脑启发人工智能导论
Introduction to Brain-Inspired Artificial Intelligence

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Celebrating Lotfi Zadeh

World-renowned Azerbaijani-American computer scientist, electrical engineer, and professor, Lotfi Zadeh. On this day in 1964, Zadeh submitted "Fuzzy Sets," a groundbreaking paper that introduced the world to his innovative mathematical framework called "fuzzy logic." The "Fuzzy Sets" paper has since been cited by scholars nearly 100,000 times.

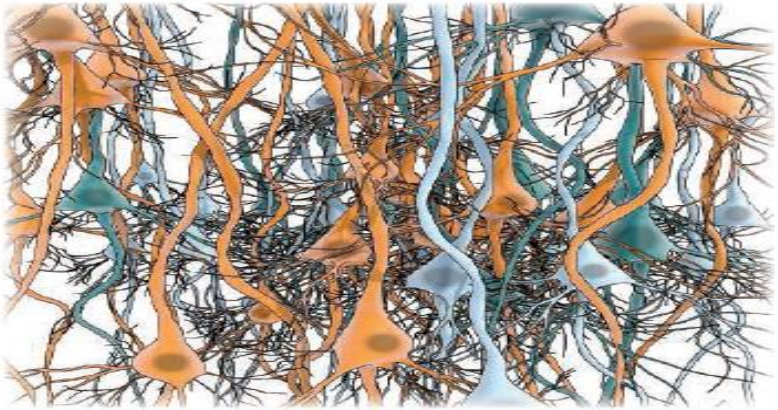
So here's to you, Lotfi Zadeh! There's nothing fuzzy about your huge impact on the scientific world.

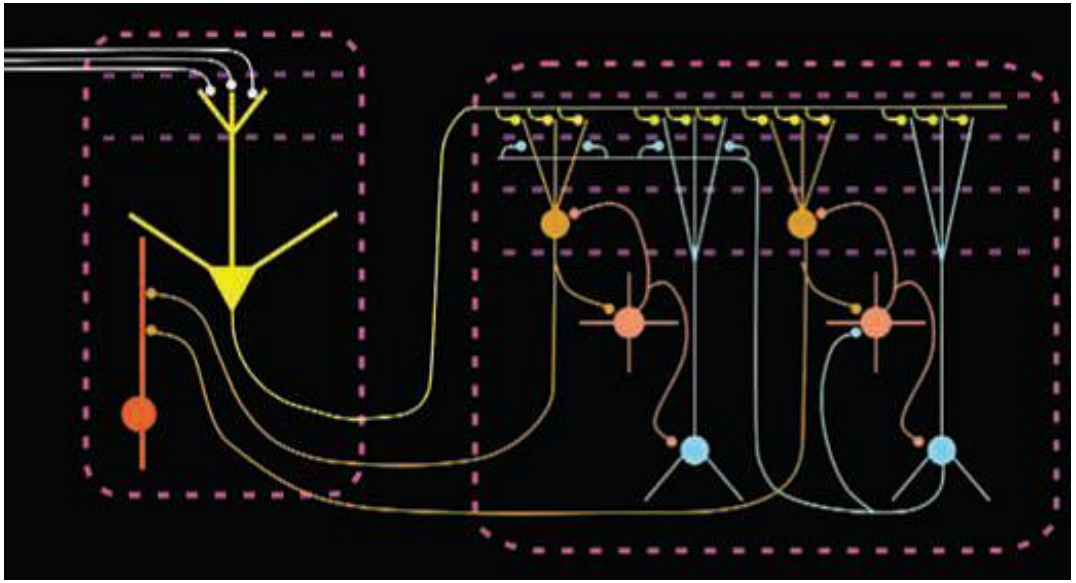
可塑性与学习规则: 神经科学机制和启发

Plasticity and Learning Rules

How Do Neurons Wire Together?

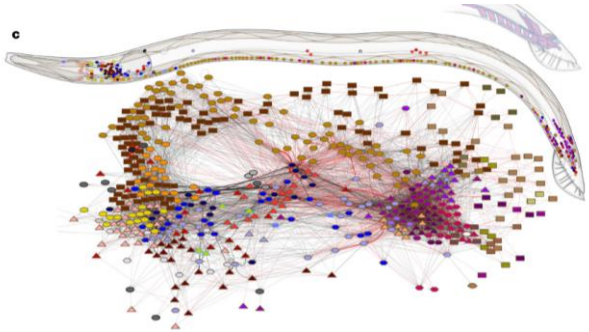
- Nervous systems face two challenges: to be plastic and able to change, adapt, and learn, while at the same time functioning reliably to ensure an animal's survival in an ever-changing environment.

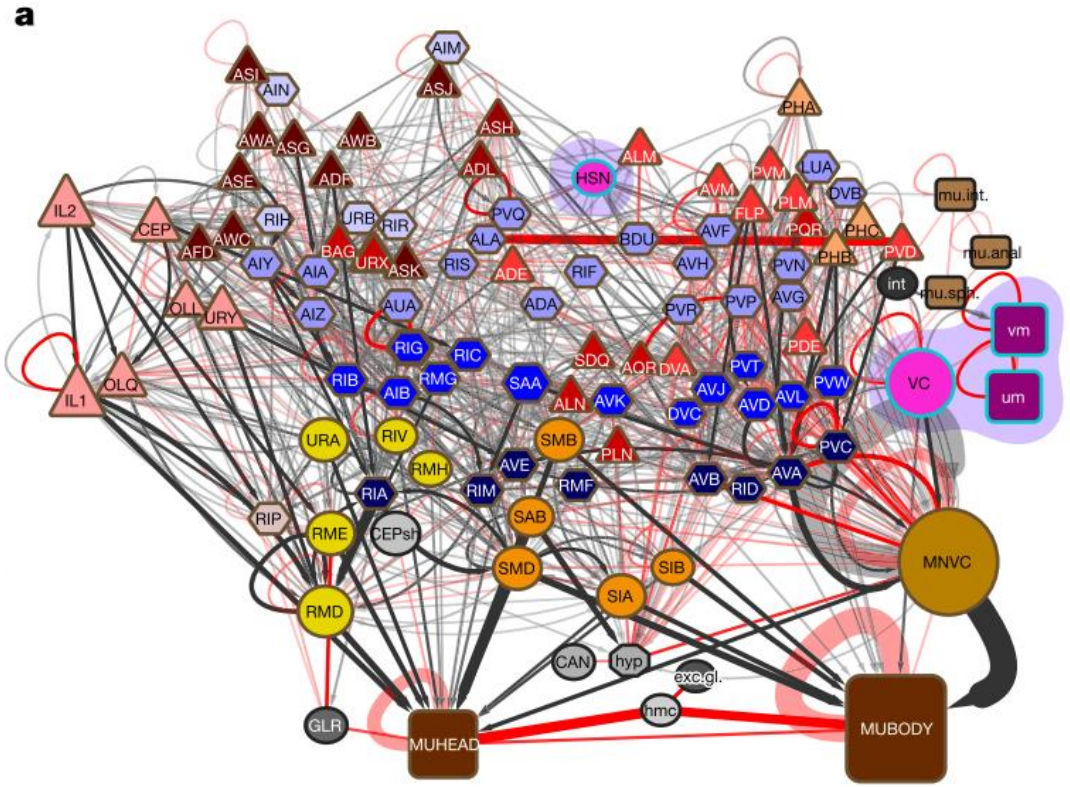




How Do Neurons Communicate Each Other?

