# Outline of the lecture

1. **Introduction to the Neuromorphic Engineering**
2. **Biological Background**
3. **Cellular Building Blocks: Neuron Models**
4. **Memory Formation: Learning and Plasticity**
   - 4.1 Introduction: Learning and Plasticity
   - 4.2 Hebbian Learning Models
   - 4.3 Memristive Hebbian Learning Models
5. **Artificial Neural Networks**

# 4.1 Introduction: Learning and Plasticity

**Learning objectives:**

The emulation of learning and memory processes is the essential goal of neuromorphic engineering. In contrast to our digital computers, learning and memory processes take place decentral between individual neurons. In particular, the variability in the strength of synaptic connections plays a key role in this process and is referred to as synaptic plasticity. In this chapter we will examine plasticity processes and the associated basic cellular mechanisms for learning and memory processes.

In this chapter I would like to answer the following question:

- What is synaptic plasticity?
- Why is synaptic plasticity so important?
- What is long-term potentiation (LTP) and and why is this so important for learning and memory?
- What is meant by persistent, input specificity, and associativity?
- What does the Hebbian learning rule say?
- What is spike timing dependent plasticity (STDP) and for what this is needed?
- What is the Song, Miller, Abbott model ?

# 4.1 Introduction: Learning and Plasticity

- The emulation of learning processes is one of the most important goals of neuromorphic engineering.
  - As we know, learning means the creation of knowledge. Furthermore, this knowledge should of course also stoared. This is memory.
- As we discussed in Chapter 2, the basis for learning and memory processes are the processes at the synapses:
  - Depending on the excitation and state of the pre and post neurons, the transmission properties at the synapses change.
  - This changes and state-dependency of synapses are called synaptic plasticity and this is what you need for the formation of memories and what occurs during learning.
- We had already seen exactly the last point when we talked about the perceptron:
  - We were able to determine that the small neural network can learn a required connectivity structure (the weight values) through a suitable learning rule.
  - For this we had derived the delta rule, which, however, must always contain information about the desired learning state. This type of learning is called supervised learning, i.e. learning with a teacher.
  - Of course, nature can do this without a teacher, i.e. unsupervised, so we will take a closer look at this in the chapter on networks.

# 4.1 Introduction: Learning and Plasticity

- What is important here in the chapter are the processes at the synapses,
  - i.e. exactly the development of learning rules that make it possible to enable a variety of different learning forms and memory formation in networks.
  - Learning itself is always a network process. However, the processes at the synapses form the basis for this: they determine the learning in the network and we want to understand these processes and then use the understanding to reproduce them in technical systems.
- Thus, we are already able to answer the question why synaptic plasticity is so important:

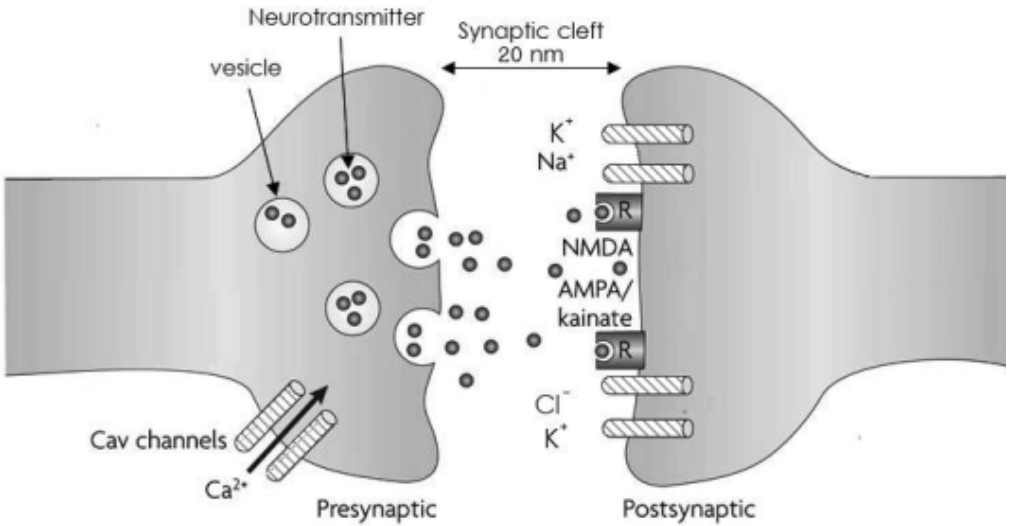
1. Synaptic plasticity shapes neural communication.
2. Synaptic plasticity is required during development such that stable and appropriate neural connections can be made.
3. Experience-dependent modifications in synaptic transmission are thought to occur during learning and underlie the storage of memories.

  - But, let's see what the points 1-3 are meaning….

# 4.1 Introduction: Learning and Plasticity

- First thing we need to remember is what happens in a synapse:
  - As discussed in chapter 2.1 most of the communication takes place chemically via chemical synapses, where 3 different regions can be distinguished at the synapse:

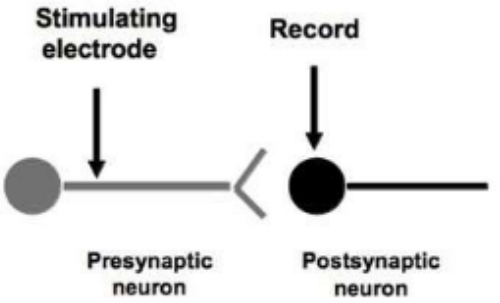
1. The pre-synaptic ending of the neuron which transmits information and contains a hundred synaptic vesicles with transmitters, which are called neurotransmitters.
2. The approximately 20-40 nm wide synaptic gap between the two neurons.
3. The cell membrane of the downstream neuron, the postsynaptic cell. There are also ion channels here, but they are different from those in the axon.




  - Thus, a conversion from action potentials into changes at the synapses leads to a experience dependent change in the brain connectivity and this again is named **synaptic plasticity**.

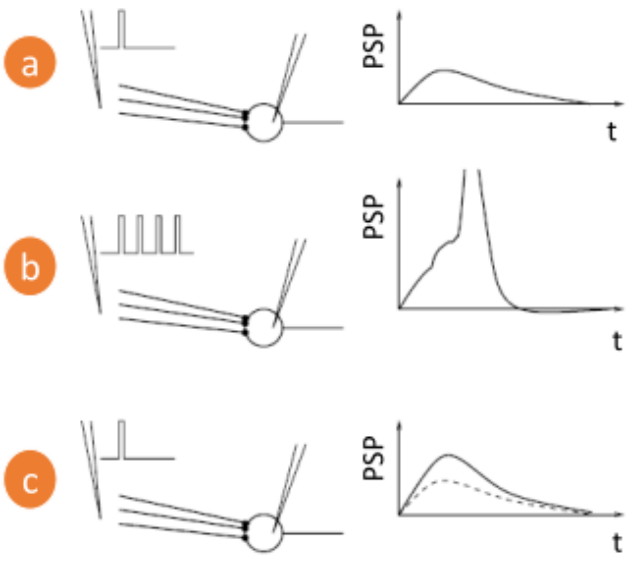
# 4.1 Introduction: Learning and Plasticity

- Synaptic plasticity can be measured by changes in the size of the post-synaptic potential induced by a single test stimulation, as sketched in the figure.




