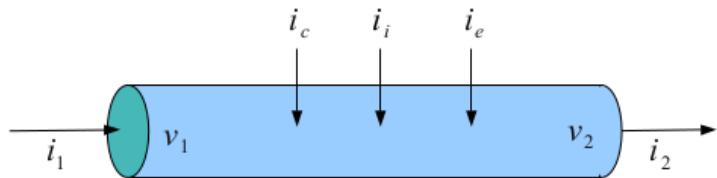
$$\frac{\partial i_a}{\partial x} = -i_m$$



$$i_m = i_c + i_i + i_e$$

$$i_m = i_c + i_i = c_m \frac{\partial v}{\partial t}$$

3 LECTURE 3 - PASSIVE (CABLE) MEMBRANE PROPERTIES (RODNEY DOUGLAS14



To cut a long story short,
the 'cable equation' becomes finally...

$$\lambda^2 \frac{\partial^2 v}{\partial x^2} = \tau \frac{\partial v}{\partial t} + v$$

$$\lambda = \sqrt{\frac{r_m}{r_a}} = \sqrt{\frac{aR_m}{2R_a}} \approx 0.6mm$$

$$\frac{\partial v(x)}{\partial t} = 0, \quad v(x) = \frac{r_\lambda i_e}{2} e^{\frac{-x}{\lambda}}$$

where r_λ is the 'input resistance' of an infinite cable

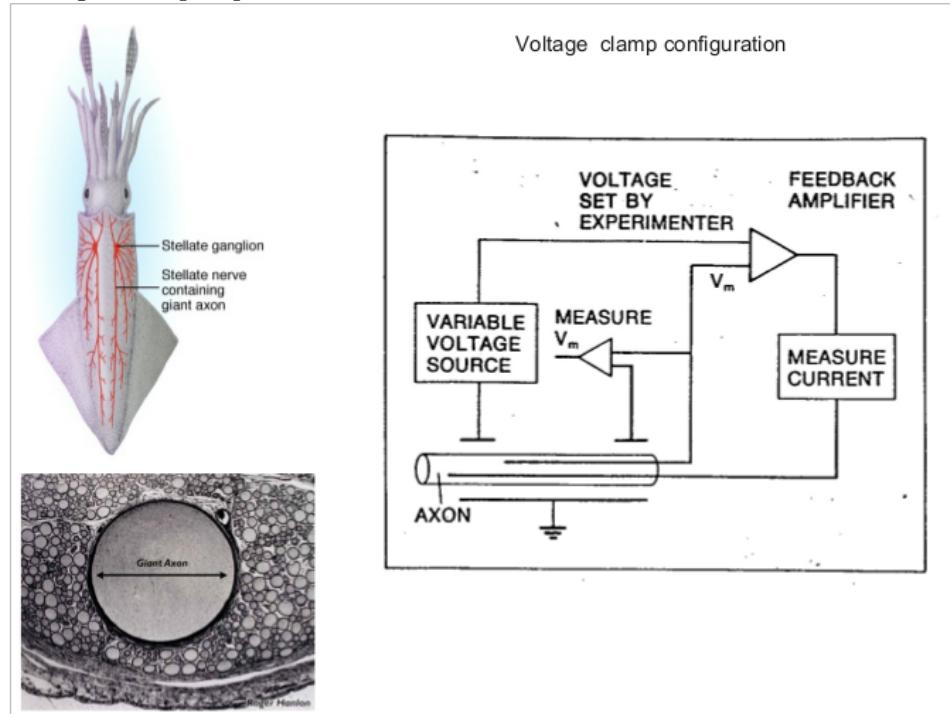
- $\lambda = 0.6mm$ means no signal left after $0.6mm$
- Increasing R_m of the cable increases λ
- Increasing diameter of the cable increases λ

3.4 From exercises: Electric laws

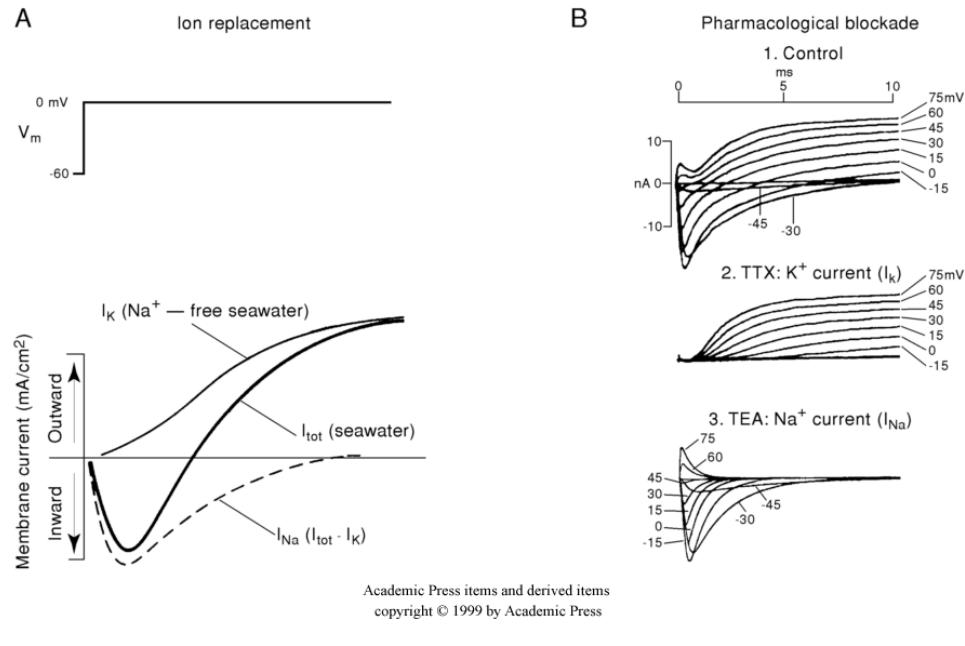
- Kirchhoff's Current Law (KCL): The sum of all currents entering and leaving any node in a circuit is not zero.
- Kirchhoff's Voltage Law (KVL): The sum of all voltages around a closed loop is zero.
- Ohm's Law: $V = I \cdot R$

4 Lecture 4 - Action Potentials (Rodney Douglas)

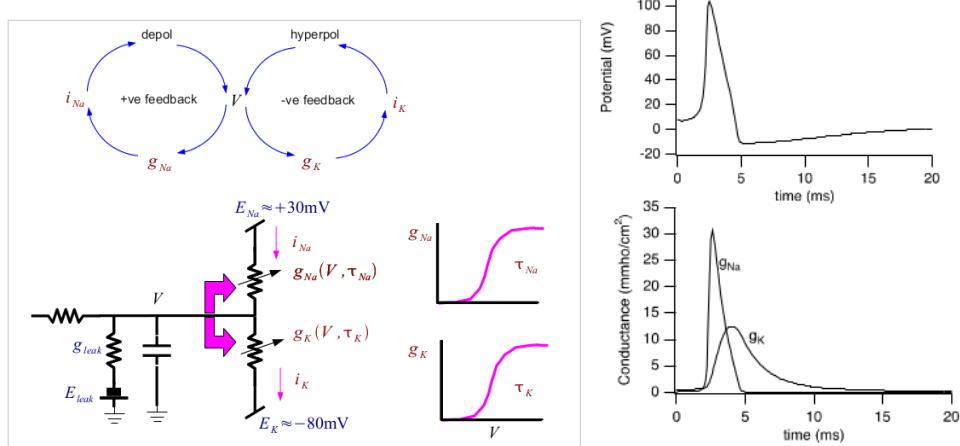
- Voltage Clamp experiment



- With negative feedback circuit
- Command voltage is set by the experimenter, the feedback circuit holds the voltage constant.
- The voltage clamp allows the membrane voltage to be manipulated independently of ionic currents, allowing the current-voltage relationships of membrane channels to be studied.