- 线虫神经系统为例：300-400个神经元, ~5000个化学连接（化学突触），~1500个间隙连接（电突触）



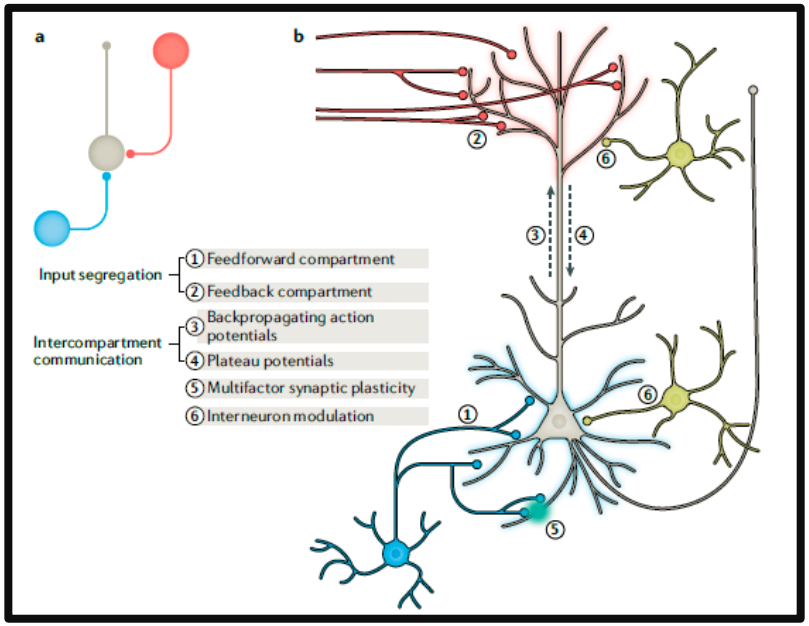


感觉神经元（三角形）->通过中间神经元(六角形)->运动神经元(彩色圈)->肌肉(方形)

How Do Neurons Wire Together?

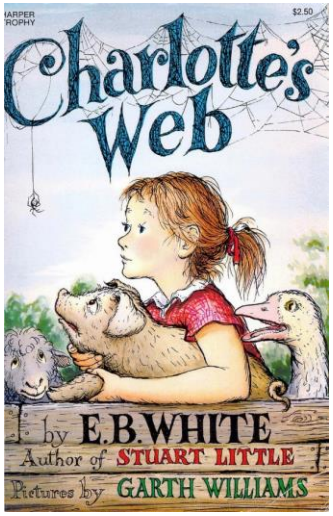
- Brains rely on multiple plasticity and homeostasis mechanisms that act on both synaptic and cell-intrinsic parameters.

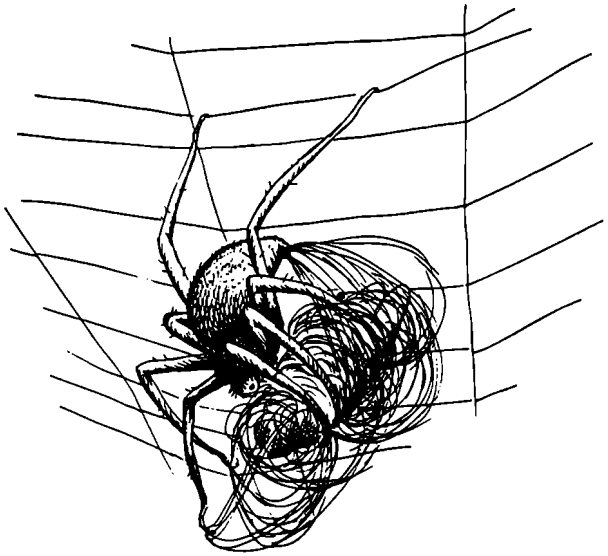
- An important finding in the area of neuronal plasticity and homeostasis is that electrophysiologically relevant parameters of neurons — for example, the magnitudes of different ionic conductances in a neuron's membrane — can vary widely between different neurons of the same type.

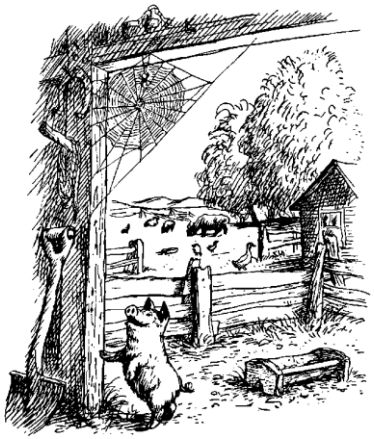


How Do Neurons Know What To Do? And When?

What tells a neuron whether and how to adjust its properties in order to maintain proper function?







A jumping spider is a perfect predator!
大脑直径<1mm

How Do Neurons Know What/When To Do?