  - In the experiment the presynaptic neuron is stimulate while the signal is recorded from the postsynaptic neuron.
  - A stimulating electrode is used for both, an induce activity (spiking of the neuron) and to test the synaptic strength (weight).

- Now of course the question arises what you measure and what the signals you receive look like:
  - In the figures a stimulus is induced via the pre-neuron, while the post-synaptic potential is recorded (PSP).

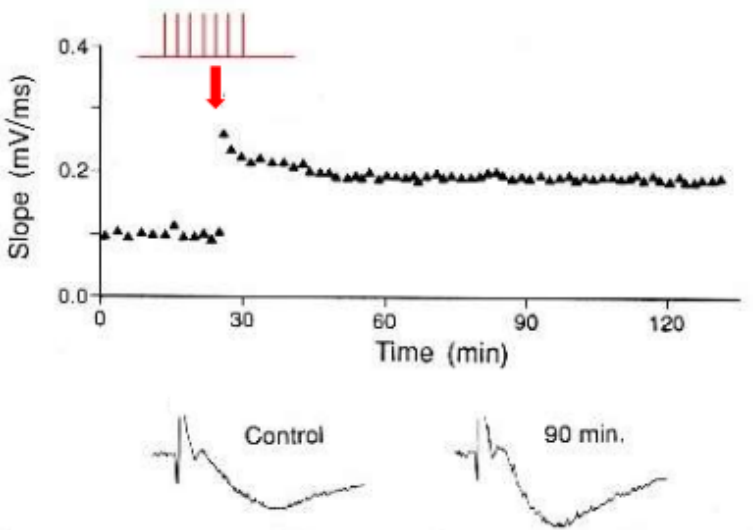



  a) A weak test pulse (sketched as single pulse) evokes the postsynaptic response: Pulse is not strong enough to trigger an action potential.
  b) A strong stimulation sequence (sketched as 4 pulses) triggers postsynaptic firing.
  c) The same test pulse as in (a) is applied some time later and evokes a larger postsynaptic response than the initial response (compare dashed line with solid line).

# 4.1 Introduction: Learning and Plasticity

- This is an interesting and for us new effect:
  - We observed an increased synaptic response in terms of the post-synaptic potential after an high frequency stimulation from the pre-synaptic neuron.
  - This effect in synaptic plasticity is called **long-term potentiation (LTP)** and is believed to be the important cellular condition for memory and leaning.
- In particular, a second aspect is important for LTP: Activity leaves the synapse potentiated for a long period of time (see figure):

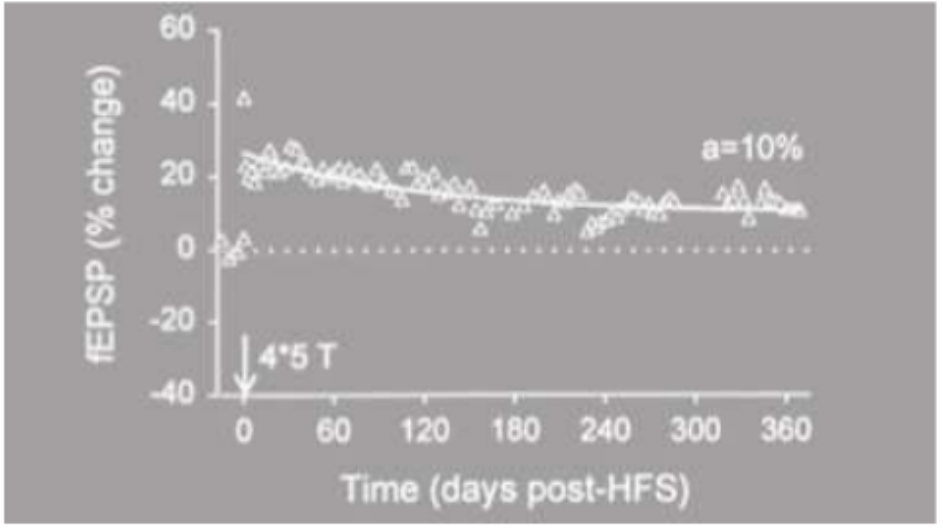
  - In the figure the response (slope [mV/ms]) of the synapse in terms of a test pulse is shown:
  - Here at 30 minutes a high frequency stimulation was applied. After that an increase in the response was observed, as discussed.
  - But, important this increase stays for a long period or biologist speak about the synapse is potentiated.
  - Thus, we might can say the synapse has learned and stored information. This is meant by the experience changes synaptic connection



# 4.1 Introduction: Learning and Plasticity

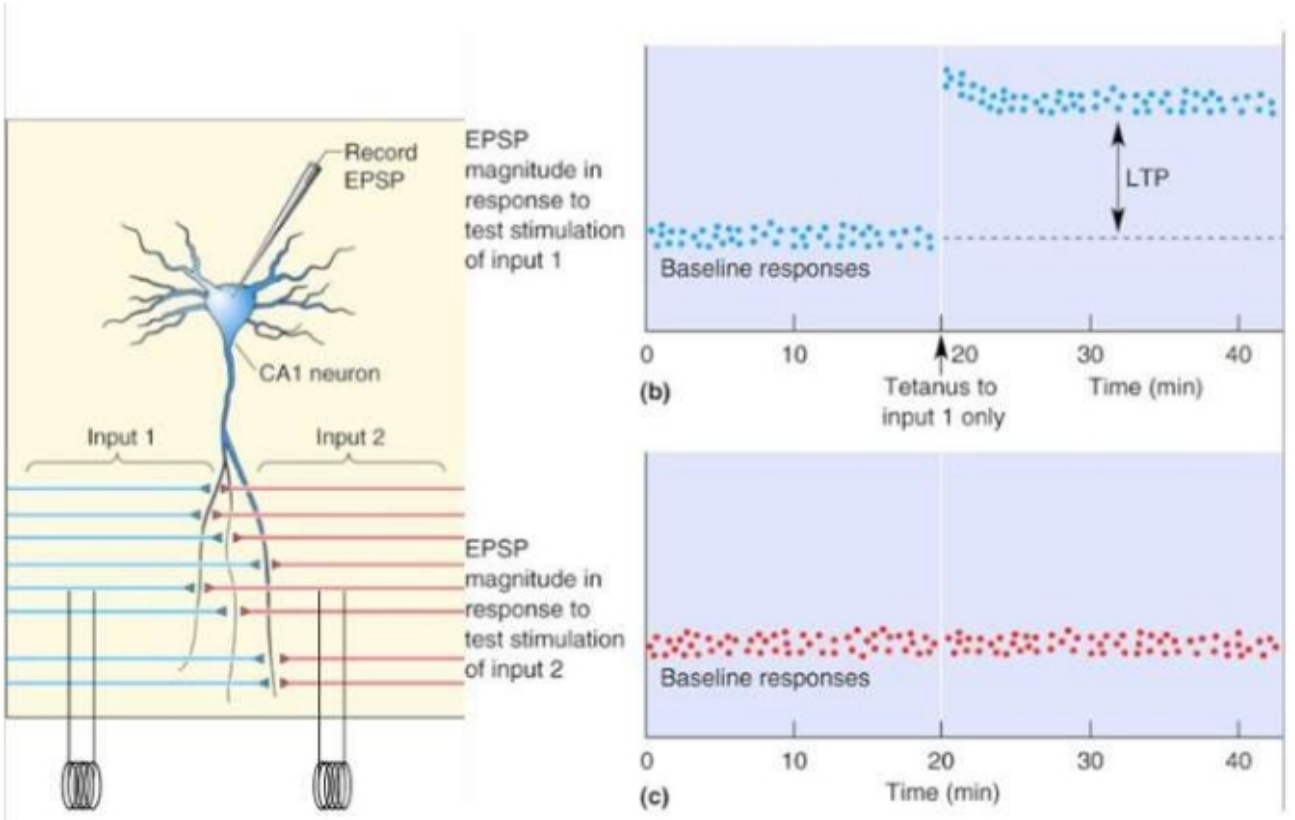
- Now it may not completely convince some people that long term potentiation offers the basis for information storage. But in fact, LTP meets the following important physiological criteria for memory formation:
  - Persistent: maintained for a long time after a brief period of synaptic activity.
  - Input specificity: induced only at appropriately stimulated synapses, and not neighbouring synapses.
  - Associativity: inputs activated together will strengthen.