- Voltage dependent conductance for g_{Na}, g_K
No static voltage
- Action potential



- g_{Na} increases quickly, but then inactivation kicks in and it decreases again.
- g_K increases more slowly, and only decreases once the voltage has decreased.
- The Na^+ current is autocatalytic. An increase in V increases g, which increases the Na^+ current, which increases V, etc.
- The threshold for action potential initiation is where the inward Na^+ current exactly balances the outward K^+ current.
 $\rightarrow g_{Na} > g_K$ (leads to the spike[depolarisation]) $\rightarrow g_K$ increases $b \rightarrow g_K > g_{Na}$ (hyperpolarisation)

4.1 The Hodgkin-Huxley equations

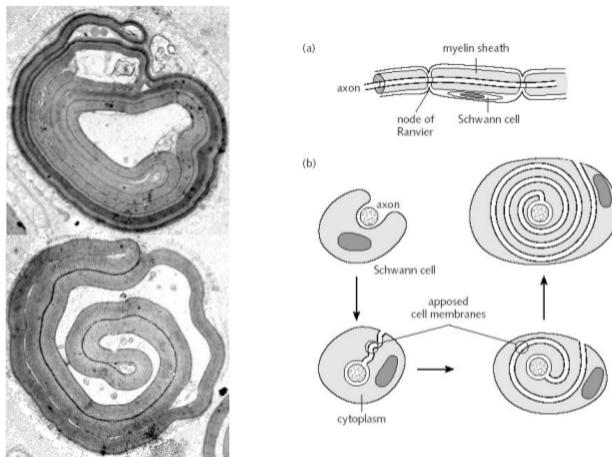
$$C \frac{dV}{dt} + \bar{g}_K n^4 (V - V_K) + \bar{g}_{Na} m^3 h (V - V_{Na}) + \bar{g}_L (V - V_L) + I_{injected} = 0$$

m^3, n^4 are part of the proposed model.

$\bar{g}_L (V - V_L)$ is the general leak.

- Both amplitude of conductance change and its time const g change with V_{clamp}
- The m and n gates open with depolarisation
- The h gate closes with depolarization
 \rightarrow Not symmetrical power (circuit currents), ap goes into one direction

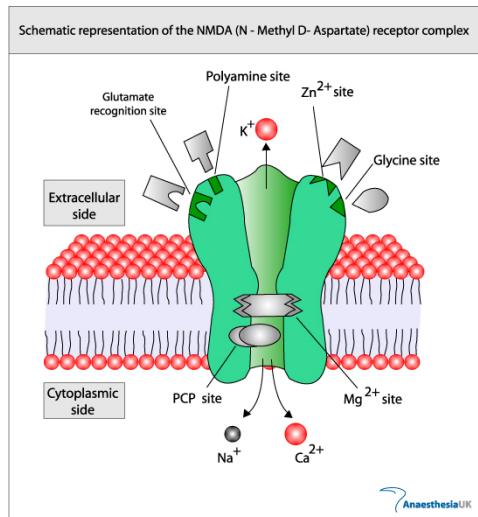
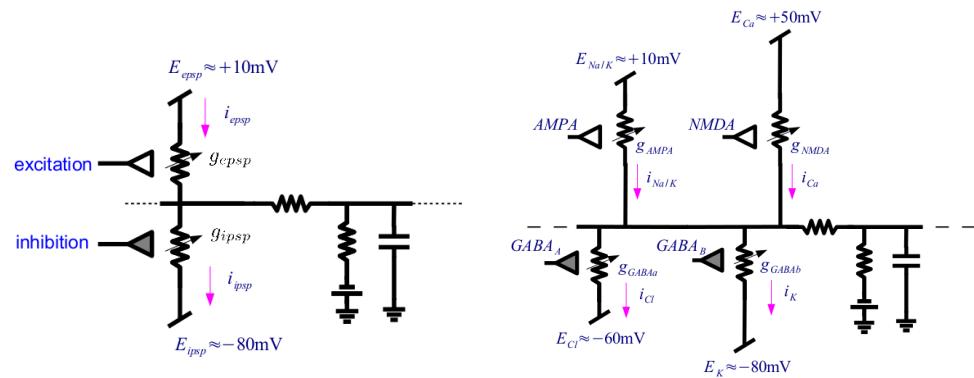
4.2 Saltatory conductance



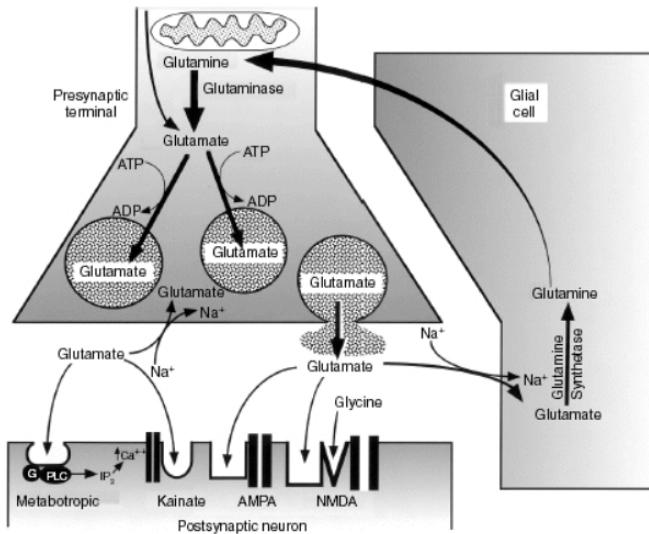
- Ranviernodes
- Myelinized by gliacells → resistance high/not leaking
- The thicker the cell the faster the signal

5 Lecture 5 - Synapse 1 (Rodney Douglas)

- Neurotransmitter
 - Glutamat = excitation
 - GABA = inhibition
- Synaptic mechanism
 1. Synthesis: Building blocks of transmitter substance are imported into the terminal where the neurotransmitter is synthesized and packaged into vesicles.
 2. Release: In response to an AP, the transmitter is released across the membrane by exocytosis.
 3. Receptor activation: The transmitter crosses the synaptic cleft and binds to a receptor.
 4. Inactivation: The transmitter is either taken back into the terminal or inactivated in the synaptic cleft.