神经网络的调控包括：突触可塑性和神经元本身行为的调控

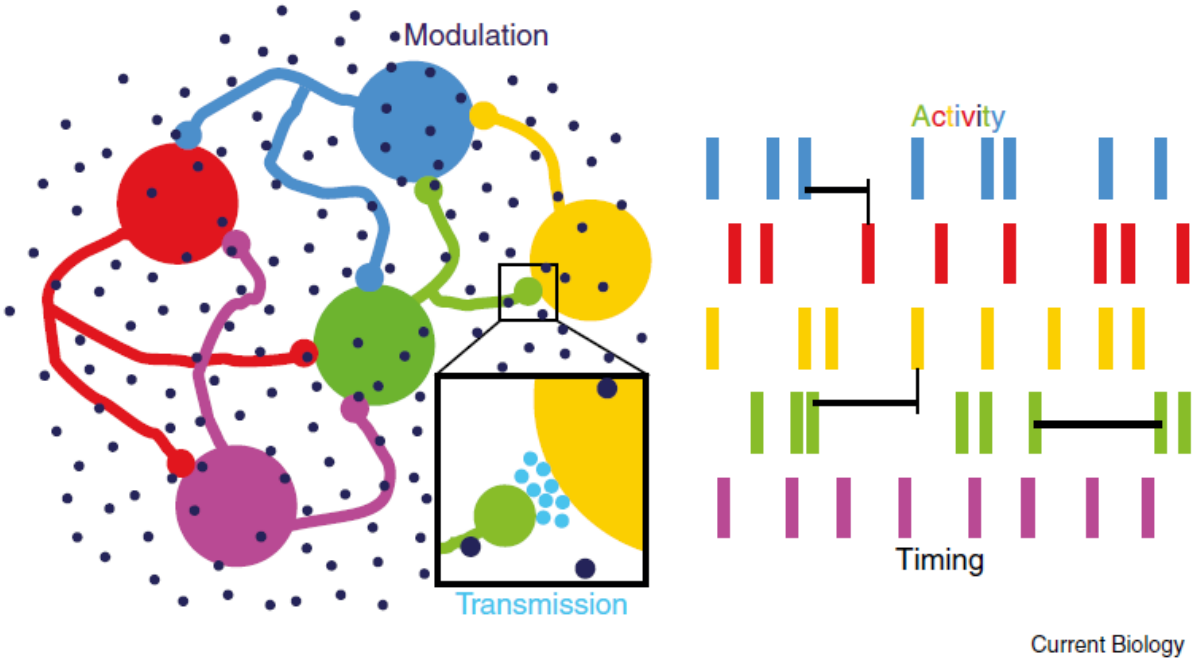


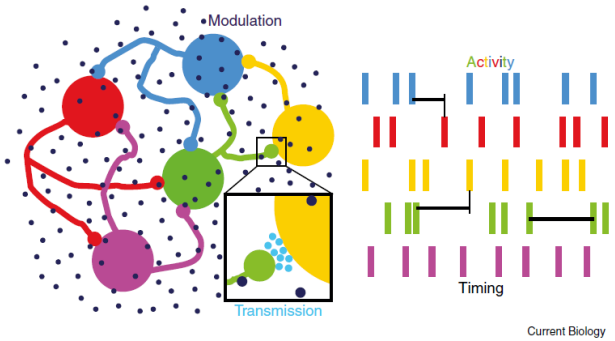
Figure 1 illustrates some of the characteristics of neuronal circuits that have been implicated as potentially involved in sensing whether a network functions properly, and in adjusting cellular and/or synaptic parameters if it doesn't.

Prinz, Astrid A. "Neuronal plasticity: how do neurons know what to do?." Current Biology 24.21 (2014): R1044-R1046.

How Do Neurons Know What/When To Do?

神经网络的调控包括：突触可塑性和神经元本身行为的调控

- Potential triggers and regulators of cellular and synaptic plasticity. Neuronal network characteristics potentially involved in **triggering and regulating cellular and synaptic plasticity** include:



① The overall level of electrical activity,

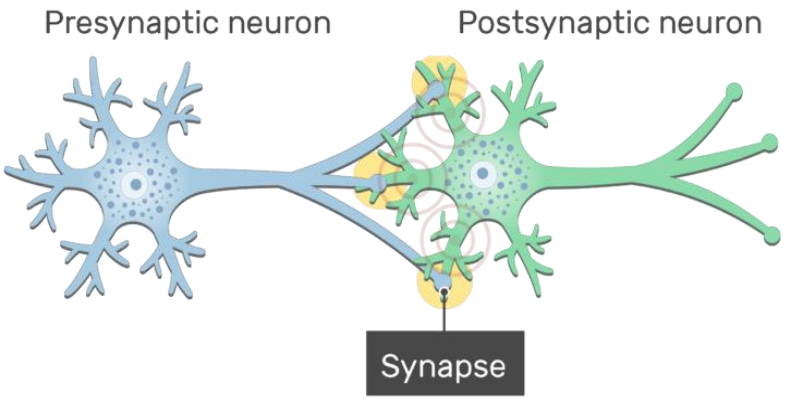
② Its precise timing within and between neurons,

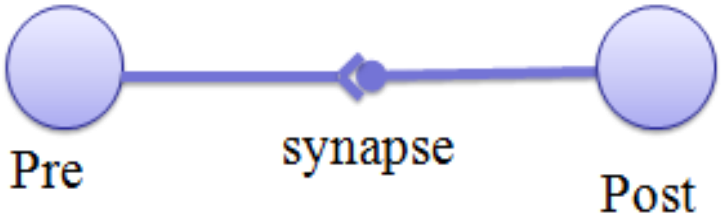
③ The presence of neuromodulatory substances, and

④ The presence of neurotransmitters.

Prinz, Astrid A. "Neuronal plasticity: how do neurons know what to do?." Current Biology 24.21 (2014): R1044-R1046.

Synaptic Plasticity





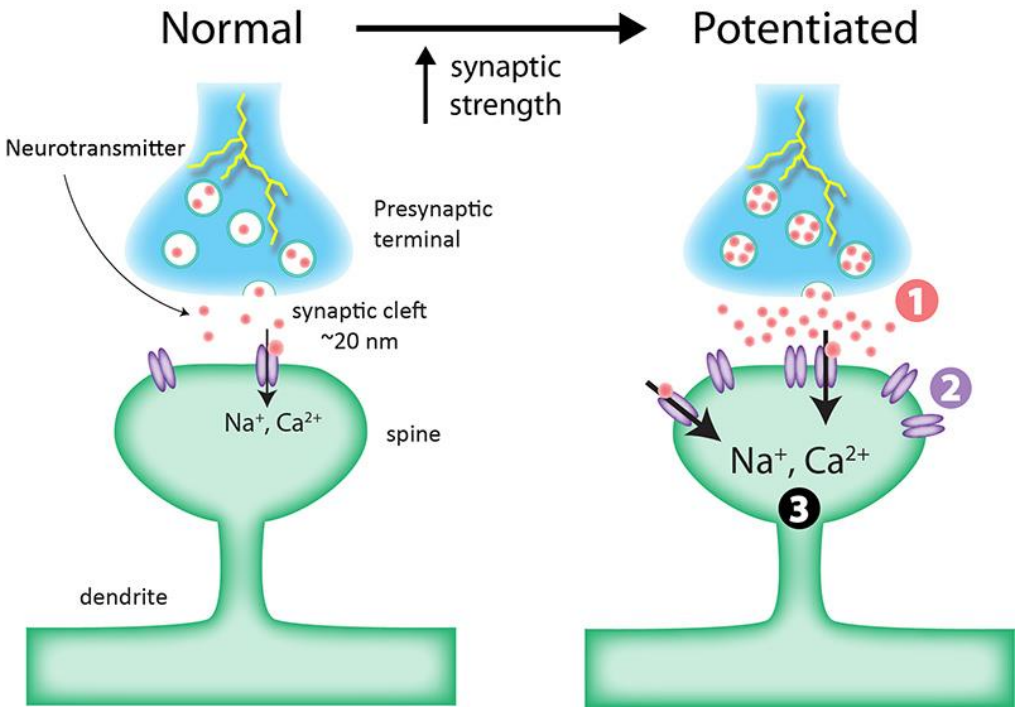
- Synapses play an important role in development, memory and learning of neural structures.
- Synaptic plasticity (learning) describes how changes in synaptic efficacy occur.
- Synaptic plasticity is the ability of synapses to strengthen or weaken over time, in response to increases or decreases in their activity.
- Synaptic plasticity provides the basis for most models of learning, memory and development in neural circuits.

How do synapses change their strength?

After a neuron fires an action potential, there are three main steps to synaptic transmission:

1. Neurotransmitter release;
2. Binding of neurotransmitter to postsynaptic receptors;
3. Opening of ion channels in the postsynaptic neuron, which allows electrical currents to flow in or out of the cell.

How do synapses change their strength?



As seen in the left figure, synaptic plasticity can change either

(1) the amount of neurotransmitter released or

(2) the number of postsynaptic receptors available;

(3) Both have the effect of altering how much electrical current flows through the ion channels;

These processes (1)-(3) change synaptic strength.

Types of Synaptic plasticity

- There are several mechanisms that cooperate to achieve synaptic plasticity, including changes in the quantity of neurotransmitters released into a synapse and changes in how effectively cells respond to those neurotransmitters.