- We should discuss this one a little more closely….
- Let us start with the term persistent and what exactly is meant by it:
  - In the figure you see a similar graphic as on the previous slide. The difference is the time axis. The y-axis shows the maximum PSP signal recorded after a test stimulus.
  - What you see is that the synaptic potentiation lasts for a long time. Here it's a year. This what is meant with persistant
  - Thus, LTP can persists for months in vivo



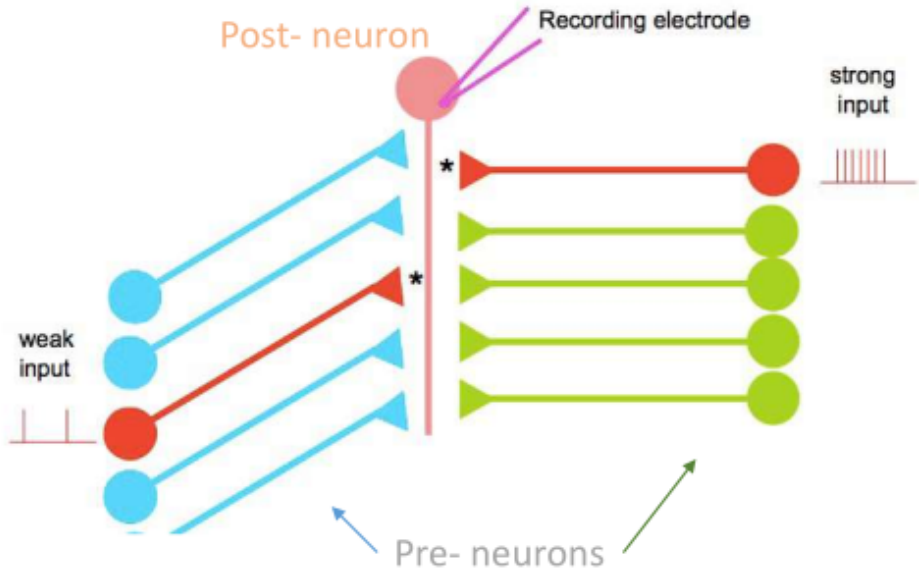
# 4.1 Introduction: Learning and Plasticity

- Now what is meant by the phrase: LTP is input specific.
- Let us look at the figure on the left:
  - Here several presynaptic neurons are sketched to a post synaptic neuron (connection in red and blue).
  - A strong stimulus is only applied to the blue connections.
  - With the help of a test pulse you can see that only LTP was injected into those synaptic connections that had previously seen the strong stimulus (see figures c and b).
  - Thus, only synapses onto a cell that have been highly active become strengthen, This is meant by input specificity



# 4.1 Introduction: Learning and Plasticity

- The third thing we said was that LTP is associative. This is one of the central properties for learning and we want to take a closer look at this:
  - Therefore we look at the figure which again shows a postsynaptic cell with connections to several presynaptic neurons.
  - The two red presynaptic neurons are stimulated at the same time and a synaptic transmission is established, which is symbolized by the star.
  - However, there is a difference: in one neuron a strong input signal is applied, while in the other the signal is relatively weak. Strong and weak again means that in one case you get LTP and in the other you don't.
  - But now one could observe that the synapse of the weak signal was also potentiated more strongly if a strong impulse was applied to the post-neuron via another synapse at the same time.




  - Hence, **LTP is associative**: Pairing a weak input with a strong input enhances later responses to a weak input

# 4.1 Introduction: Learning and Plasticity

- But why is associativity so important for learning?

  We will look at the following example which explains this, i.e. **Classical conditioning**

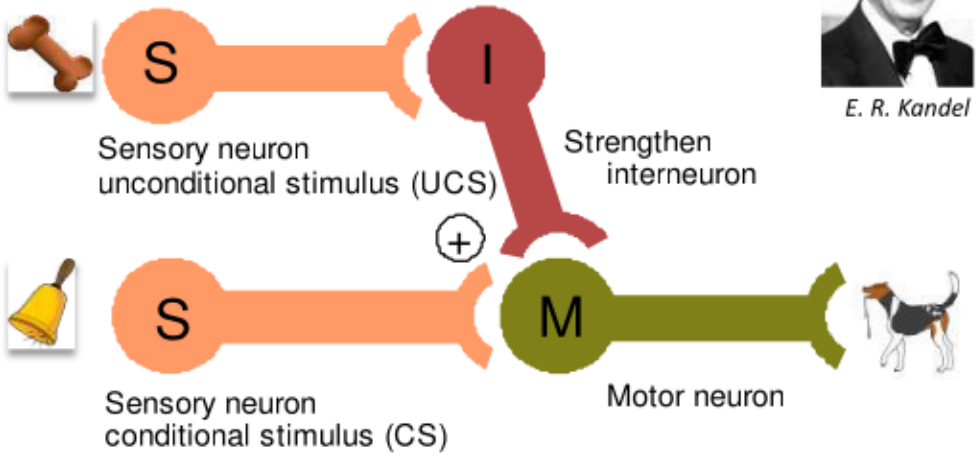
- It was first studied by Paplov more than a hundred years ago (1903). His measurement setup is shown in the picture.
- Pavlov (the man in the middle) fed his dog and always before, when he showed him how to eat, he measured how much saliva the dog produces.
- Pavlov rang a bell every time he did this.
- What Pavlov was able to show was that the saliva production of the dog is higher when he rings the bell, even if he does not feed him at all. So the dog expects to eat when he hears the bell.
- This behaviour is very similar to what we have just learned about LTP and associativity....

