Mathematical Foundations of Neuroscience - Lecture 14. Synchronization of neurons and synaptic plasticity

Filip Piękniewski

Faculty of Mathematics and Computer Science, Nicolaus Copernicus University, Toruń, Poland

Winter 2009/2010

- Neurons are either in excitable state, in which they are sensitive to incoming signals , or excited in which they become oscillators.
- Some neurons are also capable of bursting, which is a composition of oscillations working on different timescales
- Eventually, however neurons need to be connected in a network to perform any complex operations expected from the nervous systems
- One of the basic abilities of nervous systems is learning, a process in which some properties of a neural ensemble change
- Learning (at least some forms of learning) is most probably done in synapses.

Mean field approximations

- Having a set of items difficult to analyze it is sometimes convenient to get more such items!
- In particular heaving infinitely many complex items can make things a lot simpler, than having a finite number of them.
- The limit with the number of interacting items $n \to \infty$ is called a thermodynamic limit.
- The analogies here are such: it is easier to predict the action of a crowd than of an individual person. It is easier to predict the behavior of a volume of a gas, than a single particle.
- The field of science which plays with such approximations is called *statistical mechanics*. Lets see what it can do with coupled oscillators.

- Consider the Kuramoto model:

$$\varphi_i' = \omega_i + \frac{K}{n}\sum_{j=1}^{n}\sin(\varphi_j - \varphi_i)$$

where $K > 0$ is the coupling strength, and $\frac{1}{n}$ ensures that the model makes sense when $n \to \infty$

- Consider the complex valued sum

$$\frac{1}{n}\sum_{j=1}^{n} e^{i\varphi_j}$$

Any $e^{i\varphi_j}$ lays on the unit circle, and the sum is barycentric, so the value of the sum lays in the unit disc.

- Denote:

$$r = \left| \frac{1}{n} \sum_{j=1}^{n} e^{i\varphi_j} \right|, \qquad \psi = \arg\left(\frac{1}{n} \sum_{j=1}^{n} e^{i\varphi_j} \right) = \frac{1}{n} \sum_{j=1}^{n} \varphi_j$$

- we have

$$re^{i\psi} = \frac{1}{n} \sum_{j=1}^{n} e^{i\varphi_j}$$

The value $re^{i\psi}$ is called Kuramoto synchronization index, r is called the order parameter. Note that synchronized state $\varphi_i = \text{const}$ has $r = 1$. Incoherent state on the other hand with all phases chosen randomly from the unit circle has $r \approx 0$.

Now we have

$$re^{i\psi} = \frac{1}{n}\sum_{j=1}^{n} e^{i\varphi_j} \qquad / \cdot e^{-i\varphi_i}$$

$$re^{i(\psi-\varphi_i)} = \frac{1}{n}\sum_{j=1}^{n} e^{i(\varphi_j-\varphi_i)}$$

$$\Downarrow$$

$$\Im\left(re^{i(\psi-\varphi_i)}\right) = \Im\left(\frac{1}{n}\sum_{j=1}^{n} e^{i(\varphi_j-\varphi_i)}\right)$$

$$r\sin(\psi-\varphi_i) = \frac{1}{n}\sum_{j=1}^{n}\sin(\varphi_j-\varphi_i)$$

- Substituting in the original formula we obtain:

$$\varphi_i' = \omega_i + Kr\sin(\psi - \varphi_i)$$

- From this expression we can deduce that the oscillators are pulled into the direction of a synchronized cluster (with phase ψ) with the strength r proportional to the size of the cluster size.
- Now lets assume that frequencies ω_i are randomly distributed around 0 with symmetric probability density function $g(\omega)$, and moreover lets consider the limit $n \to \infty$. We can assume without the loss of generality that the cluster $\psi = 0$. We have

$$r = re^{i0} = \lim_{n\to\infty} \frac{1}{n}\sum_{j=1}^{n} e^{i\varphi_j} = \int_{\mathbb{S}^1} e^{i\varphi(\omega)} g(\omega) d\omega$$

- Now the network consists of elements oscillating with the cluster $|\omega| < Kr \Rightarrow \omega + Kr\sin(0-\varphi) = \omega - Kr\sin(\varphi) < 0$ for $\varphi \approx 0$, and those not oscillating with the cluster. The contributions from the other oscillators cancel each other on average (because they are drifting over the circle) and we have:

$$r = \int_{\mathbb{S}^1} e^{i\varphi(\omega)} g(\omega) d\omega \approx \int_{|\omega|<Kr} e^{i\varphi(\omega)} g(\omega) d\omega$$

- Since g is symmetric, the imaginary parts of $e^{i\varphi(\omega)}$ cancel out and:

$$r \approx \int_{|\omega|<Kr} e^{i\varphi(\omega)} g(\omega) d\omega = \int_{|\omega|<Kr} \cos\varphi(\omega) g(\omega) d\omega$$

- The condition for locking with the cluster is

$$0 = \omega + Kr\sin(0-\varphi) \quad \Leftrightarrow \quad \omega = Kr\sin(\varphi)$$

- Substituting $\omega = Kr\sin(\varphi)$ we obtain:

$$r \approx \int_{|\omega|<Kr} \cos\varphi(\omega) g(\omega) d\omega = rK \int_{-\pi/2}^{\pi/2} g(rK\sin\varphi)\cos^2\varphi d\varphi$$

the self-consistency equation. Note that incoherent $r = 0$ is always a solution to the equation. Assume for a moment that $r \neq 0$. Then:

$$1 = K\int_{-\pi/2}^{\pi/2} g(rK\sin\varphi)\cos^2\varphi d\varphi < K\int_{-\pi/2}^{\pi/2} g(0)\cos^2\varphi d\varphi$$

- This imposes:

$$\frac{1}{K} < g(0) \int_{-\pi/2}^{\pi/2} \cos^2 \varphi d\varphi$$

- Now recall (from high school?) that $\cos 2\varphi = \cos^2 \varphi - \sin^2 \varphi = 2\cos^2 \varphi - 1$ and so $\cos^2 \varphi = \frac{1+\cos 2\varphi}{2}$, so

$$\frac{1}{K} < g(0)\frac{1}{2} \int_{-\pi/2}^{\pi/2} 1 + \cos 2\varphi d2\varphi = g(0)\frac{1}{4}\left(2\pi + \int_{-\pi}^{\pi} \cos \varphi d\varphi\right)$$

$$= g(0)\frac{1}{4}(2\pi + 0) = \frac{g(0)\pi}{2}$$

Consequently for $K > K_c = \frac{2}{\pi g(0)}$ another, partially synchronized solution emerges.