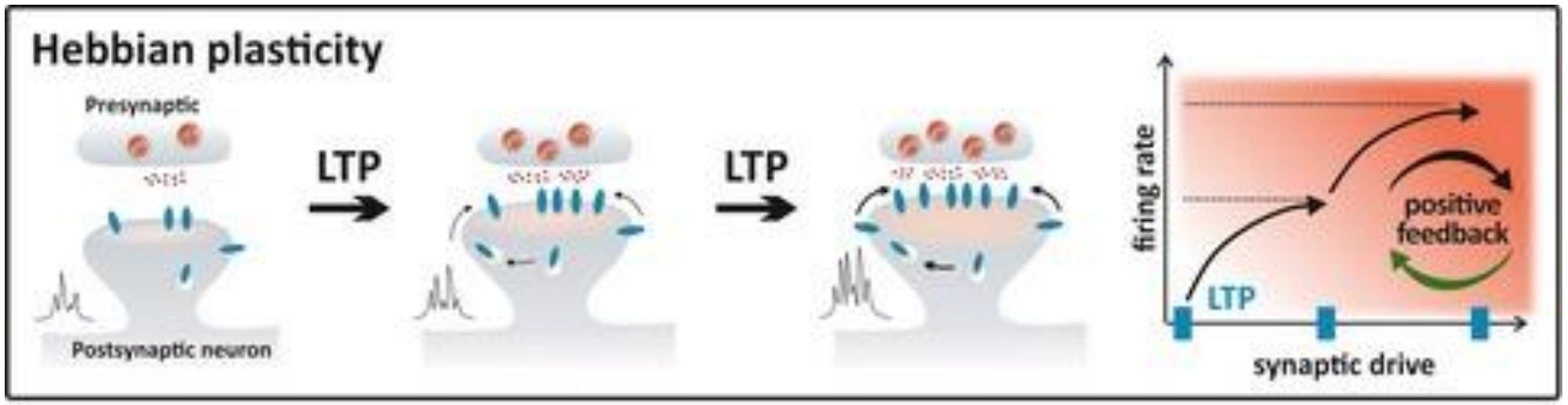
- Synaptic plasticity in both excitatory and inhibitory synapses has been found to be dependent upon postsynaptic calcium release.

- According to the time scales, there are two main types of Synaptic plasticity:
  1) Short-Term Synaptic plasticity;
  2) Long-Term Synaptic plasticity.

STP vs LTP

- The short-term synaptic plasticity (STP) acts on a timescale of tens of milliseconds to a few minutes;
- Long-term plasticity (LTP) lasts from minutes to hours.
- The Short-term plasticity（STP）is driven by correlations in afferent spike sequences (presynaptic correlations),
- Classical long-term plasticity（LTP）is driven by correlations between presynaptic and postsynaptic activities.

Hebbian Rule

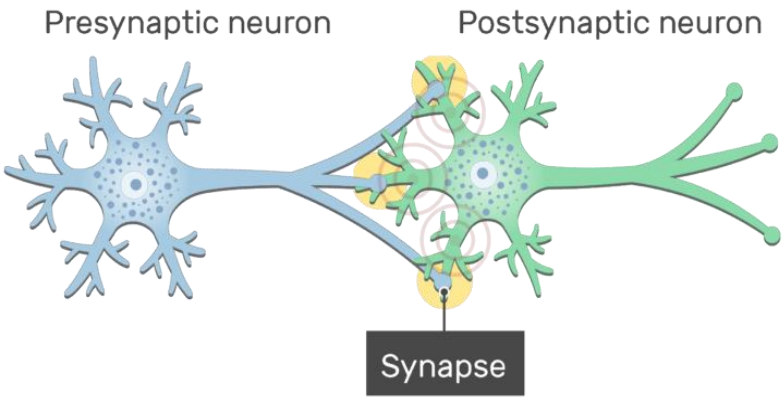


Hebbian forms of synaptic plasticity, such as long-term potentiation (LTP), induce long-lasting changes in synaptic strength, which can be destabilizing and drive activity to saturation.

Short-term synaptic plasticity

- Short-term plasticity (STP) also called dynamical synapses, refers to a phenomenon in which synaptic efficacy changes over time in a way that **reflects the history of presynaptic activity**.

- Two types of STP, with opposite effects on synaptic efficacy, have been observed in experiments. They are known as Short-Term Depression (STD) and Short-Term Enhancement (STE).



Short-Term Enhancement (STE)

- Short-term synaptic enhancement results from an increased probability of synaptic terminals releasing transmitters in response to pre-synaptic action potentials.

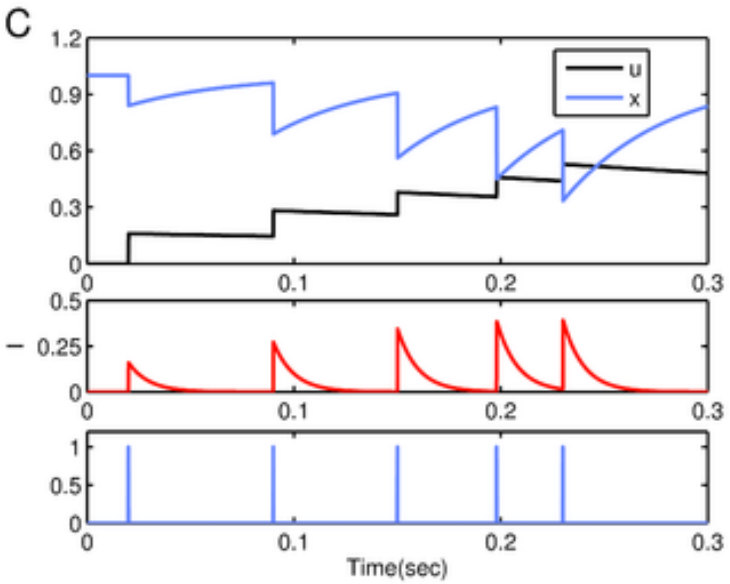
- Synapses will strengthen for a short time because of an increase in the amount of packaged transmitter released in response to each action potential.

- Depending on the time scales over which it acts synaptic enhancement is classified as neural facilitation, synaptic augmentation or post-tetanic potentiation.

Short-Term Enhancement (STE)

- Neural facilitation also known as paired-pulse facilitation (PPF), is a phenomenon in neuroscience in which postsynaptic potentials (PSPs) (EPSPs or IPSPs) evoked by an impulse are increased when that impulse closely follows a prior impulse.

- Neural facilitation may be involved in several neuronal tasks, including simple learning, information processing, and sound-source localization.



U means the voltage of post-synaptic neuron, x means the number of neurotransmitter between post- and pre- synapse.

The synaptic efficacy is increased gradually by spikes, and consequently the synapse is facilitation-dominated.

Tsodyks, Misha; Pawelzik, Klaus and Markram, Henry (1998). Neural Networks with Dynamic Synapses. *Neural Computation.* 10(4): 821-835.

Short-Term Enhancement (STE)

Facilitation of excitatory post-synaptic current (EPSC) can be quantified as a ratio of subsequent EPSC strengths. Each EPSC is triggered by pre-synaptic calcium concentrations and can be approximated by:

$$EPSC = k([Ca^{2+}]_{presynaptic})^4 = k([Ca^{2+}]_{rest} + [Ca^{2+}]_{influx} + [Ca^{2+}]_{residual})^4$$

Where k is a constant.

$$Facilitation = EPSC_2 / EPSC_1 = (1 + [Ca^{2+}]_{residual} / [Ca^{2+}]_{influx})^4 - 1$$

Jianhua, Xu; Liming, He; Ling-Gang, Wu (2007), "Role of Ca2+ channels in short-term synaptic plasticity", Current Opinion in Neurobiology

Short-Term Enhancement (STE)

- Synaptic augmentation is one of several forms of short-term synaptic plasticity that increases the probability of releasing synaptic vesicles during and after repetitive stimulation.