- Again, associativity is key for models of learning and memory –but is that really related to LTP?

# 4.1 Introduction: Learning and Plasticity

- Yes, this behavioural experiment can really be applied to cellular processes.
  - This was shown about 50 years later by Eric Kandel, who conducted experiments with marine snails Apalyisia and investigated such reflex-like learning processes.
  - Based on physiological measurements, Kandel had proposed the following model for associative learning.
  - The model consists of 4 neurons: two sensor neurons for the two stimuli bell and food, an inter-neuron to connect the sensor neurons and a motor neuron which regulates the salivary flow.

*E. R. Kandel*




- Now what does the model show: It shows that even two totally independent stimuli can be linked together if they occur simultaneously. This means that **we learn associatively and time plays an essential role**.
  - We connect things when they are given at the same time in a defined context:
  - Bell – food, but also we are not different than dogs: A child which learns that it should not touch a hot plate has to learn (most of the time they don't believe their parents before) to feel the pain temporally in relation to touching the plate. If a child feels a pain in his hand only one day later he will hardly blame the hot plate….

# 4.1 Introduction: Learning and Plasticity

- The importance of the temporal context of information for learning was barely also suspected by Hebb, who as a psychologist has put forward the following postulate, which today is referred to as Hebbian learning rule:

> "When an axon of cell *A is near enough to excite cell B or 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.*"
>
> *(Hebb, 1949)*

D. Hebb

  - Now often and somewhat more simply stated this sentence says:

    **"neurons that fire together wire together."**

  - Thus the temporal correlation of signals determines the strengthening of synaptic connections.

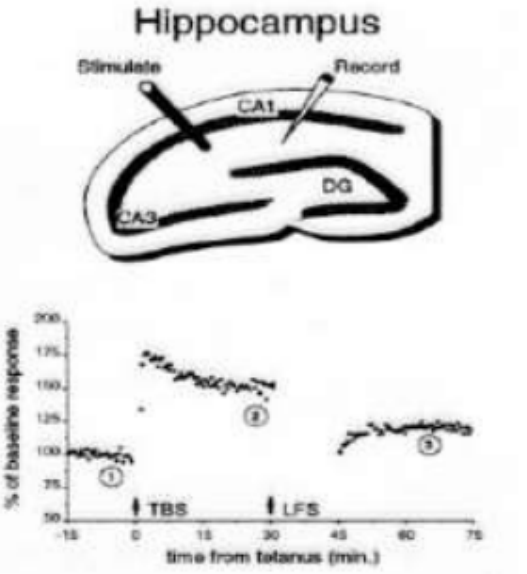
- But is this also true for the other direction? Is that also true for unlearning?
  - This is an important point and even if unlearning is perceived as rather unfavourable in our daily life, it is very important on a cellular level.
  - if one did not lose one's memory, the entire storage capacity of our brain would be used up at some point and, in order to strengthen certain synaptic connections, others must also be weakened so that we can achieve targeted connectivity of the neurons through learning.

# 4.1 Introduction: Learning and Plasticity

- so it makes sense to extend Hebb's learning rules by a depression, as was done by Stent in 1973:

> "Neural connections weaken when they are inactive at the same time that the postsynaptic neuron is active, or the will weaken if they are active, but consistently fail to cause the postsynaptic cell to fire an action potential"

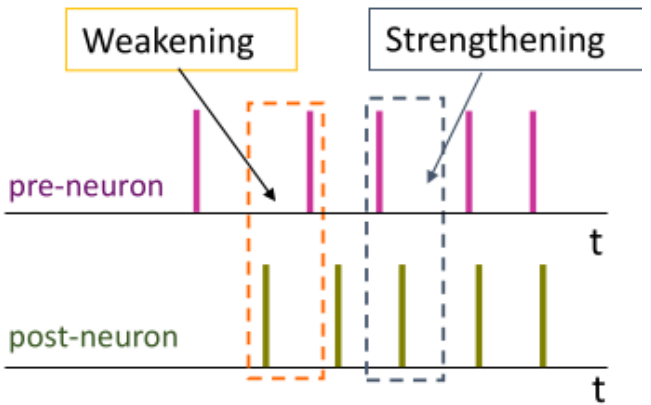
- Thus a counterpart to LTP can be defined, which is called LTD (Long-term depression). Here are a few important features of LTD:
  - LTD is a prolonged decrease in the response at a synapses occurring when axons have been activated at low spiking rates (hence the opposite to LTP).
  - First LTD (but also LTP) was detected in the 1970s by Bliss and Lomo in the Hippocampus, as shown in the figure.
  - this example shows that potentiating can also be reversed, i.e. depressed
  - Thus, LTD counter balances LTP
  - and might play a key role in **keeping memories separate and erasing old memories.**



# 4.1 Introduction: Learning and Plasticity

- Now there is the question of a detailed mechanism that changes the strength of a synaptic connection, i.e. a plasticity rule:
  - As we have seen, for a strong stimulus LTP is induced, while a weak stimulus leads to LTD.
  - But, all this is also dependent on time and not every weak stimulus necessarily leads to a weakening (depression). Here we think of the important property of associativity.
- All in all one of the puzzles in neurobiology and there are still a number of open questions here. But there is an important mechanism that can lead us towards a learning rule and this is called **spike timing dependent plasticity (STDP):**

- In the figure two spike patterns are shown: the upper one is from the pre-neuron and the lower one from the post-neuron.
- It is a sketch of the experiment of Bi and Poo: what the two neurobiologists could show was that the temporal order of the spikes has an effect on the change of the synapse:
  - if the post-neuron is active before the pre-neuron, there is a weakening.
  - if the pre-neuron is active before the post-neuron, there is a strengthening.



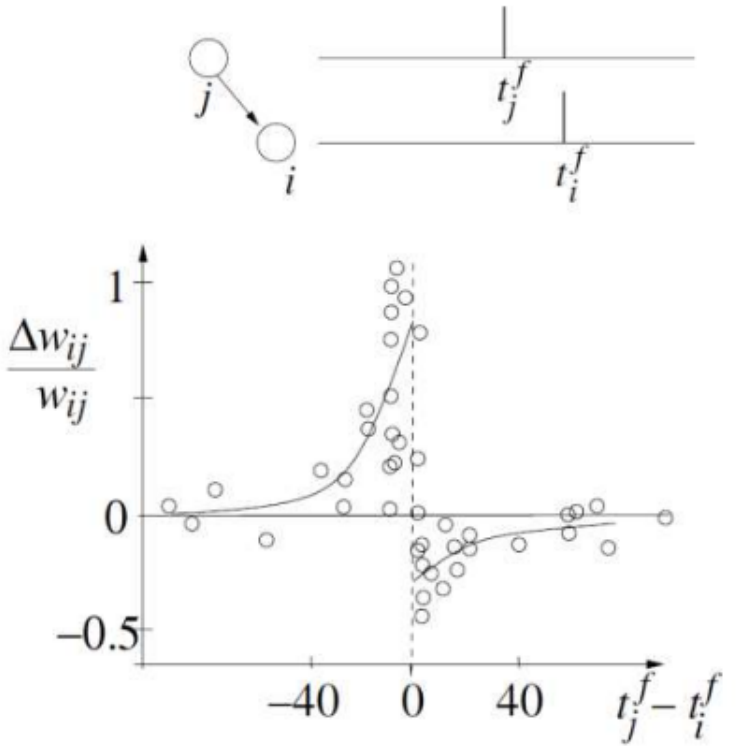

- This observation is especially relevant because **it allows causality**:
  - Information is transferred from the pre- to the post-neuron. Like with the example of the child who touches the hotplate: first he touches the plate and then he feels the pain. On the other hand, one would hardly think that the pain comes from the hotplate...