SNIC oscillators

- Before we investigate synapses, we will analyze an example of how theory of coupled oscillators works with Saddle-node on invariant circle oscillators.
- Recall that a neuron close to the saddle node on invariant circle bifurcation can be expressed (by a continuous change of variables) in a canonical model

$$\frac{dx}{dt} = 1 + x^2$$

  The model is called a quadratic integrate and fire neuron.
- x diverges to infinity in finite time. In models we usually assumed that there is a cutoff value, and a reset.

SNIC oscillators

- For theoretical studies we may use a different approach: identify $-\infty$ and ∞, and assume the model is defined in an extended real line $\mathbb{R} \cup \{-\infty, \infty\}$. In such a case we obtain an oscillator.
- The solution of

$$\frac{dx}{dt} = 1 + x^2$$

that originates at $-\infty$ is $-\cot x$ since

$$(-\cot t)' = \frac{1}{\sin^2 t} = \frac{\sin^2 t}{\sin^2 t} + \frac{\cos^2 t}{\sin^2 t} = 1 + \cot^2 t$$

- Let us now derive the PRC. We know that the solution is $x(t) = \cot t = \tan\left(t - \frac{\pi}{2}\right)$ with period $T = \pi$. Inverting the formula we get:

$$t = \frac{\pi}{2} + \operatorname{arctan}(x)$$

Now assume the state of the system jumps by some value A, so $x(t) = \cot t + A$. PRC is the phase difference:

$$\begin{aligned}
\mathrm{PRC}_A(\vartheta) = \vartheta_{\mathrm{new}} - \vartheta &= \frac{\pi}{2} + \operatorname{arctan}(x + A) - \frac{\pi}{2} - \operatorname{arctan}(x) = \\
&= \operatorname{arctan}\left(\cot\vartheta + A\right) - \operatorname{arctan}\left(\cot\vartheta\right) = \\
&= \operatorname{arctan}\left(\tan\left(\vartheta - \frac{\pi}{2}\right) + A\right) - \operatorname{arctan}\left(\tan\left(\vartheta - \frac{\pi}{2}\right)\right) = \\
&= \operatorname{arctan}\left(\tan\left(\vartheta - \frac{\pi}{2}\right) + A\right) - \left(\vartheta - \frac{\pi}{2}\right)
\end{aligned}$$

- Note that:

$$\frac{\partial \mathrm{PRC}(A,\vartheta)}{\partial A} = \frac{\partial}{\partial A}\arctan\left(\tan\left(\vartheta-\frac{\pi}{2}\right)+A\right) =$$
$$= \frac{1}{1+(\cot(\vartheta)+A)^2} \approx |A\approx 0| \approx \frac{1}{1+(\cot(\vartheta))^2} =$$
$$= \sin^2\vartheta = \mathrm{iPRC}(\vartheta)$$

so for small A where we assume that the PRC scales linearly we have $\mathrm{PRC}(A,\vartheta) = A\sin^2\vartheta$

Weak coupling

- Given the infinitesimal PRC we can transform the weakly coupled system:

$$\frac{dx}{dt} = 1 + x^2 + \varepsilon p(t)$$

into an equivalent phase model:

$$\frac{d\vartheta}{dt} = 1 + \varepsilon \sin^2(\vartheta) p(t)$$

- We may note right away, that the system is insensitive to inputs occurring during the spike (when $\sin^2 \vartheta \approx 0$). The system is most sensitive to inputs near the ghost attractor. Furthermore $\sin^2 \vartheta > 0$ for all ϑ so excitatory inputs can only advance the spike, while inhibitory inputs can only delay the spike.

Gap junctions

- Recall that electrical synapse (gap junction) conducts current from one neuron to another proportionally to its intrinsic conductance and the potential difference between the membranes.
- Assume we have two SNIC oscillators coupled via gap junctions:

$$\frac{dx_1}{dt} = 1 + x_1^2 + \varepsilon(x_2 - x_1)$$
$$\frac{dx_2}{dt} = 1 + x_2^2 + \varepsilon(x_1 - x_2)$$

Gap junctions

- The system can be converted into a phase model:

$$\frac{d\vartheta_1}{dt} = 1 + \varepsilon \sin^2(\vartheta_1)(\cot\vartheta_1 - \cot\vartheta_2)$$
$$\frac{d\vartheta_2}{dt} = 1 + \varepsilon \sin^2(\vartheta_2)(\cot\vartheta_2 - \cot\vartheta_1)$$

which can be further simplified and expressed in terms of phase deviation coordinates using the average interaction function:

$$H(\chi) = \frac{1}{\pi}\int_0^\pi \sin^2 t(\cot t - \cot(t+\chi))dt = \frac{1}{2}\sin 2\chi$$

where $\chi = \vartheta_2 - \vartheta_1$

Gap junctions

- Finally we obtain

$$\frac{d\varphi_1}{dt} = \frac{\varepsilon}{2}\sin(2(\varphi_2 - \varphi_1))$$
$$\frac{d\varphi_2}{dt} = \frac{\varepsilon}{2}\sin(2(\varphi_1 - \varphi_2))$$

- Subtracting the equations we find that the phase difference $\chi = \varphi_2 - \varphi_1$ satisfies:

$$\frac{d\chi}{dt} = -\varepsilon \sin 2\chi$$

- Following Ermentrout's condition we find that the synchronized state $\chi = 0$ is stable ($(\sin 0)' > 0$), while the antiphase $\chi = \frac{\pi}{2}$ is not.