- Augmentation can be differentiated from the other components of enhancement by its kinetics of decay and by pharmacology. Augmentation selectively decays with a time constant of about 7 seconds and its magnitude is enhanced in the presence of barium.

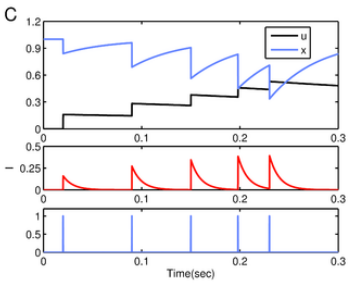
Short-Term Enhancement (STE)

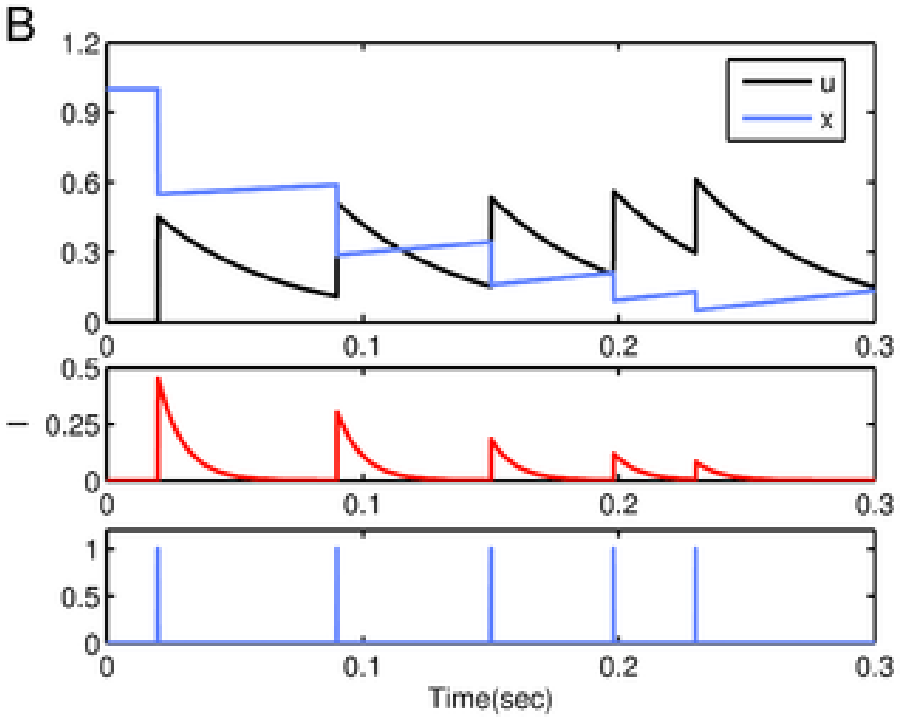
- Post-tetanic potentiation (PTP) is a form of synaptic plasticity which is short-lived and results in increased frequency of miniature excitatory postsynaptic potentials (mEPSPs) or currents (EPSCs) with no effect on amplitude in the spontaneous postsynaptic potential. It usually lasts in the range of several minutes (shorter potentiations are usually referred to as 'augmentations').

- PTPs are observed when synapses are stimulated with repetitive (tetanic) pulses, by means of prolonged trains of stimuli applied at high frequencies (10 Hz to 200 Hz stimuli applied for .2 seconds to 5 seconds).

Short-Term Depression (STD)

STD is caused by depletion of neurotransmitters consumed during the synaptic signaling process at the axon terminal of a pre-synaptic neuron.





U means the voltage of post-synaptic neuron, x means the number of neurotransmitter between post- and pre- synapse.

The post-synaptic current generated by an STD-dominated synapse

Tsodyks, Misha; Pawelzik, Klaus and Markram, Henry (1998). Neural Networks with Dynamic Synapses. *Neural Computation.* 10(4): 821-835.

Long-Term Synaptic plasticity

- Long-term synaptic plasticity was first reported in 1973. Studying a pathway in the rabbit hippocampus, researchers discovered that rapidly and repeatedly activating the synapses made them stronger; the volume control was turned up and stayed that way.

- They called this long-lasting increase in synaptic strength long-term potentiation, or LTP. The reverse phenomenon, in which synapses become weaker for extended periods, also exists, and is called long-term depression, or LTD.

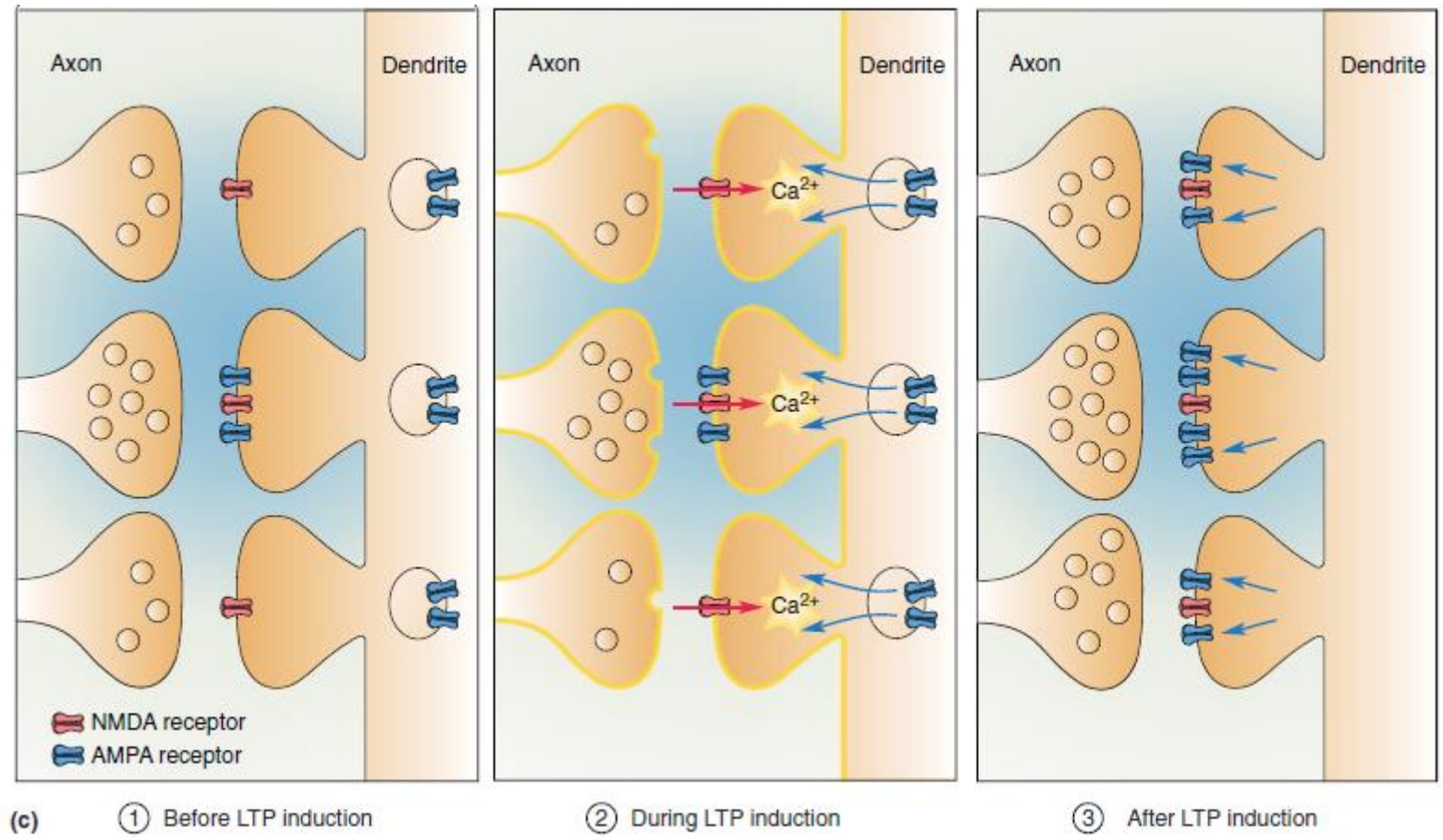
Bliss TV, Lomo T. Long-lasting potentiation of synaptic transmission in the dentate area of the anaesthetized rabbit following stimulation of the perforant path. J Physiol. 1973;232(2):331-356. doi:10.1113/jphysiol.1973.sp010273