# 4.1 Introduction: Learning and Plasticity

- In the following we will take a more detailed look at the important STDP mechanism:
  - In the figure, experimental observations of changes in synaptic strength are plotted as a function of time between the spike time of the pre neuron (j) and post neuron (i).
  - You can see that the closer you get to zero the bigger the change is and because with further distances between the two pulses the change is exponentially towards zero.
  - This contains two main pieces of information:
    - firstly, plasticity has a time window of ± 40ms.
    - secondly, the temporal order of the signals describes the direction of change (strengthening or weakening).
- This experimental observation can be very elegantly described mathematically with the Song, Miller, Abbott model and directly integrated into our already discussed LIF neuron models:

$$C_m \frac{dV}{dt} = g_L(V_{rest} - V) + g_{ex}(E_{ex} - V) + g_{in}(E_{in} - V)$$

  - where $g_{ex}$ and $g_{in}$ are the excitatory and inhibitory weighted (plastic) synapse currents.

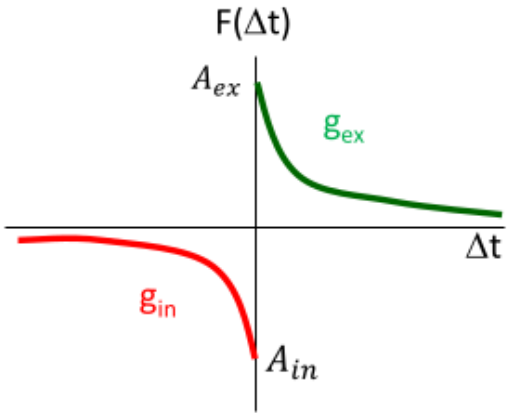



*Guo-qiang Bi and Mu-ming Poo, J. of Neuroscience, 1998.*

# 4.1 Introduction: Learning and Plasticity

- now we have a differential equation for the synaptic input current, which we can solve based on the experimental observations:
  - This can be done most easily for both sides of the STDP graph, i.e. for the depression ($g_{in}$) and potentiation ($g_{ex}$), as plotted in the figure.

$$\frac{dg_{ex}}{dt} = -\frac{1}{\tau_{ex}} \cdot g_{ex} \qquad \frac{dg_{in}}{dt} = -\frac{1}{\tau_{in}} \cdot g_{in}$$




  - Where $\tau_{ex}$ and $\tau_{in}$ are the time constant for the potentiation and depression branch, respectively.
  - Further w define the difference in the spike times of the two neurons as $\Delta t = t_{post} - t_{pre}$ and thus get:

$$g_{ex} = A_{ex} \exp\left(-\frac{\Delta t}{\tau_{ex}}\right) \quad \text{and} \quad g_{in} = A_{in} \exp\left(\frac{\Delta t}{\tau_{in}}\right)$$

  - which results in the following functional relationship:

$$F(\Delta t) = \begin{cases} A_{ex} \exp\left(-\frac{\Delta t}{\tau_{ex}}\right) & if \Delta t > 0 \\ A_{in} \exp\left(\frac{\Delta t}{\tau_{in}}\right) & if \Delta t < 0 \end{cases}$$

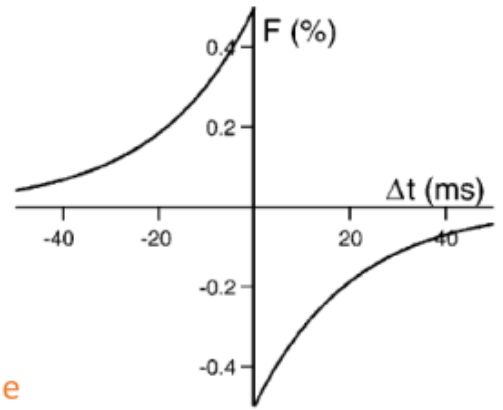
# 4.1 Introduction: Learning and Plasticity

- A further modification is still useful to obtain an additive learning rule, i.e. a rule that allows to adjust the synaptic current in a time-dependent manner and in dependence of the activities of the pre-neuron and post-neuron. So in-operando:
  - This is done iteratively as with the simple learning model we studied for the perceptron:

$$g_{syn}(t) = g_{ex}(t) + g_{in}(t) = g_{syn}\,(t-1) + \Delta g_{syn}$$

  - ➢ Now again with the distribution of the currents of the reinforcing and weakening synapses:

$$\Rightarrow \begin{cases} \Delta g_{syn} = g_{ex}(t) \cdot g_{ex-max} & \text{if } \Delta t > 0 \\ \Delta g_{syn} = g_{in}(t) \cdot g_{in-max} & \text{if } \Delta t < 0 \end{cases}$$




  - ➢ While $g_{ex-max}$ is the maximal potentiated current and $g_{in-max}$ is the minimal depressed current.
  - ➢ Thus, this allows an adaptive updating of the synaptic input current in respect to the spiking order and this model can be particularly realized technically well in hardware with the sub-threshold MOSFET circuits we discussed in chapter 3.5.

# 4.2 Hebbian Learning Models

**Learning objectives:**

Our brain has the incredible ability to make predictions from incomplete and noisy information. In almost every minute of the day we make decisions even if most of us are not so conscious. This is called behaviour and our brain uses different time scales to provide the relevant information at the exact moment it is needed. This is the core task of our memory which ranges from an ultra short term memory to a long term memory. The task of the learning process is to create a condensed representation of information from the incoming information and events of the past, which allows a correct classification of information. Now the exciting question is how this process can be described mathematically. This is exactly what we want to look at in this chapter. A good access to it is given by Hebb's learning rule, since it allows us to deal with plasticity and learning.

In this chapter I would like to answer the following question:

- How to convert the Hebbian learning rule into a mathematical model?
- What is post- and pre-synaptic gating
- What is the differences betwenn the BCM, Oja, and covariance rule?