5.1 Glutamate receptor



- NMDA-receptor: Depolarization → Mg²⁺ is removed. Activation by Glutamate and co-agonist → Ca²⁺ can float in
 ↘ Ca²⁺, Na⁺, K⁺
- AMPA-receptor: Calcium activates NMDA-receptor. Second messenger activates. Weak stimulation by Glutamate only AMPA receptor is bound to Glutamate.
 ↘ Na⁺, K⁺, Ca²⁺
- Important feature of NMDA-receptor

Activation of glutamate requires co-agonists glycine or serine.

Effect requires coincidence of depolarization of post-synaptic membrane to dislodge Mg²⁺ and binding of agonist.

Relatively slow post-synaptic EPSP

10x more permeable to Ca²⁺ than to Na⁺ or K⁺

Receptor	Transmitter	ion	Approx E _{rev}	Agonist	Antagonist
AMPA	glutamate	Na, K, Ca	+0mV	AMPA	CNQX
NMDA	glutamate	Ca, Na, K	+10mV	NMDA(glycine)	AP5,ketamine,MK-801
mGLU	glutamate	G-coupled			
GABAa	gaba	Cl	-60mV	muscimol	bicuculine
GABAb	gaba	K/G-coupled	-80mV	baclofen	saclofen

6 Lecture 6 - Plasticity/Learning (Michael Pfeiffer)

6.1 Learning & Memory

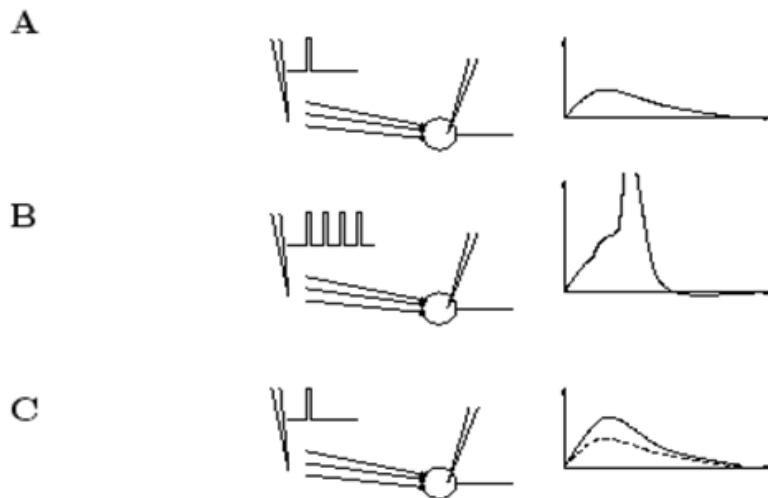
- Learning is the acquisition of new information or knowledge
- Memory is the retention of learned information
- Types of memory
 - Declarative Memory (Facts,Events)
 - Non-Declarative Memory
 - Procedural Memory (Skills, Habits)
 - Emotional responses
- Facts about Synapses
 - Neurons communicate via AP and are interconnected via synapses
 - Information is represented by distributed activity
 - Learning and memory is based on changes in synaptic connections (Formation & retraction of synapses (development), Changes in synaptics efficacies (plasticity))

6.2 Plasticity

What is plasticity?

Axiomatic rule: Everything is somehow encoded in synapses.

No information in the shape of the spike, but in the frequency and synchronicity.

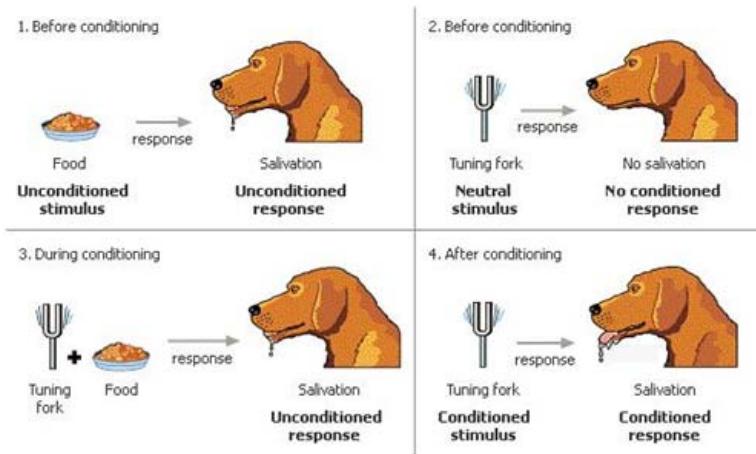


- Modification of postsynaptic potentials (PSP) evoked by presynaptic spikes
 - A. Postsynaptic response triggered by a weak test pulse (left).
 - B. Strong stimulation sequence (left) triggers postsynaptic firing.
 - C. A later test pulse evokes a larger postsynaptic response than initially.
- Parameters that define synapse strengths
 - Neurotransmitter and receptor type
 - Position of synapse
 - Availability of vesicles
 - Re-uptake
 - Neuromodulators(Dopamin etc.)
 - „Non-synaptic“ plasticity(excitability of neurons, dendritic branch strength)
 - Postsynaptic cellular processes
 - Pre-/postsynaptic firing
 - etc.
- Diseases affect plasticity: Alzheimer, Parkinson

6.3 Models of plasticity

- Non-synaptic plasticity (Excitability of neurons, dendritic branch strength)
- Synaptic plasticity
 1. Phenomenological models (High-level, relationships between activity & plasticity etc. exp: Pavlov Classical conditioning)
 2. Biophysical models (Low-level, cellular processes etc. exp: Hebbian learning)

6.4 Pavlovian Learning



6.5 Hebbian Learning

- "Fire together, wire together"
- Learning based on correlations between pre- and postsynaptic firing
- Uses only variables locally available at the synapse
- Rate-based model: $\Delta \text{synaptic-efficacy}_{\text{neuronA}, \text{neuronB}} \propto \text{firing-rate}_{\text{neuronA}} \cdot \text{firing-rate}_{\text{neuronB}}$
- Only weight increase modelled / No depression
→ Can lead to instability (positive feedback loops)
- Other rules: BCM rule, Oja's rule

- Implications:

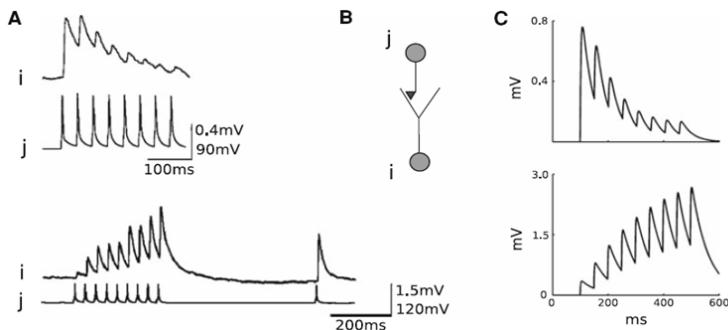
Global effects arise from local learning

Variables(pre- & postsynaptic action potential, efficacy(weight),local concentration)

6.6 NMDA synapse

- Can act as coincidence detector for pre- and postsynaptic firing
- Backpropagation action potentials
- Depolarization from other synapses
- Calcium influx crucial for plasticity
- Strong NMDA activation → potentiation
- Weak NMDA activation → depression

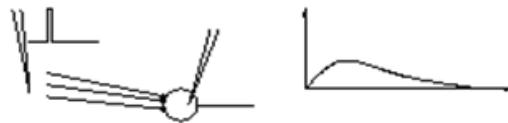
6.7 Short term plasticity(STP)



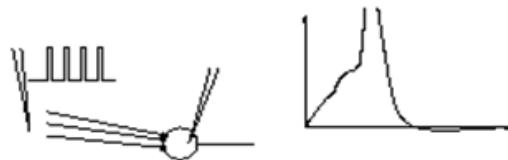
- A neuron j fires several times, neuron i fires as well and the spike size is increased(the higher the spike, the more efficient the neuron), but decreases after a short time.(Caused by loss of vesicles)
- Effect goes away in order of seconds.