Long-Term Synaptic Potentiation

- Long-term potentiation（LTP), is an increase in synaptic response following potentiating pulses of electrical stimuli that sustains at a level above the baseline response for hours or longer.

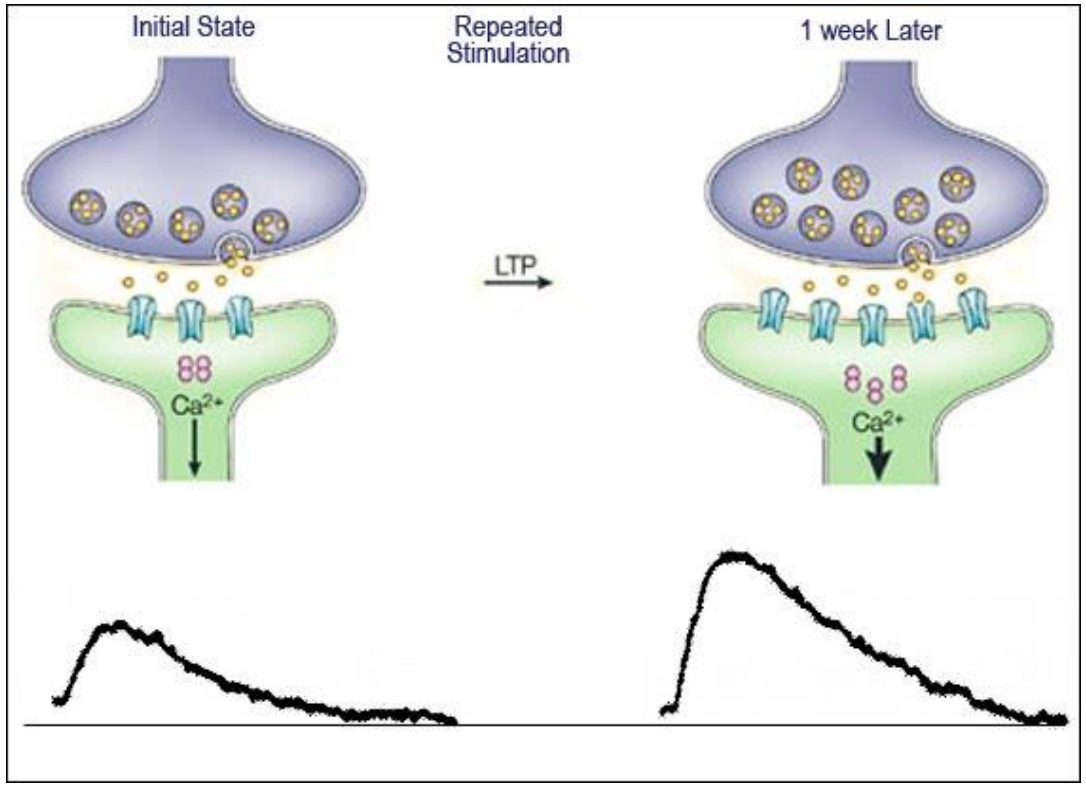
- LTP involves interactions between postsynaptic neurons and the specific presynaptic inputs that form a synaptic association, and is specific to the stimulated pathway of synaptic transmission.

Long-Term Synaptic Potentiation



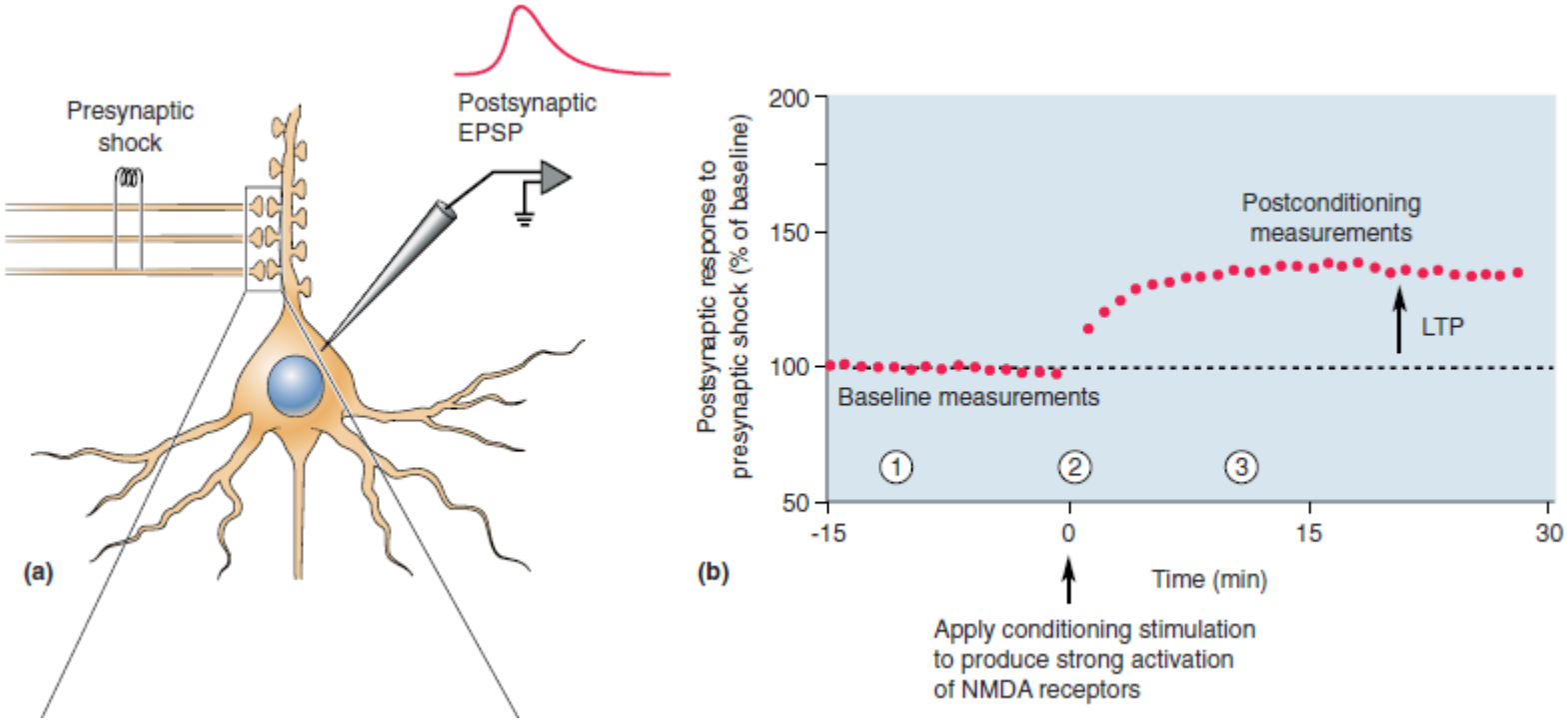
LTP at many synapses is associated with the insertion of AMPA receptors into synapses that previously had none. As illustrated in figure, after LTP induction, the number of AMPA receptors in post-synaptic neuron increases.