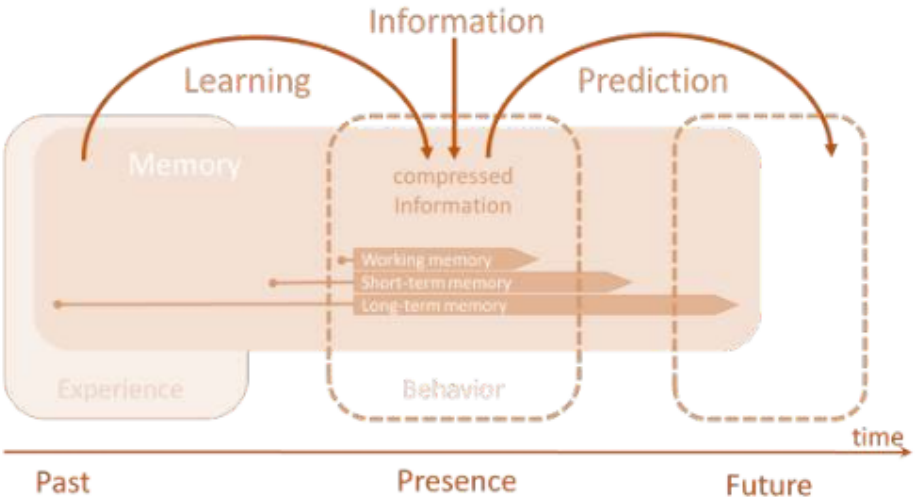
# 4.2 Hebbian Learning Models

- In the last chapter we have seen the fundamental cellular mechanisms that form the basis for learning and memory processes.
  - Here we could learn that LTP and LTD are the important foundations, because these mechanisms generate a long lasting change in synaptic connective strength.
  - A particularly important aspect for learning mechanisms was the property of associativity, which is closely related to the temporal correlation of neuronal activity.
  - We have discussed STDP as a simple learning mechanism that leads to LTD or LTP depending on the temporal order of the activity of the post and pre neuron, a fundamental plasticity mechanism.

- Such plasticity mechanisms are the content of this chapter.
  - We want to see how local mechanisms of cellular forms of learning can be reconstructed in such a way that global, network dependent learning and memory is obtained.
  - But first of all we want to take a look at memory formation in a more general way and make clear to ourselves what actually happens in our brain during memory formation and what are the necessary requirements for this?
  - Thus, we should first realize what the tasks of learning mechanisms are….

# 4.2 Hebbian Learning Models

- Learning processes and memory formation in living organisms make use of various time-dependent mechanisms ranging from a few milliseconds to the duration of a complete life.
  - These time-scale dynamics enable cognitive information processing and form the basis of our behaviour and is illustrated in the figure.
  - Time-dependent learning processes are used to form compressed memory content, which enables the information to be transferred into a time-relevant context.
  - Through the data compression and data selection that is carried out in this way, only the information that is relevant in the future is stored.




  - This allows living organisms to react to incomplete and noisy input information, to make predictions and plan future actions.
- **Memory has a bridging function and is formed by learning.**
  - Information is only available for a (short) specific period of time. Learning mechanisms create a compressed efficient image from this and arrange it in different memories, which keep information in memory for certain periods of time.
  - This allows predictions to be made about the future, which determines our behaviour.

# 4.2 Hebbian Learning Models

- The Hebbian learning rule offers a good starting point for this, as it allows a connection of cellular mechanisms of synaptic plasticity with the aforementioned network functionalities:
  - However, Hebb was not a mathematician and his rule was a description or better at the time a conjecture.
  - So the first step we have to do now is to c.
- For this we want to start with the first important characteristic - **locality**:
  - This means change of the synaptic weight $w_{ij}$ can only depend on local variables, i.e., on information that is available at the site of the synapse.
  - Therefore, the rate of change of the synaptic coupling strength can be formulated as:

$$\frac{d\omega_{ij}}{dt} = F(\omega_{ij}, A_i, A_j)$$

  - Where the pre and post-synaptic activity $A_i$ or $A_j$ is voltage dependent: $A_{i(j)} = g\left(u_{i(j)}\right)$
- Further important mechanism is ***Cooperativity:***
  - This means that the pre- and post-synaptic neurons have to be active *simultaneously for a synaptic weight* change to occur. Hence this what Hebb postulated with the sentence: "*neurons that fire together wire together*"
  - In the simplest case the function $F(\omega_{ij}, A_i, A_j)$ can be selected as:

$$F\left(\omega_{ij}, A_i, A_j\right) = \alpha\, A_i \cdot A_j$$

    - The coefficient $\alpha$ is called learning rate and is usually positive: $\alpha > 0$
    - A learning rule with $\alpha < 0$ is usually called anti-Hebbian

# 4.2 Hebbian Learning Models

- So far so good. We have a simple learning rule, whose structure is very similar to the delta rule we learned at the perceptron.
  - Well this is because the delta rule is a Hebbian learning learning rule.
  - If you don't believe it, you should convince yourself and have a look at the derivation of the delta rule.
- An extension that makes sense is to ensure that the learning rule reaches a saturation value. A maximum learning state, so to speak.
  - Thus, for saturation F should depend on $\omega_{ij}$ which avoid unlimited weight growth: $\alpha(\omega_{ij}) = \gamma \cdot (1 - \omega_{ij})$
  - That would solves the problem: for $\omega_{ij} \to 1$ goes the learning rate $\alpha \to 0$. And of course,$\omega_{ij}$ should be normalized and only change in the interval $\omega_{ij}, min = 0$ to $\omega_{ij}, max = 1$ .
- Synaptic depression was not in Hebb's original proposal, but we have seen that is important to have and it can also be modelled in this way:

$$\alpha_{dep}(\omega_{ij}) = -\gamma_{dep} \cdot \omega_{ij}$$

  - $\gamma_{dep}$ is a (small) positive constant that describes the rate by which $\omega_{ij}$ decays back to zero in the absence of a stimulation.

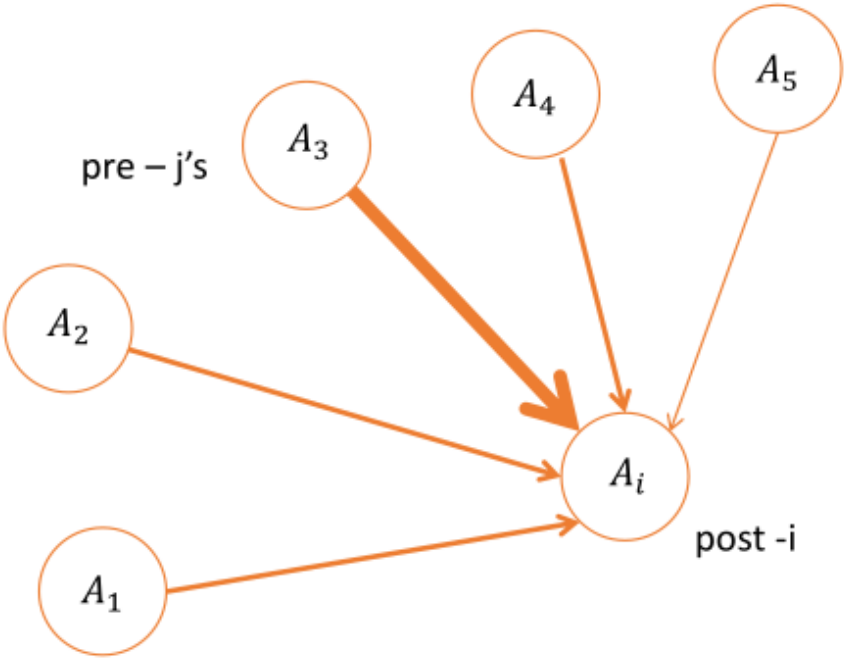
# 4.2 Hebbian Learning Models

- All together one gets the following equation for Hebbian learning:

$$\frac{d\omega_{ij}}{dt} = \gamma \cdot (1 - \omega_{ij}) \cdot A_i A_j - \gamma_{dep} \cdot \omega_{ij}$$