Weak pulses

- Now assume the two oscillators are pulse coupled, that is each oscillator is reset by a delta function at the moment the other "spikes". The system

$$\frac{dx_1}{dt} = 1 + x_1^2 + \varepsilon_1 \delta(t - t_2)$$
$$\frac{dx_2}{dt} = 1 + x_2^2 + \varepsilon_2 \delta(t - t_1)$$

has a phase model:

$$\frac{d\vartheta_1}{dt} = 1 + \varepsilon_1 \sin^2(\vartheta_1)\delta(t - t_2)$$
$$\frac{d\vartheta_2}{dt} = 1 + \varepsilon_2 \sin^2(\vartheta_2)\delta(t - t_1)$$

- Note that

$$H(\chi) = \frac{1}{\pi}\int_0^{\pi} \sin^2(t)\delta(t+\chi)dt = \frac{1}{\pi}\sin^2\chi$$

(convolution of any function with δ returns the original function).

- The corresponding phase deviation model is

$$\frac{d\varphi_1}{dt} = \frac{\varepsilon_1}{\pi}\sin^2(\varphi_2-\varphi_1)$$
$$\frac{d\varphi_2}{dt} = \frac{\varepsilon_2}{\pi}\sin^2(\varphi_1-\varphi_2)$$

- The phase difference satisfies:

$$\frac{d\chi}{dt} = \frac{\varepsilon_2-\varepsilon_1}{\pi}\sin^2\chi$$

Weak pulses

- Since
$$\frac{d\chi}{dt} = \frac{\varepsilon_2 - \varepsilon_1}{\pi} \sin^2 \chi$$
when the coupling is identical ($\varepsilon_1 = \varepsilon_2$) we have
$$\frac{d\chi}{dt} = 0$$
and the oscillators preserve their initial phase difference.
- If $\varepsilon_1 \neq \varepsilon_2$ the equilibrium $\chi = 0$ is only neutrally stable $\chi'(0) = 0$, and it becomes unstable for networks three and more pulse coupled SNIC oscillators.

Weak synapses

- Now assume the coupling is no longer delta, but some long lasting $p(t)$

$$\frac{dx_1}{dt} = 1 + x_1^2 + \varepsilon_1 p(t - t_2)$$
$$\frac{dx_2}{dt} = 1 + x_2^2 + \varepsilon_2 p(t - t_1)$$

has a phase model becomes:

$$\frac{d\vartheta_1}{dt} = 1 + \varepsilon_1 \sin^2(\vartheta_1) p(t - t_2)$$
$$\frac{d\vartheta_2}{dt} = 1 + \varepsilon_2 \sin^2(\vartheta_2) p(t - t_1)$$

Weak synapses

- The phase deviation interaction function $H(\chi)$ is given by:

$$H(\chi) = \frac{1}{\pi}\int_0^{\pi} \sin^2(t)p(t+\chi)dt$$

- The phase synchronized solution $\chi = 0$ i stable when $H(0)' > 0$, that is:

$$\left.\frac{dH(\chi)}{d\chi}\right|_{\chi=0} = \frac{1}{\pi}\int_0^{\pi} \sin^2(t)p'(t)dt > 0$$

- Now everything depends where $p'(t)$ is positive/negative with respect to $\sin^2(t)$ which weights the integral.

Synaptic plasticity and learning

- Coupled oscillators can be cleverly arranged to perform certain complex computational tasks
- However the brain is not explicitly wired to include all of the complex tasks that it may come across during lifetime. If it were, we wouldn't have to learn anything!
- But the brain is capable of learning new actions and abilities, therefore it has to be plastic and rewire itself.
- There are many ways of learning, and many types of memory. Short term memory can be encoded in the neural activity (by keeping the neural ensemble in some kind of an attractor). Long term memory is encoded in connectivity and synapses.

Hebbian learning

- The foundations of synaptic learning were established by a Canadian scientist, Donald Olding Hebb.
- In his important book "The Organization of Behavior" (1949) he noted: *When an axon of cell A is near enough to excite cell B and repeatedly or persistently takes part in firing it, some growth process or metabolic change takes place in one or both cells such that A's efficiency, as one of the cells firing B, is increased*
- In other words: "Neurons that fire together wire together"
- If we denote by σ_i and σ_j the activity of i-th and j-th neuron respectively, then for $\eta > 0$

$$\frac{dw_{ij}}{dt} = \eta \sigma_i \sigma_j$$

Hebbian learning

- Hebb's original formulation is rather general, and there are may ways to implement his principle. The weight for example may depend on the average cooperative activity in response to some number of samples:

$$w_{ij} = \frac{1}{n} \sum_{k=1}^{n} \sigma_i(\xi_k)\sigma_j(\xi_k)$$

  note that when new sample ξ arrives the weight change is proportional to $\sigma_i(\xi)\sigma_j(\xi)$ as postulated.
- σ may denote the average spiking rate in response to a pattern, and is usually assumed to $\sigma \in [0, 1]$ or even binary $\sigma \in \{0, 1\}$

Hebbian learning

- Hebb's principle is very general and useful, many neural algorithms rely on it in the theory of artificial neural networks (where a neuron is usually assumed to sum the input and wrap it with sigmoidal activation function).
- However essentially the synaptic weight formula is unstable, and requires renormalization (otherwise the weights would hit infinity). Various renormalization techniques are used, for example Oja rule (after prof. Erkki Oja):

$$\frac{dw_{ij}}{dt} = \eta \sigma_j (\sigma_i - \sigma_j w_{ij})$$

- Oja rule is self normalizing (when w_{ij} becomes large, it dominates the equation and converges).