Long-Term Synaptic Potentiation



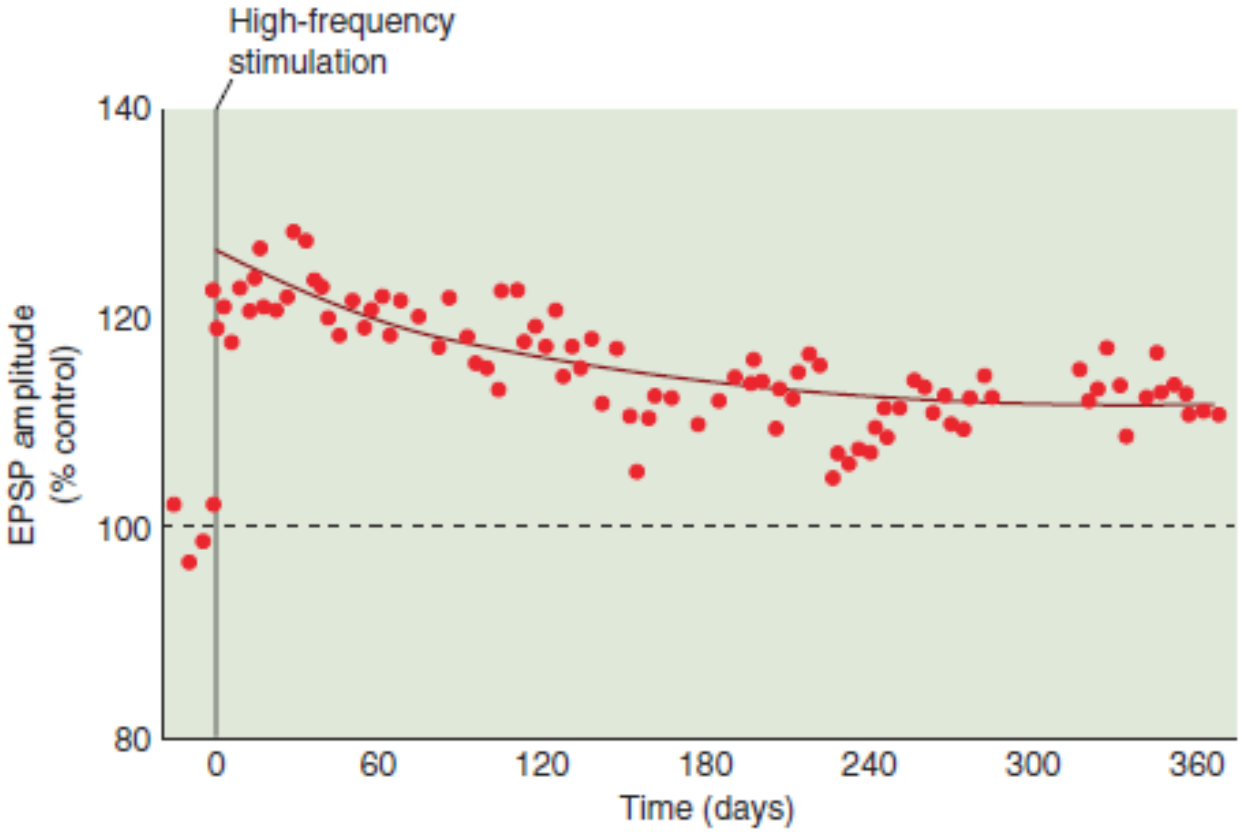
In LTP , the AMPA receptors become sufficiently excited which leads to an influx of Na+ which leads to a dramatic depolarization of the postsynaptic cell (Excitatory Post Synaptic Potential EPSP). This EPSP releases the Magnesium ion blocking the NMDA receptor, and allows a Calcium-glutamate molecule to enter the cell. As intracellular [Ca] increases protein kinases such as calcium/calmodulin-dependent protein kinase II (CaMKII) and protein kinase C (PKC) are activated. The activation of these two proteins allows for the 2 major mechanisms of LTP to proceed.

Long-Term Synaptic Potentiation



The left figure shows an experiment that presynaptic axons are stimulated electrically to evoke an action potential and the right figure shows how the strength of synaptic transmission is changed. LTP is the resulting enhancement of synaptic transmission.

Synaptic plasticity occurs with LTP



LTP can last a long, long time. In this experiment, LTP was induced with tetanic stimulation using electrodes implanted into the hippocampus of an awake rat. The LTP was still evident a year later.