- The essential step towards the emulation of learning and memory is to transfer these local learning rules to a multi-dimensional network level:

  - Here **competition** is one of the most important mechanisms that can and should be used here a limitation of common synaptic resources.
  - As sketched in the figure synaptic weights can only grow at the expense of others (the thickness of the line should indicate the strength of the synaptic connections).
  - Competition can be implemented in the model by normalizing the sum of all weights converging onto the same postsynaptic neuron

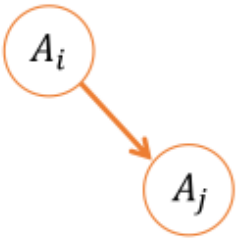


# 4.2 Hebbian Learning Models

- Let's have a look at some examples to understand the Hebbian learning model in more detail:

**Example 1:** Hebb rules as a function of pre- and postsynaptic activity

1. Post-synaptic gating: weight change occurs only if the postsynaptic neuron is active, $A_i > 0$:




$$\frac{d\omega_{ij}}{dt} = \alpha \cdot A_i \left[ A_j - A_{ref}(\omega_{ij}) \right]$$

- $\alpha$ is a positive constant and $A_{ref}$ is some reference activity, which is weight dependent.
- This results in the fact that a firing of the postsynaptic neuron leads to LTP, which is called for that case homosynaptic LTP.
- Furthermore, at inactive synapses LTD is induced: which is called for that case heterosynaptic LTD.

1. Pre-synaptic gating: change of synaptic weights can only occur for $A_j > 0$:

$$\frac{d\omega_{ij}}{dt} = \alpha \cdot \left[ A_i - A_{ref} \right] A_j$$

- For $\alpha > 0$, the synapse is strengthened if the postsynaptic cell is highly active ( $A_i > A_{ref}$) and otherwise it is weakened.
- For $\alpha < 0$, the correlation term has a negative sign: anti-Hebbian plasticity

# 4.2 Hebbian Learning Models

**Example 2:** the covariance rule (Sejnowski and Tesauro , 1989):

$$\frac{d\omega_{ij}}{dt} = \alpha \cdot [\, A_i - \langle A_i \rangle] \cdot [\, A_j - \langle A_j \rangle]$$

- The rates $A_i$ and $A_j$ fluctuate around mean values $\langle \cdot \rangle$ that are taken as running averages over the recent firing history.
- For $\alpha < 0$, the correlation term has a negative sign: anti-Hebbian plasticity

**Example 3:** the Oja rule (Oja, 1982):

$$\frac{d\omega_{ij}}{dt} = \alpha \cdot \left( A_i A_j - \omega_{ij} {A_i}^2 \right)$$

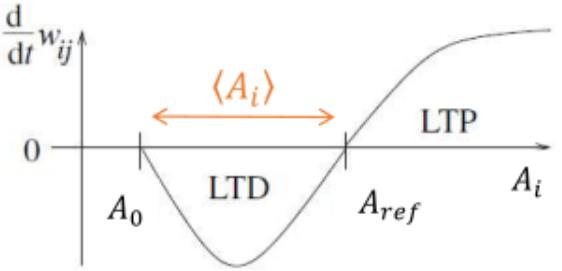
- Converges asymptotically to synaptic weights that are normalized to $\sum_j {\omega_{ij}}^2 = 1$
- The normalization implies competition between the synapses.

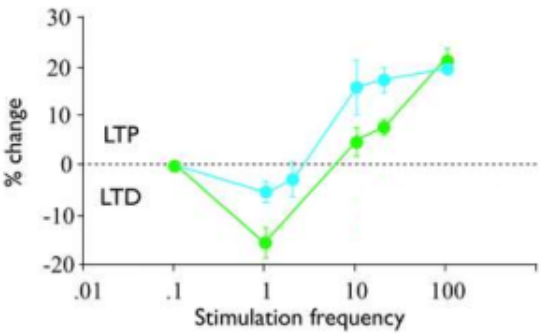
# 4.2 Hebbian Learning Models

**Example 4:** the Bienenstock–Cooper–Munroe rule (Bienenstock–Cooper–Munroe rule , 1982):

$$\frac{d\omega_{ij}}{dt} = \eta\phi\left[A_i - A_{ref}\right] - \gamma_{dep} \cdot \omega_{ij}$$

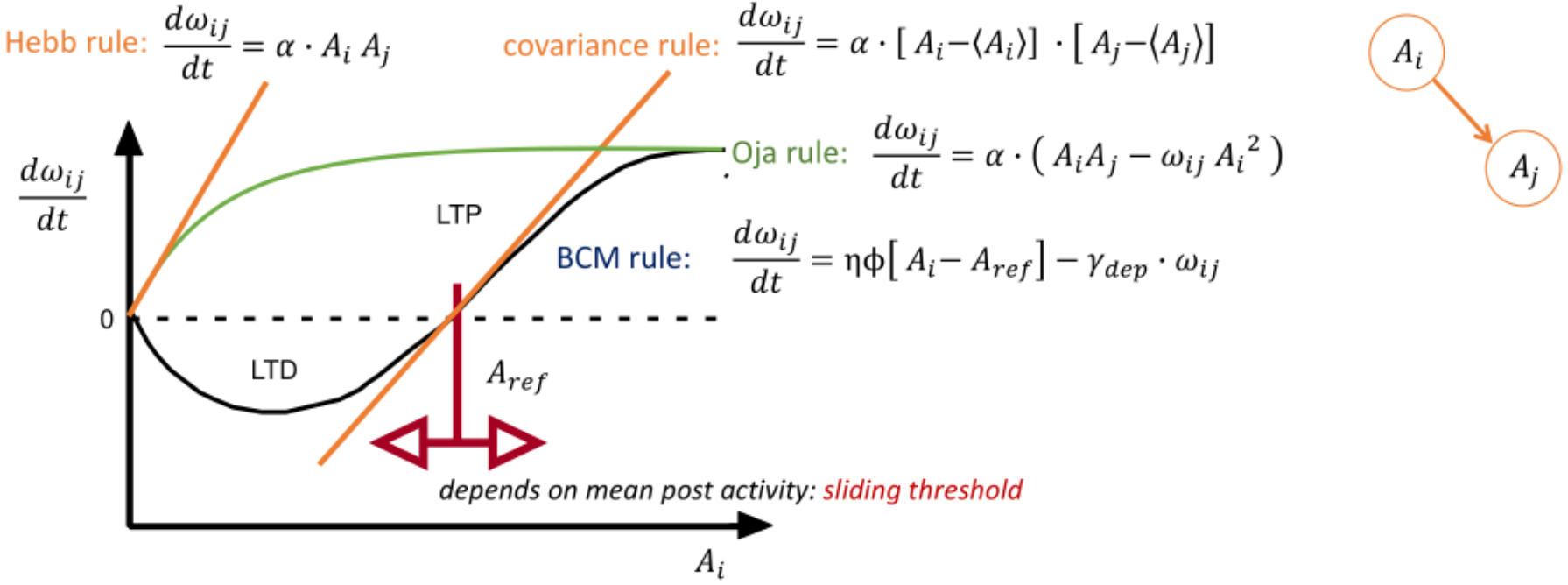
- This Hebbian learning rule is a generalization of the presynaptic gating rule
- It contains a nonlinear function $\phi$ and a reference rate $A_{ref}$
- If the reference rate $A_{ref}$ is replaced by the average of the output rate $\langle A_i \rangle$, it builds a sliding threshold for the induction of LTP (see figure).
- Furthermore using that learning rule a **competition** between synapses appears
  - Strengthening some synapses results in threshold increasing
  - This means that it is harder for others to be strengthened
- In 1996 Kirkword and colleagues were also able to experimentally verify the behaviour of the shifting threshold value predicted by the BCM model, as shown in the adjacent graph.