Bienenstock-Cooper-Munro (BCM) synapse

- Some experiments have shown that the synaptic plasticity satisfies the following properties. The change of weights w_{ij} is:
  - proportional to presynaptic activity σ_i
  - proportional to a non monotonic function of postsynaptic activation $\Phi(\sigma_j)$. For low σ_j the weights are decreased, while for larger increased. The border point between growth and decay is called the activation threshold θ_M
  - θ_M is itself a superlinear (growing faster than linear) function of the history of postsynaptic activity σ_j
- There are many possible rules that satisfy the above conditions, for example

$$\frac{dw_{ij}}{dt} = \eta\sigma_j(\sigma_j - \theta_M)\sigma_i - \varepsilon w_{ij}$$

Bienenstock-Cooper-Munro (BCM) synapse

- Where for example

$$\theta_M(t) = \frac{1}{\vartheta}\int_{-\infty}^{t} \sigma_j^2(\tau) e^{\frac{t-\tau}{\vartheta}} d\tau$$

  is the decaying temporal average of postsynaptic activity σ_j
- BCM synapses result in neuronal selectivity (neuron becomes sensitive to only one input pattern), and competition. It also explains the phenomenon of synaptic scaling, in which the synapses scale with the reciprocal of the average neuronal activity (synapses get stronger when activations become weaker due to metaplasticity of the variable activation threshold).

Spike timing dependent plasticity (STDP)

- Neither Hebb's principle, neither Oja rule, nor BCM takes into account temporal structure of spikes arriving a the pre and postsynaptic neurons (though Hebb's original formulation mentioned of a presynaptic activity contributing to a postsynaptic spike).
- Spike Timing Dependent Plasticity is a synapse modifying rule, that relies on temporal structure of spikes in ms scale. STDP has been found experimentally in many synapses. In many aspects it materializes Hebbian learning, but with respect to single spikes, not spiking rates.
- STDP has been discovered by Henry Markram and others in mid 90'ties.

Spike timing dependent plasticity (STDP)

- STDP modifies the synapse by taking into account relative distances of pre and postsynaptic spikes.
- Conceptually, if presynaptic spike precedes postsynaptic one by a short period of time, then probably the presynaptic activity contributed to the postsynaptic spike. In such cace synapse should be amplified (in agreement with Hebbian approach)
- If a presynaptic spike arrives late, then it is obvious it did not contribute to the postsynaptic activity. Therefore such synapse should be weakened.
- These principled get inverted for inhibitory connections.
- The influence of the spikes on the synapse should quickly decay as the relative distance between the spikes grows (since their mutual influence becomes negligible).

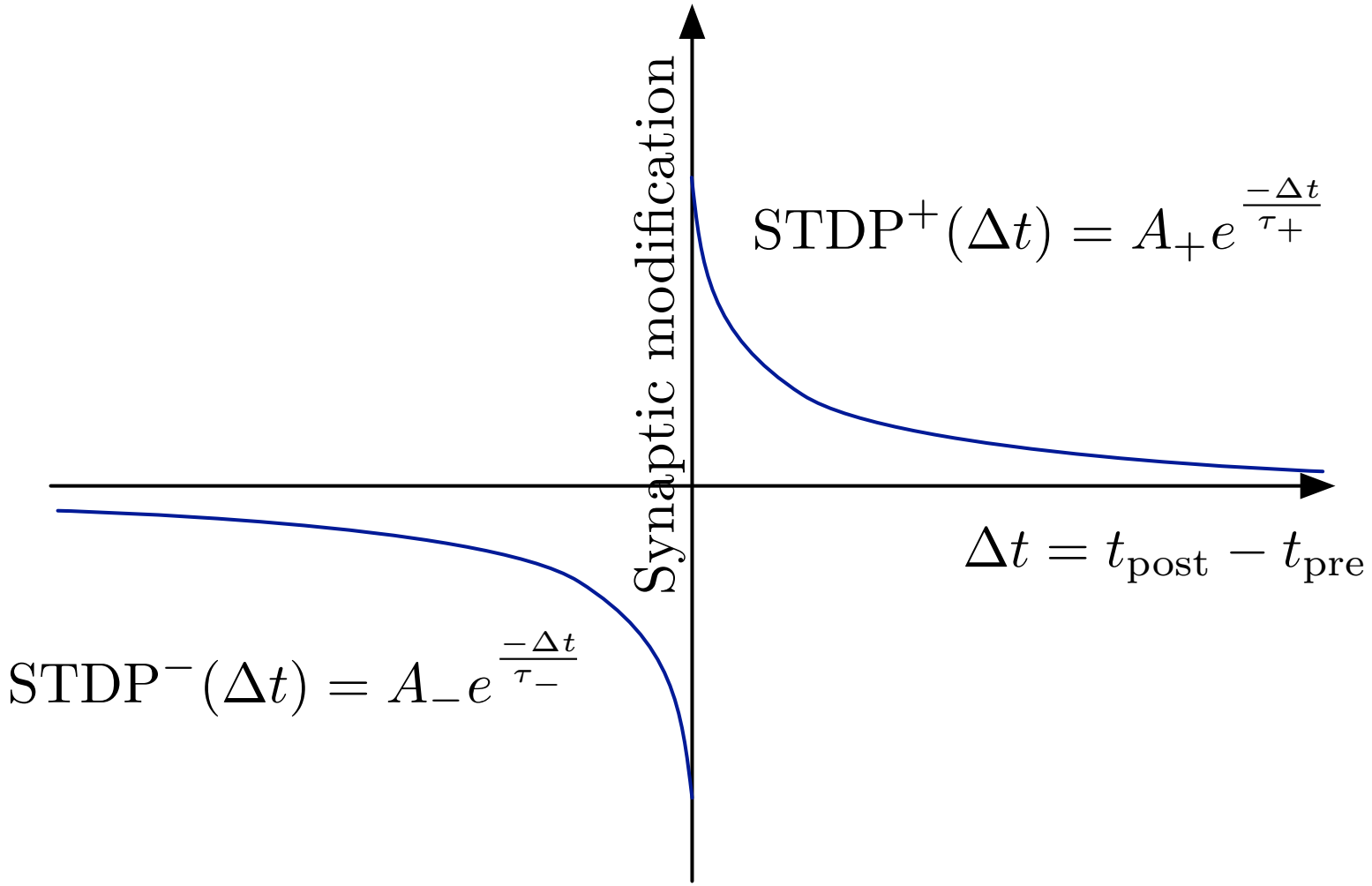

Figure: Spike timing dependent plasticity - diagram.