6.8 Long term plasticity(LTP)

A



B



C



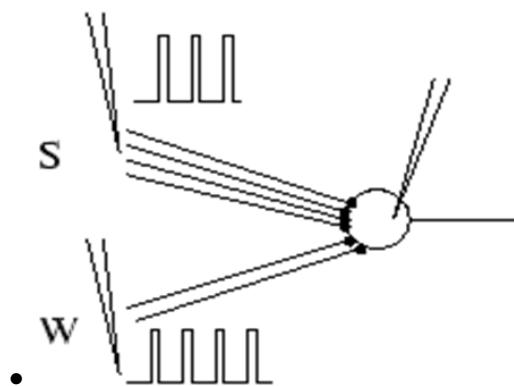
- Schematic drawing of a paradigm of LTP induction.

A. A weak test pulse (left) evokes the postsynaptic response sketched on the right-hand side of the figure.

B. A strong stimulation sequence (left) triggers postsynaptic firing (right, the peak of the action potential is out of bounds).

C. A test pulse applied some time later evokes a larger postsynaptic response (right; solid line) than the initial response. The dashed line is a copy of the initial response in A. (schematic figure).

- LTP occurs if a synapse and the post-synaptic neuron are simultaneously depolarized beyond a threshold. This can occur in cooperation (weak and weak or weak and strong signals)

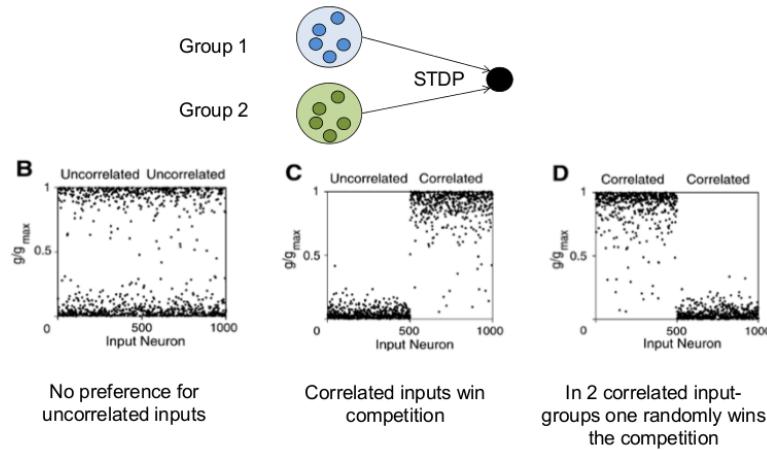


Cooperativity in the induction of LTP. Weaker synapse W is strengthened if the postsynaptic neuron is active and both presynaptic sites are firing.

6.9 Spike-timing dependent plasticity (STDP)

- Not only correlation, but also timing of the spikes determines plasticity.
- Sign of plasticity is determined by local calcium concentration
- Postsynaptic spike travels back to the dendritic tree and activates voltage-dependent Ca channels
- Presynaptic activity can allow Ca influx through NMDA channels (if postsynaptic part is sufficiently depolarized)
- If pre-spike is soon afterwards followed by post-spike, NMDA-R activity is supralinearly enhanced by depolarization due to backpropagating spike $\rightarrow \text{Ca}^{2+}$ determines the strength of plasticity
- Functional consequence:

Correlated firing groups win the battle against uncorrelated groups (depression). Battle of two correlated groups have a random winner.



6.10 Facts

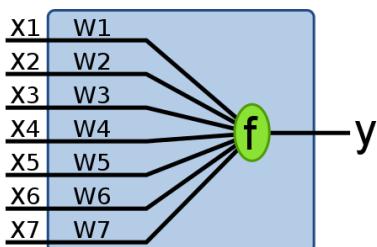
- A lot of diversity in STDP occurrences
- Different plasticity in different brain areas

- Diversity of neuron and synapse types
- Large number of control parameters for plasticity experiments (frequency, timing, postsynaptic voltage, position on the dendrite, ...)
- Influence of neuromodulators, calcium, drugs, and various proteins
- Long-term vs. short-term effects
- It is unlikely that there is one single model that explains all plasticity effects found in biology

6.11 Dopamine

- Neurotransmitter and neuromodulator
- Significant for motor processes (Parkinson), pleasure and reward, motivation, attention, emotions
- DA activation is a relatively homogeneous, global population signal
- DA activation is related to rewarding stimuli or reward prediction errors
- Reinforcement learning
- Dopamine can extend the timing window for LTP
- Dopamine can convert LTD into LTP

7 Lecture 7 - Perceptron Learning Algorithm



Perceptron, McCulloch-Pitts Neuron, Linear Threshold Unit

- Model represents a neuron as a number of inputs X (dendrites) and one output Y (axon). The weights W determine the influence of a dendrite input, which is either excitatory or inhibitory. f is a function that defines how to combine the weights x and w(normally $\sum(w_i \cdot x_i)$) and θ (not shown in picture but usually at the place between y and f) is normally a bias that is added to the sum to compare the result to 0.
- Each neuron has two states: active(1) & inactive(0)
- Summing up the products + the bias $\sum x_i \cdot w_i + bias$, then compared to 0 gives us either an output of 1 for sums ≥ 0 or a 0 if < 0 .
- Using this model, we can create conventional electronic gates such as AND-Gate, OR-Gate and NOT-Gate.
- However the XOR-Gate/NXOR-Gate is not possible in this model. Both functions are not linear.
- Similarities to biological neurons

Active or inactive state

Directionality(input/output)

Activity dependent of weighted functions of other neurons

- Differences to biological neurons
 - Continous time vs. discrete time
 - Degrees of activation
 - Activation as a function of the inputs of a real neuron is not linear.