# 4.2 Hebbian Learning Models

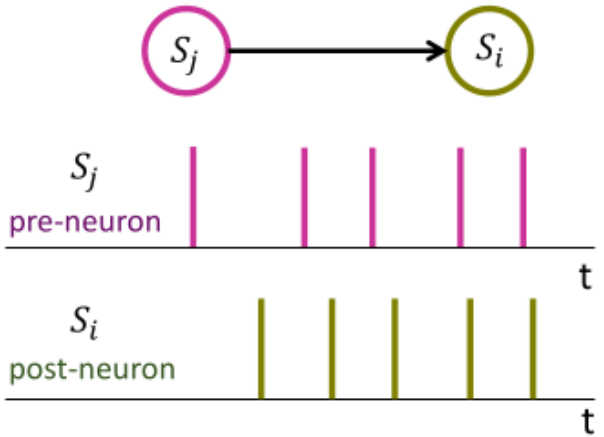
- Finally, all the examples discussed are summarised again in the figure:
  - It shows the synaptic weight change as function of the post synaptic activity



# 4.2 Hebbian Learning Models

- Up to now we have dealt with spike rate models, where the activity of the neurons was important but not the temporal order of the activity patterns. But we had seen in the last chapter that the temporal order of neuronal activity is an important mechanism of synaptic plasticity and define the STDP rule:
  - STDP creates causality in networks and thus allows temporal orders of events. An important property of our memory.
  - STDP can be generally understood as a special asymmetrical form of the Hebbian learning rule.
- Let us take the following approach to STDP to show this:
  - Therefore we start with the definition of two arbitrary spike trains for the pre and post neuron:

$$S_j = \sum_f \delta\left(t - t_j^{(f)}\right) \quad \text{and} \quad S_i = \sum_f \delta\left(t - t_i^{(f)}\right)$$




  - The Hebbian learning rule:

$$\frac{d\omega_{ij}}{dt} = S_j \cdot F_i(\Delta t) + S_i\, F_j(\Delta t)$$

  - with $F_i(\Delta t)$ and $F_j(\Delta t)$ as defined in the previous chapter:

$$F(\Delta t) = \begin{cases} F_j(\Delta t) = A_{ex} \exp\left(-\dfrac{\Delta t}{\tau_{ex}}\right) & if\, \Delta t > 0 \\ F_i(\Delta t) = A_{in} \exp\left(\dfrac{\Delta t}{\tau_{in}}\right) & if\, \Delta t < 0 \end{cases}$$

$$\Delta t = t_j - t_i$$

# 4.2 Hebbian Learning Models

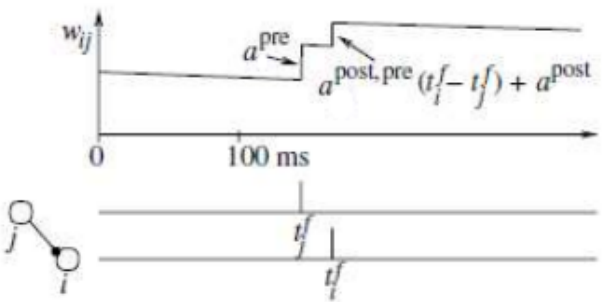
- An important extension of the simple model, which we already learned about in the previous chapter, is given by the model of Kistler and van Hemmen (2000):
  - Within this model an arbitrary learning window is defined:

$$\frac{d\omega_{ij}}{dt} = S_j \cdot \left[a_{pre} + \int_0^\infty \alpha_{ex}(\omega_{ij}) F_i(s) S_i(t-s) ds\right] + S_i \cdot \left[a_{post} + \int_0^\infty \alpha_{in}(\omega_{ij}) F_j(s) S_j(t-s) ds\right]$$

  - Where $\alpha_{ex}$ and $\alpha_{in}$ describes the dependence of the update on the current weight of the synapse. Usually $\alpha_{ex}$ is positive and $\alpha_{in}$ is negative. $a_{pre}$ and $a_{post}$ are non-Hebbian contributions.
- For the better understanding of the model let's discuss the following two cases:
  - The effect of pre-synaptic spikes:

$$\alpha_{ex} F_i(s) = a_{pre-post}(s) \Rightarrow \frac{d\omega_{ij}}{dt} = S_j \cdot \left[a_{pre} + \int_0^\infty a_{pre-post}(s) S_i(t-s) ds\right]$$

    - each spike that arrives at the presynaptic terminal can trigger a change in the synaptic efficacy (effect of $a_{pre}$) even without additional postsynaptic action potentials: Non Hebbian
    - Additional $a_{pre-post}$ gives the weight change if a postsynaptic action potential is followed by presynaptic spike arrival with delay s (see figure).



# 4.2 Hebbian Learning Models

- The effect of post-synaptic spikes:

$$\frac{d\omega_{ij}}{dt} = a_0 + S_j \cdot \left[ a_{pre} + \int_0^\infty a_{pre-post}(s) S_i(t-s) ds \right] + S_i \cdot \left[ a_{post} + \int_0^\infty a_{post-pre}(s) S_j(t-s) ds \right]$$

- Amplitude of the weight change consists of at least two contributions:
  1. a non-Hebbian term $a_{post}$
  2. a correlation term $a_{post-pre}$

- All parameters depend upon the actual value of the synaptic efficacy:
  - it becomes increasingly difficult to strengthen a synapse that has already been potentiated and vice versa to weaken a depressed synapse.