- The basic STDP model looks as follows:

$$\frac{dw_{ij}}{dt} = \sum_{s=1}^{n} \sum_{k=1}^{n} W(t_s^{(j)} - t_k^{(i)})$$

where $t_s^{(j)}$ is the time of s-th spike of postsynaptic neuron j, $t_k^{(i)}$ is the time of k-th spike of presynaptic neuron i, $W(t) = A_+ e^{t/\tau_+}$ for $t > 0$ and $W(t) = A_- e^{t/\tau_-}$ for $t < 0$. A_+, A_-, τ_+, τ_- are parameters.

- The summation goes over all pairs of spikes. There are other versions of STDP in which only nearest neighbors spikes contribute to plasticity. Other variants are also possible.

- Though seems complicated, the STDP as presented can be efficiently implemented online.
- Assume that each presynaptic spike leaves an exponentially decaying trace:

$$\tau_+ \frac{dP}{dt} = -P + \sum_{k=1}^{n} \delta(t - t_k^{(i)})$$

- Similarly each postsynaptic spike leaves a trace

$$\tau_- \frac{dD}{dt} = -D + \sum_{s=1}^{n} \delta(t - t_s^{(j)})$$

- The weight change is then:

$$\frac{dw_{ij}}{dt} = A_+ \cdot P \sum_{s=1}^{n} \delta(t - t_s^{(j)}) + A_- \cdot D \sum_{k=1}^{n} \delta(t - t_k^{(i)})$$

- The weight therefore is potentiated by $A_+ \cdot P$ at the time of postsynaptic spike, and depressed by $A_- \cdot D$ at the time of presynaptic spike.
- The implementation is quite straightforward, each synapse has two traces updated whenever spikes occur. Each pre(post)synaptic spike modifies the weight proportionally D(P) and increases P(D) (notice the inversion).
- Other variants of STDP can be obtained by limiting the sums (in practice this may require simulating additional traces).

- It turns out that nearest neighbors STDP is equivalent to BCM for weakly correlated Poissonian pre/post synaptic spike trains (Izhikevich, Desai 2003)
- Nevertheless is seems that STDP it the most accurate synaptic plasticity mechanism.
- If the area below the negative STDP curve is larger that of positive part, then on average depression will be stronger than potentiation, and the neural ensemble should converge to a homeostatic regime, with balanced synapses and spiking rates.
- It is however reasonable to limit the possible synapse growth and decay, either clipping or by wrapping the equations with a symmetric quickly decaying function to limit growth rate at the extreme regimes.

- In real life, the synapse may potentiate or depress as time progresses
- On the contrary to naive thinking, even if one synapse turns strong it may get depressed by subsequent input (once a synapse turns strong it may change to local spiking activity, and in turn diminish)
- After some time however, a kind of equilibrium may be achieved in which most of the synapses remain constant. Such a structured neuronal circuit may have interesting properties.
- If the model is also equipped with axonal delays, then the corresponding neuronal circuits can become very complex.

Dendritic and axonal delays

- Recall, that a neuron is a highly structured entity with dendritic tree and possibly a very long axon.
- There are many types of neurons found in various brain structures. The most common include:
  - Pyramidal neurons - triangular shaped soma, single axon emerging opposite to the dendritic tree. These are the basic building block of the cortex and thalamus.
  - Basket neurons - with freely branching dendrites, lack of axon. These cells are usually inhibitory interneurons.
  - Purkinje cell - very large cells with very complex dendritic tree, found in cerebellum
  - Granule cell - tiny cells found in various parts of the brain (including some layers of the cortex)

Dendritic and axonal delays

- When a spike is generated in a neuron, it will usually reach the soma, and from there it will proceed into the axon or to the opposite part of the dendritic tree.
- Things can become very complex (as we've seen in simulations), but on average the spike will reach terminal synapse after some delay.
- In a coarse grained setup we may as well assume, that each neuron is connected to some others with certain (order of ms) delays. Experiments show that the signal proceeds with the speed of about 0.15m/s. Signal are much faster in long myelinated fibers (axons), in which the speed o propagation reaches 1m/s

Dendritic and axonal delays

- Most long axons are surrounded by a layer of myelin, electrically insulating material made of lipids (80%) and proteins (20%) produced by tiny Schwann Cells.
- Myelin is white in appearance, and hence the fibers in the brain are called white matter.
- Myelinated axons are able to propagate signals as far as meters with enhanced speed (1m/s) and reliability.
- Propagation delays can therefore reach 10ms for non-myelinated local axonal collaterals (assuming the reach at most 1.5mm) and on the order of 100ms for myelinated connections assuming they reach 10cm within the brain (they may be longer in the spinal cord, spike the propagation velocities are somewhat larger as well).