7.1 Perceptron Learning Algorithm

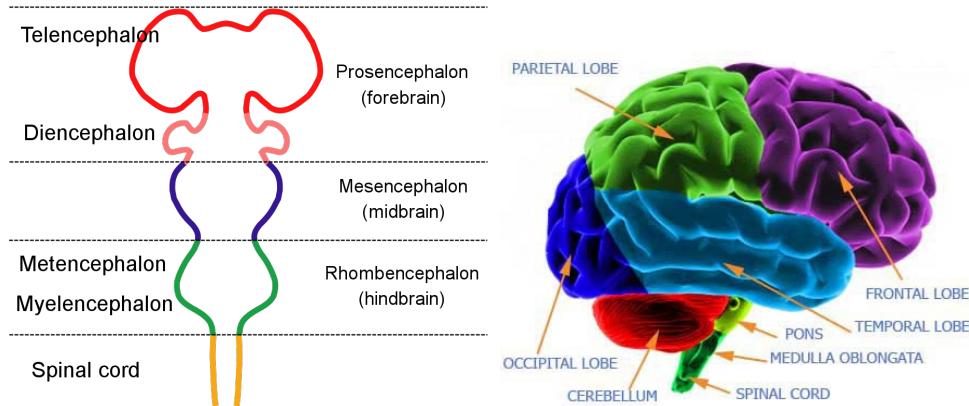
Given a set of (input vector x_i , desired output d_i) pairs, finds weights that produce the desired output d_i . (If such weights exist)

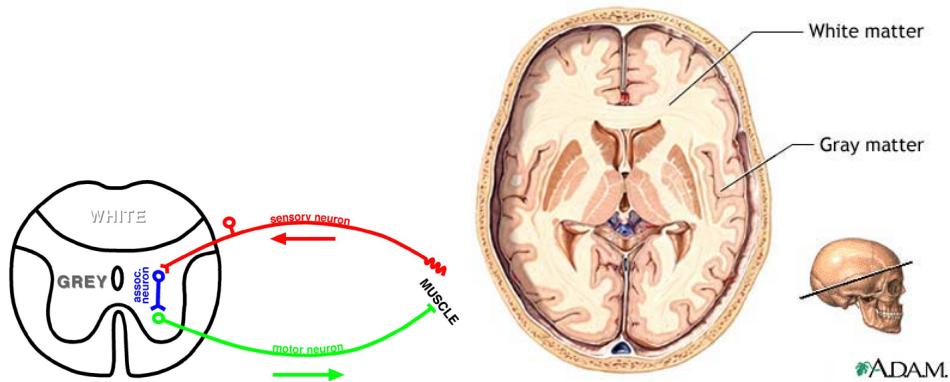
- Choose random weights
- Calculate actual output.
- $y_i = f[w(t) \cdot x_j] = \sum w_i \cdot x_i + \text{bias}$
- If wrong output \rightarrow change weights

- For every weight $w_i(t + 1) = w_i(t) + \alpha(d_j - y_j(t))x_i$
- This is repeated until the iteration error $\frac{1}{s} \sum_j^2 [d_j - y_j(t)]$ is less than a user-specified error threshold γ or until we completed a predefined number of iteration.
- Algorithm creates a sufficient result if a solution exists.

8 Lecture 8 - Nervous System Organization(Kevan Mastin)

- Brain consists of:
 - Spinal cord
 - Hindbrain
 - Midbrain - Thalamus
 - Forebrain - Neocortex, Hippocampus
- Whitematter
 - Glia cells, myelinated axons
- Greymatter
 - Neurons





White matter $1mm^3 \rightarrow 9m$ axons
 Grey matter $1mm^3 \rightarrow 50'000$ neurons
 → Long distance communication needs space

9 Lecture 9 - Synapse 2(Kevan Mastin)

- Sherrington (1873)

First research on synapses

- Vagus nerve

Stimulating the vagus nerve slows down the heart beat → inhibitory function.

- Synapse

Only vesicles which are already on the presynaptic membrane will be released after the AP (not all vesicles are released after an AP)

One single synapse produces only a small potential. it needs many synapses to create an actual AP. → Release of neurotransmitters is Ca dependent.

- Probabilistic release of neurotransmitter

Presynaptic problem

All synapses have probabilistic release

Factors (# of synapses, # of postsynaptic receptors) = Plasticity

10 Lecture 10 - Rate/Event Coding (Michael Pfeiffer)

- What is neural coding?

How is information encoded?

Single neuron firing \leftrightarrow Population firing

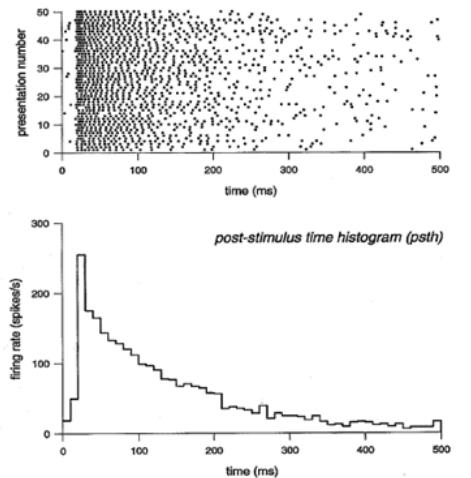
How does a neuron encode information?

Firing rate, Timing of spikes

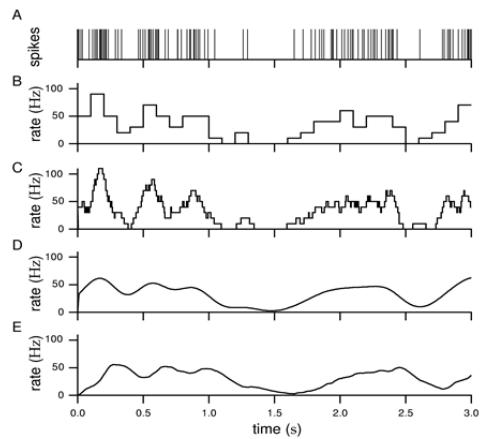
What do different measurement techniques tell us about the neural code?

Spatial/temporal resolution

What is a useful visualization of firings for interpretation?



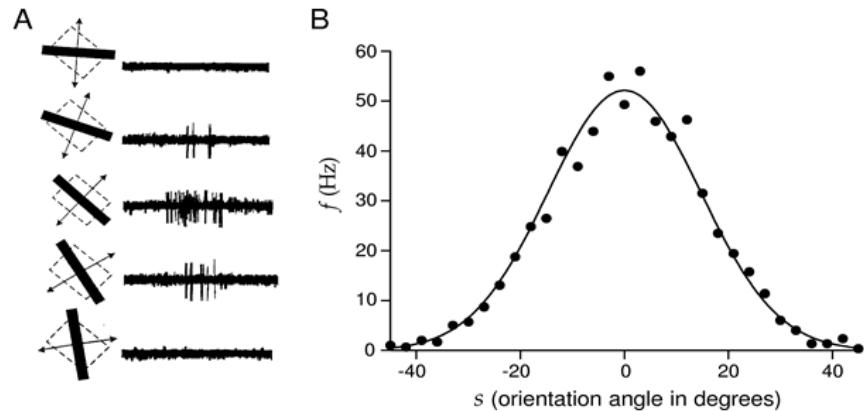
(a) Raster plot = spikes & Histogram



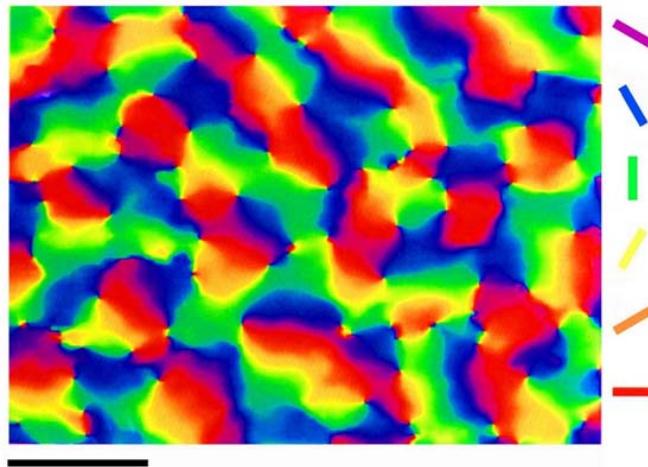
(b) 100ms time bins \rightarrow 100ms sliding window \rightarrow gauss filter \rightarrow causal filter