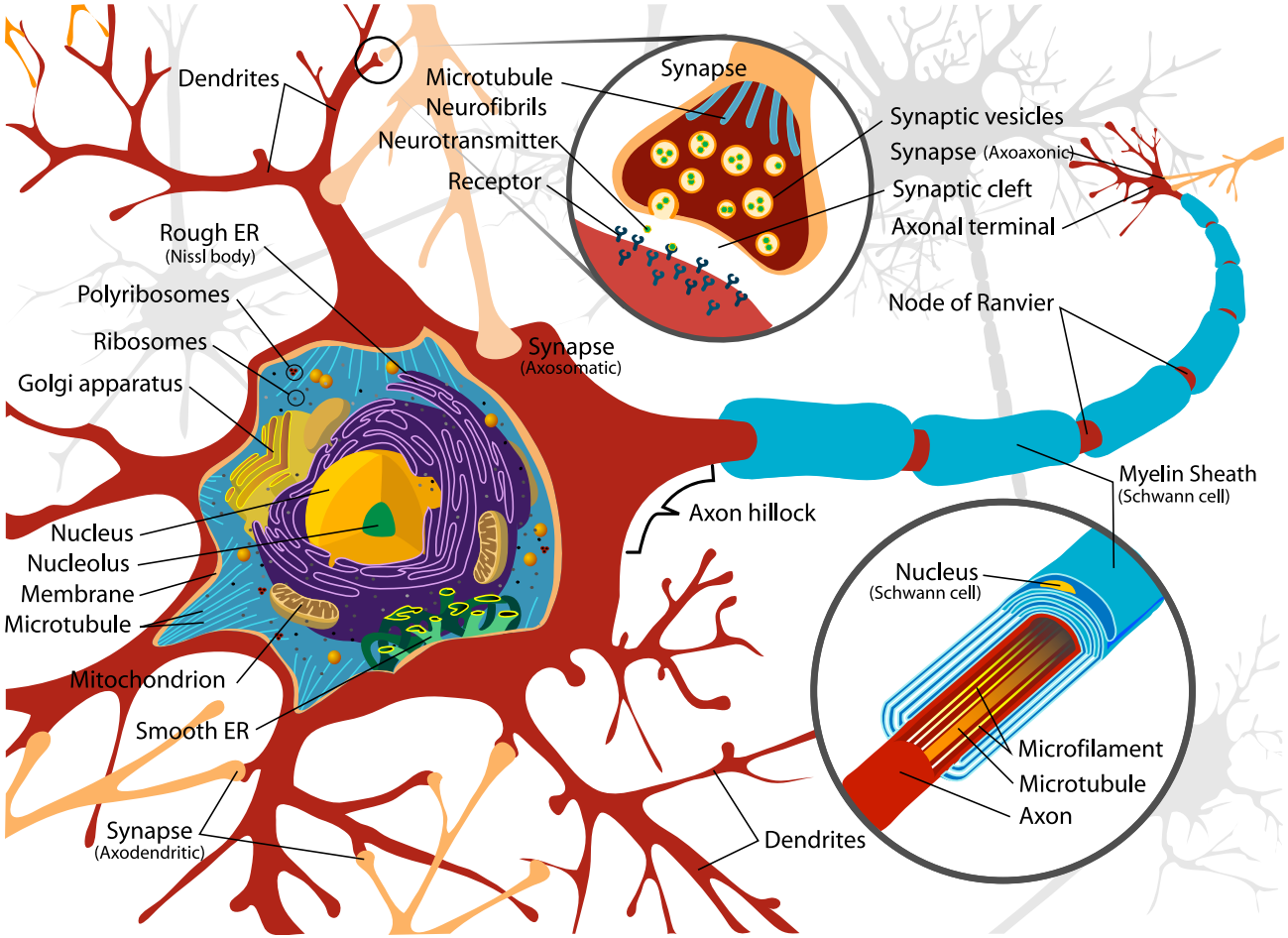

Figure: Complete neuron cell diagram. Image courtesy of Mariana Ruiz Villarreal (Available at: http://en.wikipedia.org/wiki/Image:Complete_neuron_cell_diagram_en.svg)

Polychronous groups

- Propagation delays in combination with spike timing dependent plasticity may result in spontaneous wiring of selective neuronal circuits.
- The firing within such a circuit are not synchronous, but time locked (depending on the local structure of axonal delays). E. Izhikevich coined a name *polychronous* to emphasize precise time locking, but possible lack of synchrony.
- STDP selects those connections that likely trigger postsynaptic spiking, while depressing others (therefore reducing noise). Such neuronal circuits are highly selective to input. It is likely that such spontaneous groups are the brains workhorse.

Polychronous groups

- A 2004 model by Izhikevich, Gally and Edelman shows the concept on an example of a neuronal sphere with 100000 neurons.
- The neurons are wired somewhat randomly, with some local connections and some global connections, conduction delays reaching 10ms and STDP and short depression/facilitation
- After some time the model reaches homeostatic regime, with fairly equal spiking rate.

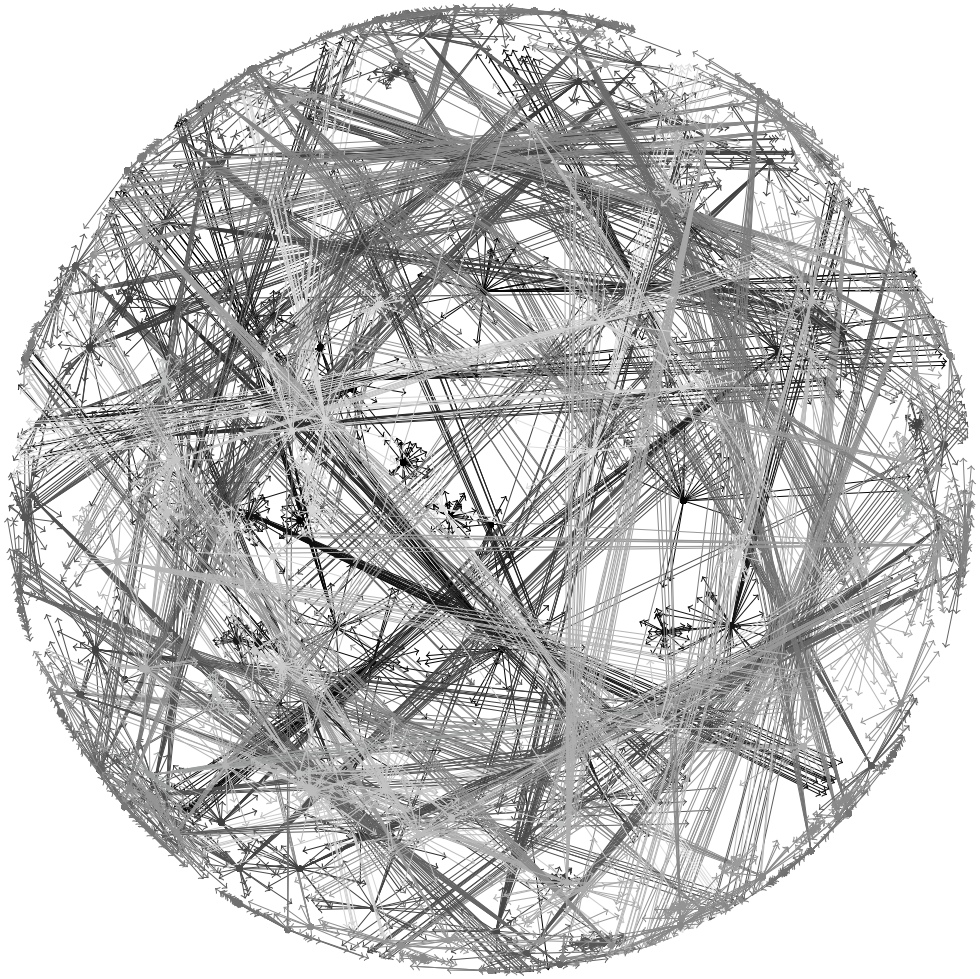

Figure: A sphere of 100000 neurons (on a tiny fraction showed).