10.1 Neuronal rate codes(average over time(single neuron))

- Tuning curves



Shown line and its response primary visual cortex. Turning curve shows average firing rate of varying stimulus parameters. Tuning curves characterize a single cell.



Nearby neurons have similar preferred orientations(colors)

- Rate codes
 - + easy to understand
 - No timing effects
 - Might be misleading (More than one stimulus might be encoded)

10.2 What can a single neuron encode?

- Places (on entering a particular region)

- Grids (regularly arranged triangular grid of locations)
- Head-direction
- Single cell responds to one single human face ("Grandmother cell")

10.3 Population rate(average over pool of equivalent neurons)

- Population codes

Different cells encode different range of the stimulus → allows accurate reconstruction of the signal (sparse coding, exp. 3 types of color cones in retina)

Population vector code

Populations of neurons stand for vector directions, encoded direction is vectorial addition weighted by firing rate.

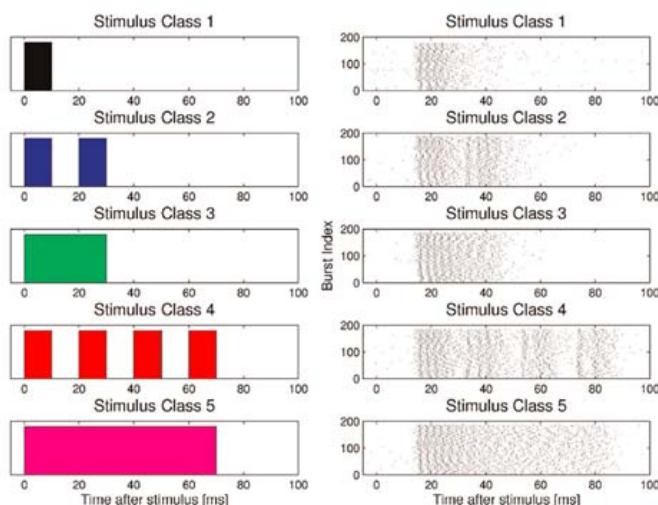
Neuronal Event codes

Time-to-first spike codes

- Can implement competition among different cells
- Can be rank-order code (sequence matters)

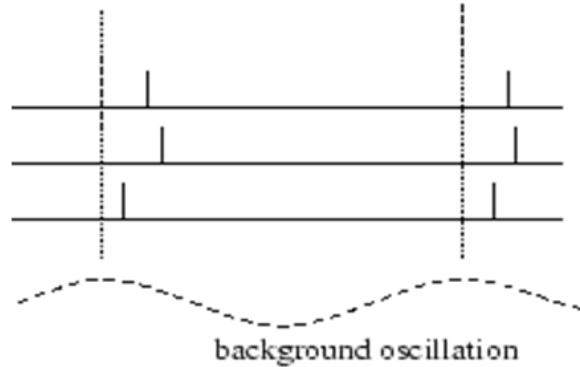
Burst- and Temporal Codes

Bushcricket auditory neurons in natural environment preserve very high coding precision in extreme noise



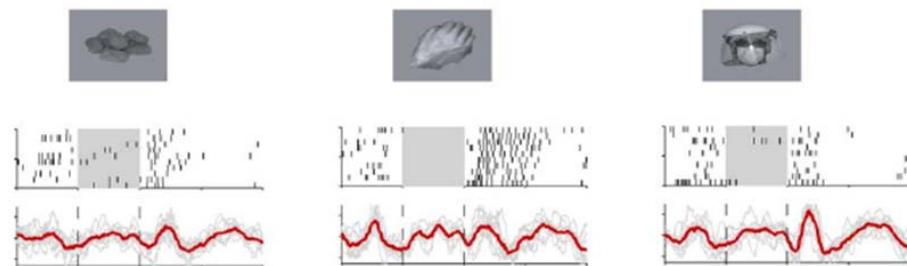
Oscillations and Phase Coding

The neurons fire at different phases with respect to the background oscillation



Local Field Potential(LFP)

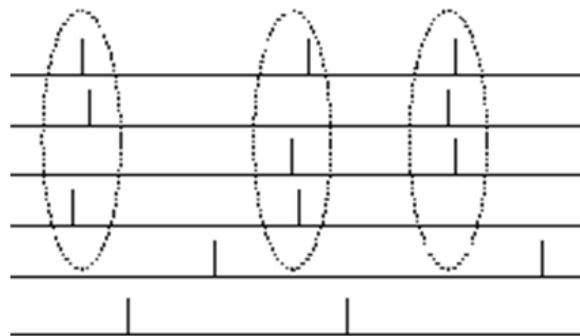
- Low-pass filtered extracellular recording
- Reflects the integration of membrane currents in a local region
- Dominated by dendritic synaptic activity
- Might encode different properties of the stimulus than single cell firing
- Where does it come from / what does it show?



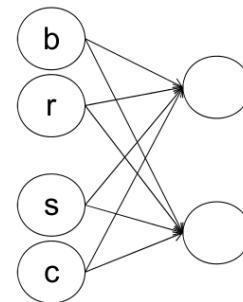
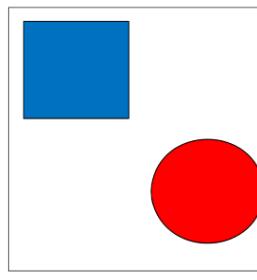
MUA(upper signal) & LFP (lower signal)

fMRI (functional magnetic resonance imaging)
based on blood oxygenation level

Synchrony coding



10.4 Binding problem



Occurs frequently:

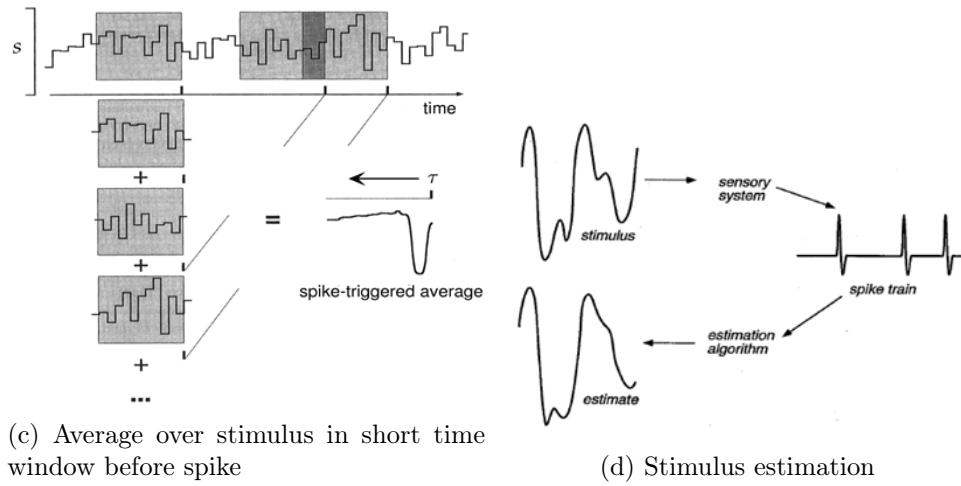
- Visual processing (what? where?)
- Memory
- etc.

Potential mechanisms:

- Temporal synchrony
- Hierarchical coding
- Population coding

10.5 Averages & Estimation

- Spike Triggered Average



- Issues to remember:

Whole stimulus reconstruction may not be relevant

Evolution may have shaped us to encode particular features better than others (e.g. faces)

Cells may respond to only particular aspects of stimulus

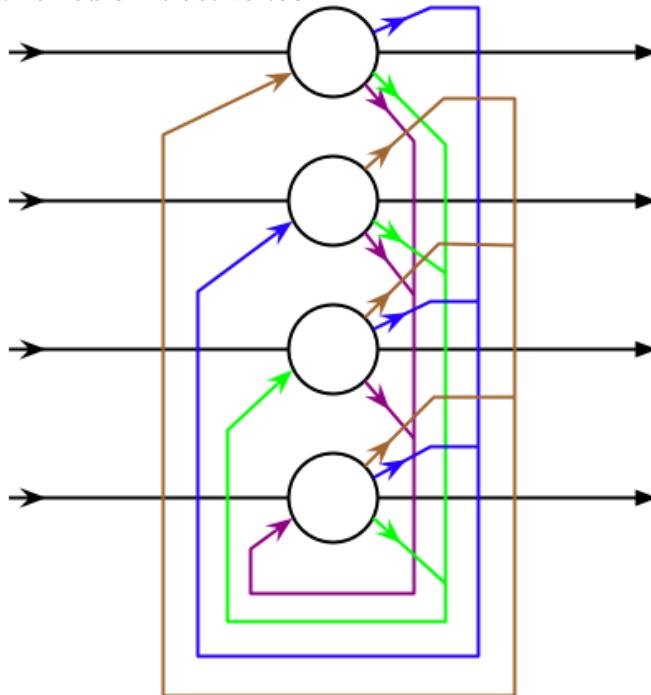
Cells may respond to multiple aspects of stimulus

Artificial stimuli used for studies may be predictable

11 Lecture 11 - Hopfield Networks(Matthew Cook)

- In a hopfield network, every node is connected to every other node(One single layer of nodes). The network of nodes is in some state at any time. Some of the states are stable and some are not. The nodes are connected by edges with weights and each node holds a value of -1/1 or 0/1. While the network is not in a stable state, updating the network leads to a state change that ultimately converges to a certain stable state (local minimum). The update of the nodes can be done synchronous or asynchronous over the nodes.
- Synchronous updates yields a stable pattern or a cycle of 2 patterns.
- Asynchronous updates are done with a greedy min-cut algorithm.

- When we update a node, we consider weights to that node from all other active nodes.
- If the sum of all weights of the active nodes is higher than zero then this neuron is active too.

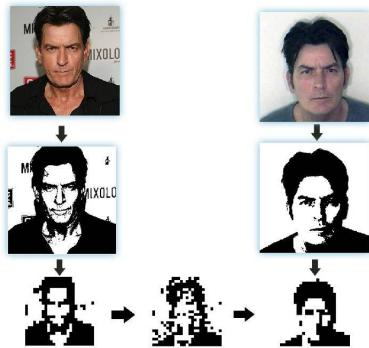


Update function: $a_{i,t}$ = output of unit at time t

$$a_{i,t+1} = \begin{cases} 1 & \sum_{j=1}^N a_{j,t} \cdot w_{ij} > \theta_i \text{ (often 0)} \\ -1 & \text{otherwise} \end{cases}$$

Features:

A hopfield network is an associative type of memory just as the human memory and is able to remember states it learned before. A Hopfield network of 100 nodes can store about approximately 15 pictures (stored as local minima in the network or stable states). Important is that the pictures are distinct, otherwise it can be that the network gets new stable states and the pictures only get "half remembered".



12 Lecture 12 - Feed-Forward Networks(Matthew Cook))

- Feed-Forward Network with back-propagation is a network that is single-directed(multiple layers of nodes) and has a certain number of inputs x and a certain number of outputs f . Every layer of nodes "feeds" the next layer with information.
- In many applications the nodes use a sigmoid function as an activation function.
- Back-propagation is a way method that lets information flow backwards inside of the single directed network, but that seems not the way biology is doing it. It is a learning technique that compares the output values f_i to the desired values g_i to compute corrective values with a predefined error function.

$$E = \sum(f_i - g_i)^2$$

- The error is then propagated back through the network to adjust the weights of each connection to reduce the deviation from the desired values. The network will converge to either no or just a small error.
- To adjust weights properly, one optimizes by a method called gradient descent:

$$\frac{\delta E}{\delta w_k} = \sum 2(f_i - g_i) \cdot \frac{\delta(f_i - g_i)}{\delta w_k} = \sum 2(f_i - g_i) \frac{\delta f_i}{\delta w_k}$$
- Feed inputs to network one at a time. Compare output to desired output $f_i - g_i$
- For each weight, compute the sensitivity of output $\frac{\delta f_i}{\delta w_k}$

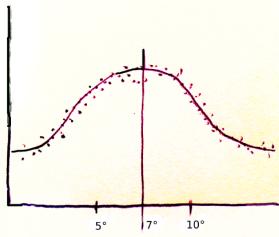
- Adjust weights w_k by $\epsilon \cdot (f_i - g_i) \frac{\delta f_i}{\delta w_k}$

13 Lecture 13 - Interacting Neural Populations(Matthew Cook)

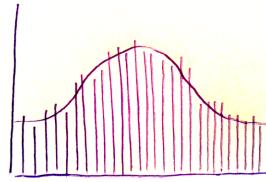
Population codes

Idea: Information is represented by the pattern of activity in a neural population. Specially, neurons are "tuned" to preferred stimuli.

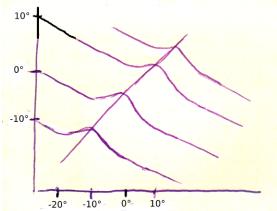
- We train a monkey to hold its gaze fixed while we move a visual stimulus around. The monkey holds its gaze fixed at $E = -15^\circ / 0^\circ / 15^\circ$ (eye angle) while we move the stimulus from $R = -40^\circ \dots 40^\circ$ (retinal angle).