- A simple algorithm was used to find polychronous groups:
  - Select an anchor excitatory neuron having two or more strong (within 5% of the strongest connections) connections to other excitatory neurons. This ensures that firing of the anchor neuron increases the probability that target neurons will fire in the appropriate time, resulting from axonal delays.
  - For the descendants of the anchor neuron, find any common postsynaptic targets that have strong connections and matching delays, that is to say if the descendants of the anchor neuron fire excited by the anchor, the resulting spikes will reach the common target synchronously (within 2ms interval). If no such common targets are found, the anchor neuron is discarded.
  - If there are such common targets, they are added to the group. The process is repeated, that is to say common postsynaptic targets with matching delays for all the current members of the group are found. The process finishes when there are no more such targets.

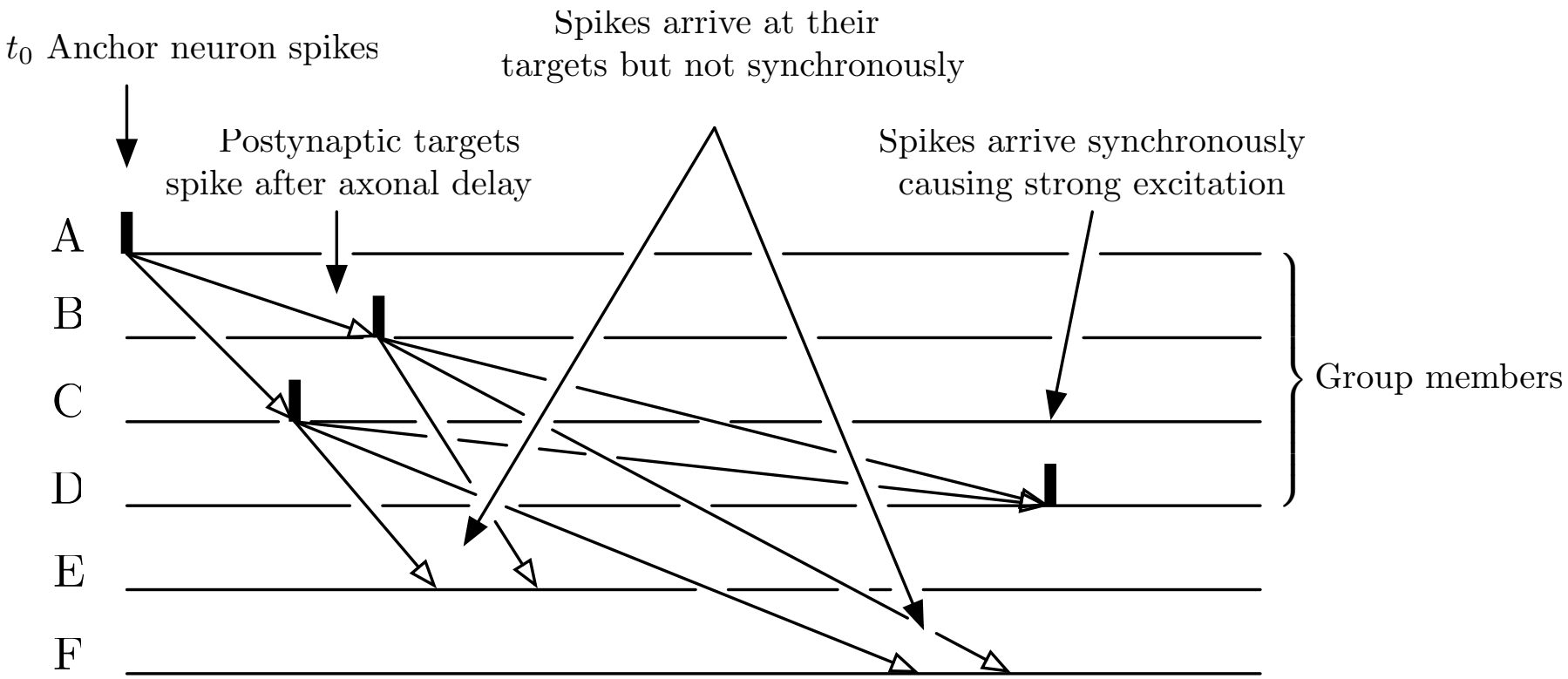

Figure: A simple algorithm of finding polychronous groups based on anchor neurons. In fact each neuron can be a member of many groups, while this simple algorithm only finds at most one group per anchor neuron.

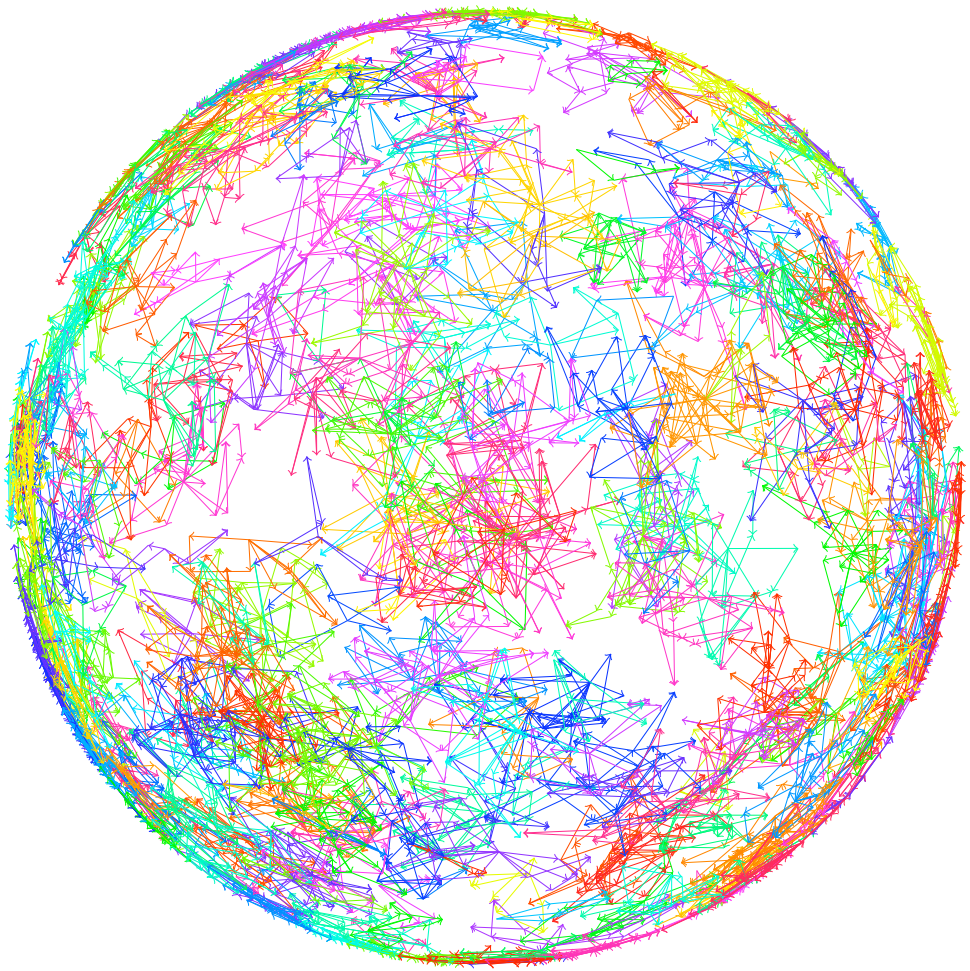

Figure: 2% and 10% of polychronous groups found in a sphere of 100000 neurons.

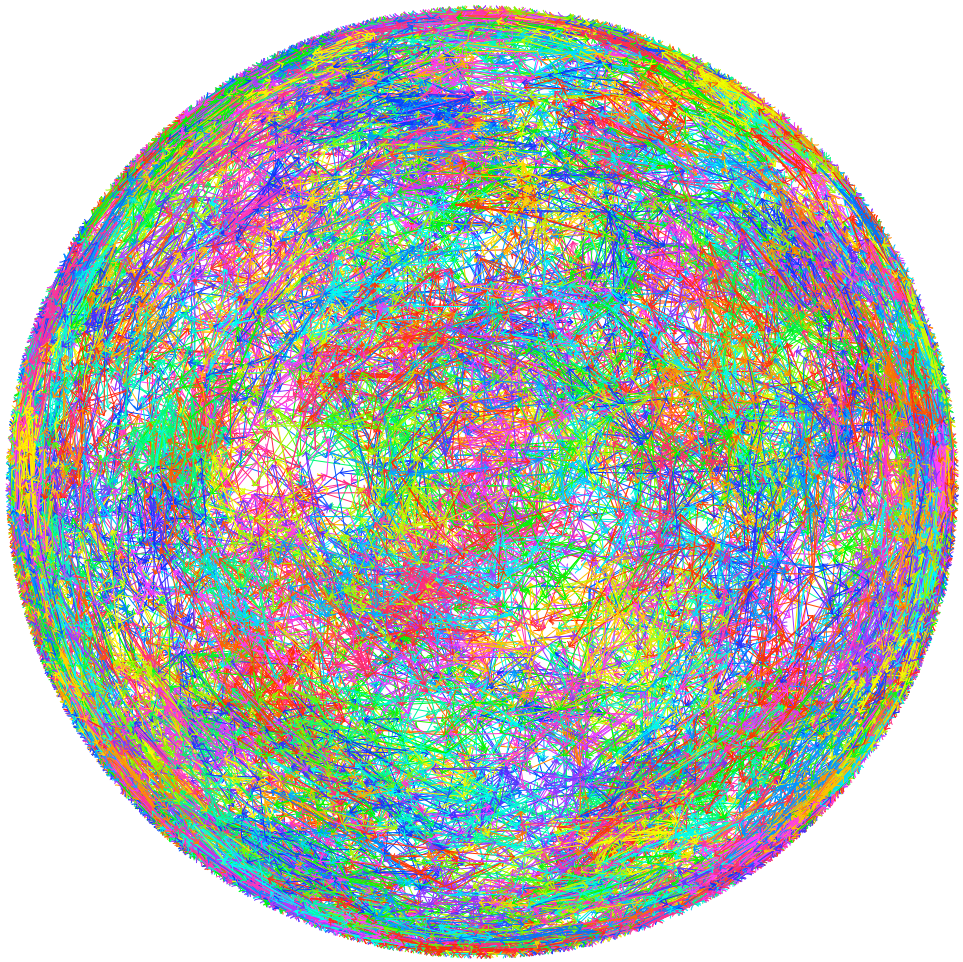

Figure: 2% and 10% of polychronous groups found in a sphere of 100000 neurons.

- In the initial period of the simulation (weights are random) there are no groups at all.
- Soon however, the algorithm is able to pick up growing number of groups. After about an hour of model time (the timing depends on the details of STDP which we will discuss next week) the number of groups levels of at a couple of thousands. Some of the groups persist for quite a while, others disappear.
- The careful study if the spike trains reveals group activation events (though some groups might actually never be activated).
- The algorithm uses anchor neurons to find groups, but one may search over say all triplets heaving common targets, In such case the number of groups can explode and easily exceed the number of neurons!

Recapitulation

- When the number of oscillators is large, mean field approximations can become useful.
- We've seen various properties of SNIC oscillators depending on their coupling mechanism
- The brain is a self wiring computer. The process of learning is related to tuning synaptic connections.
- Donald Hebb noticed that "neurons that fire together, wire together"
- The more adequate incarnation of the above principle is obtained via STDP
- STDP together with conduction delays results in spontaneous emergence of selective circuits (polychronous groups).