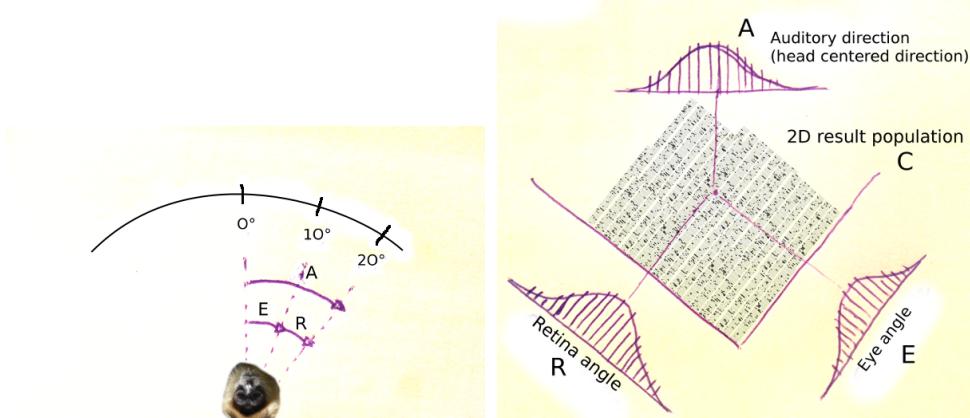
(e) Tuning curve of one cell



(f) Cells ordered by response to 20°

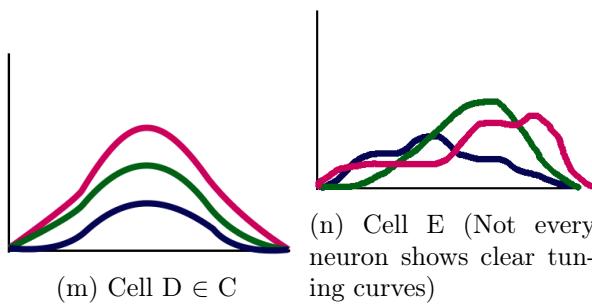
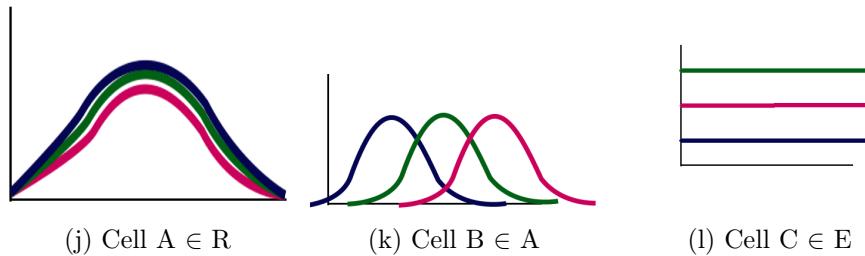


(g) 3D visualization of cell's response to different degrees



(h) Monkey holding gaze fixed on point 10° and light falling in from 20°

(i) Visualization of Retina angle ordering cell set R, Eye angle ordering set E, 2D result population C and auditory direction set A.



14 Lecture 14 - Neuromorphic VLSI(Giacomo Indiveri)

14.1 VLSI

- Very Large Scale Integration Technology allows us to fabricate chips and memories. Digital VLSI(Today's computers) not analog, not low power, not fault tolerant, not robust to inhomogeneities, not asynchronous (clocked), not massively parallel

- Neuromorphic = VLSI systems containing electronic analog/digital circuits that exploit the physics of silicon to reproduce the bio-physics of neural circuits present in the nervous system. Two main goals:

To understand the computational properties of biological neural systems using standard CMOS VLSI technology as a tool.

To exploit the known properties of biological systems to design and implement efficient devices for engineering applications.

- Neuromorphic VLSI neuron circuits

To reproduce the physics of neural computation using subthreshold analog circuits and asynchronous digital circuits.

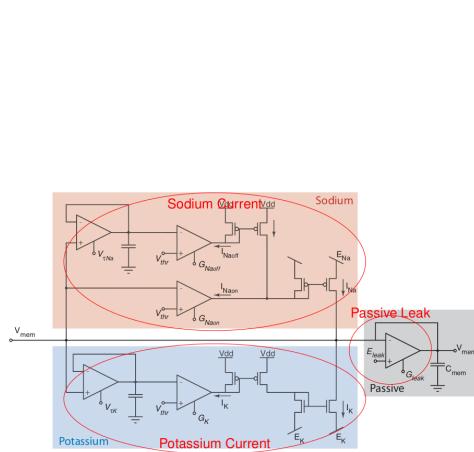
To build autonomous learning behaving systems that can interact with the environment in real-time

14.2 Why VLSI for neural computation?

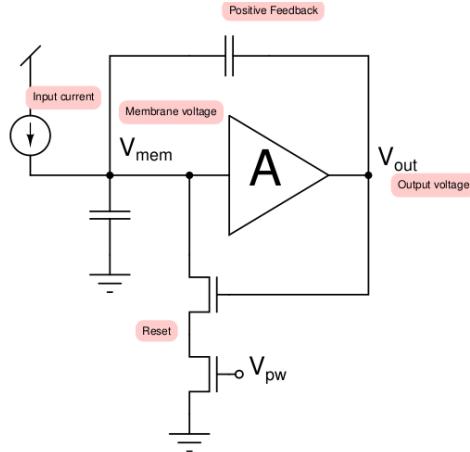
- Best exploit current and future VLSI technologies
- Optimally suited for nano- and future emerging technologies
- Ideal tools for real- and accelerated-time modeling of neural systems
- Compact, low-power sensory processing devices for autonomous/flying robots, embedded systems, etc.
- Direct interface to living systems

14.3 Different remarkable circuits

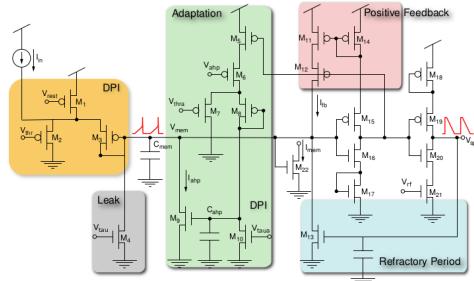
- Artificial neuron model by McCulloch & Pitts
- Integrate & fire model (I&F)



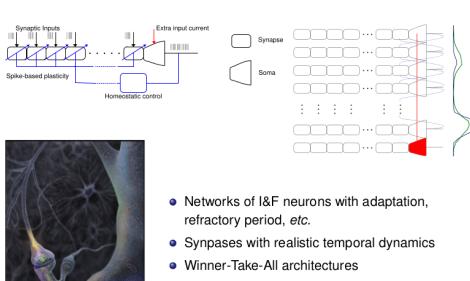
(o) Conductance-based silicon neuron



(p) Axon-Hillock-circuit



(q) Ultra low-power generalized I&F circuit



(r) Spiking multineuron architectures