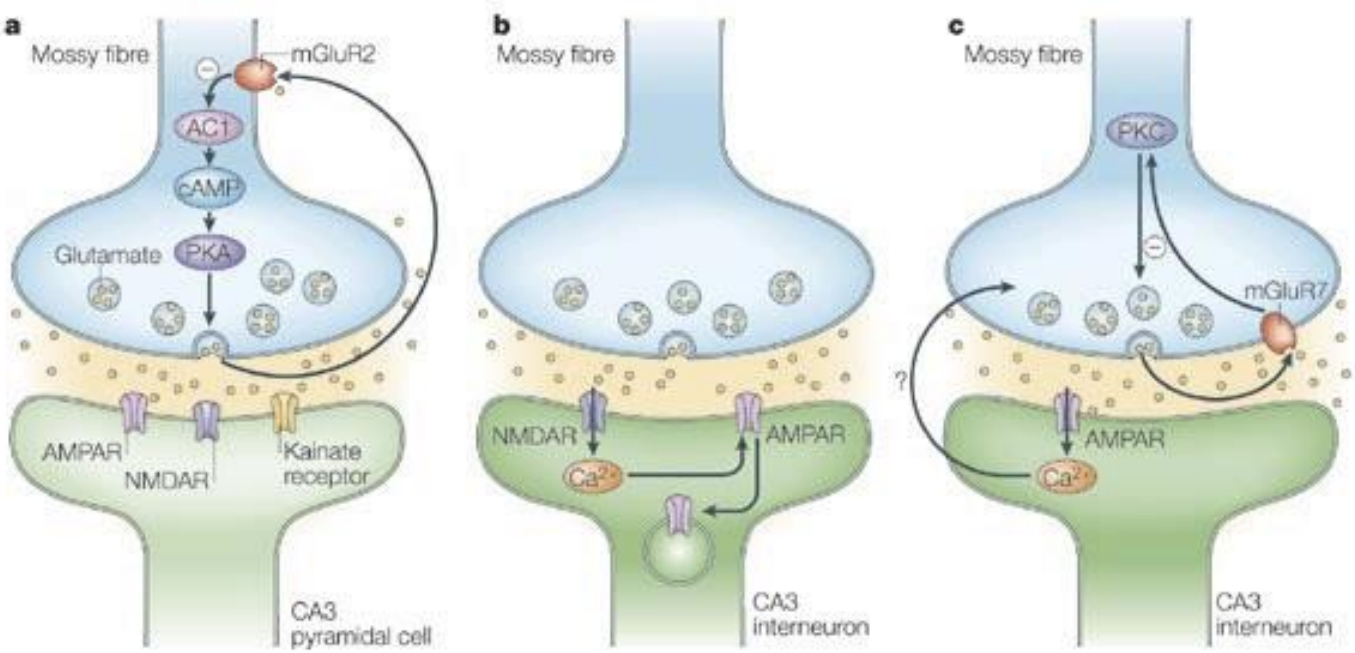
Long-Term Synaptic Depression

- Brief activation of an excitatory pathway can produce what is known as long-term depression (LTD) of synaptic transmission in many areas of the brain.

- LTD is induced by a minimum level of postsynaptic depolarization and simultaneous increase in the intracellular calcium concentration at the postsynaptic neuron.

- LTD is necessary because, if allowed to continue increasing in strength, synapses would ultimately reach a ceiling level of efficiency, which would inhibit the encoding of new information.

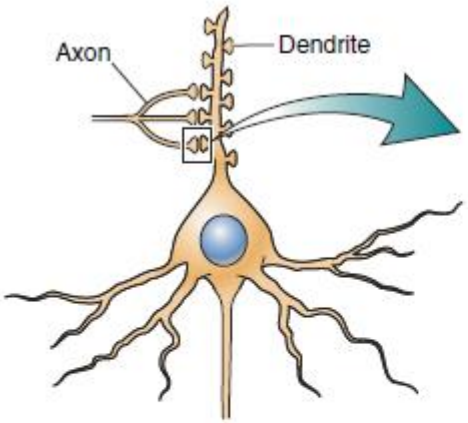
Long-Term Synaptic Depression

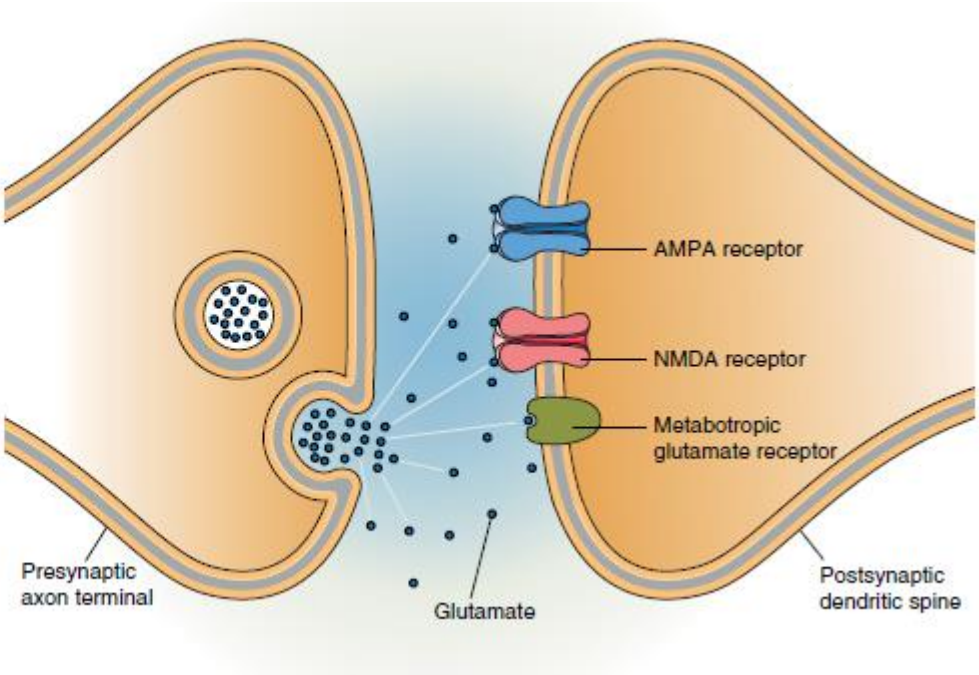


Generally the mechanism proceeds due to low frequency stimuli and a slow rise in postsynaptic [Ca]. In the hippocampus (displayed in figure) [Ca] below threshold level leads to an activation of phosphatases which dephosphorylate AMPAr's causing them to be internalized, thereby causing the total sensitivity of the synapse to decrease.

Correlation

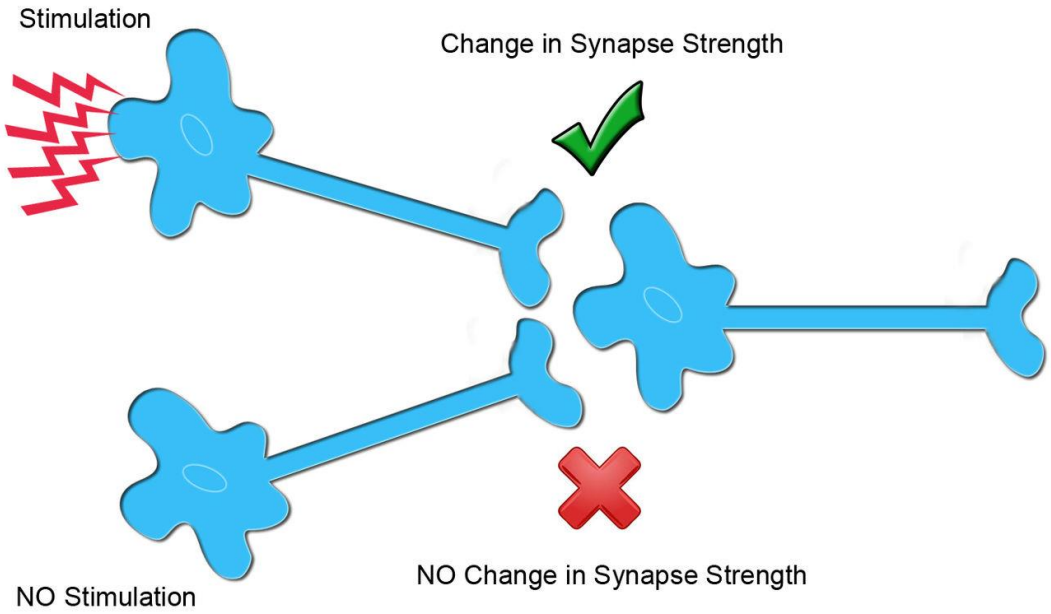
A single synapse has little influence on the firing rate of the postsynaptic neuron. The activity of the synapse must be correlated with the activity of many other inputs converging on the same postsynaptic neuron which is called correlation.



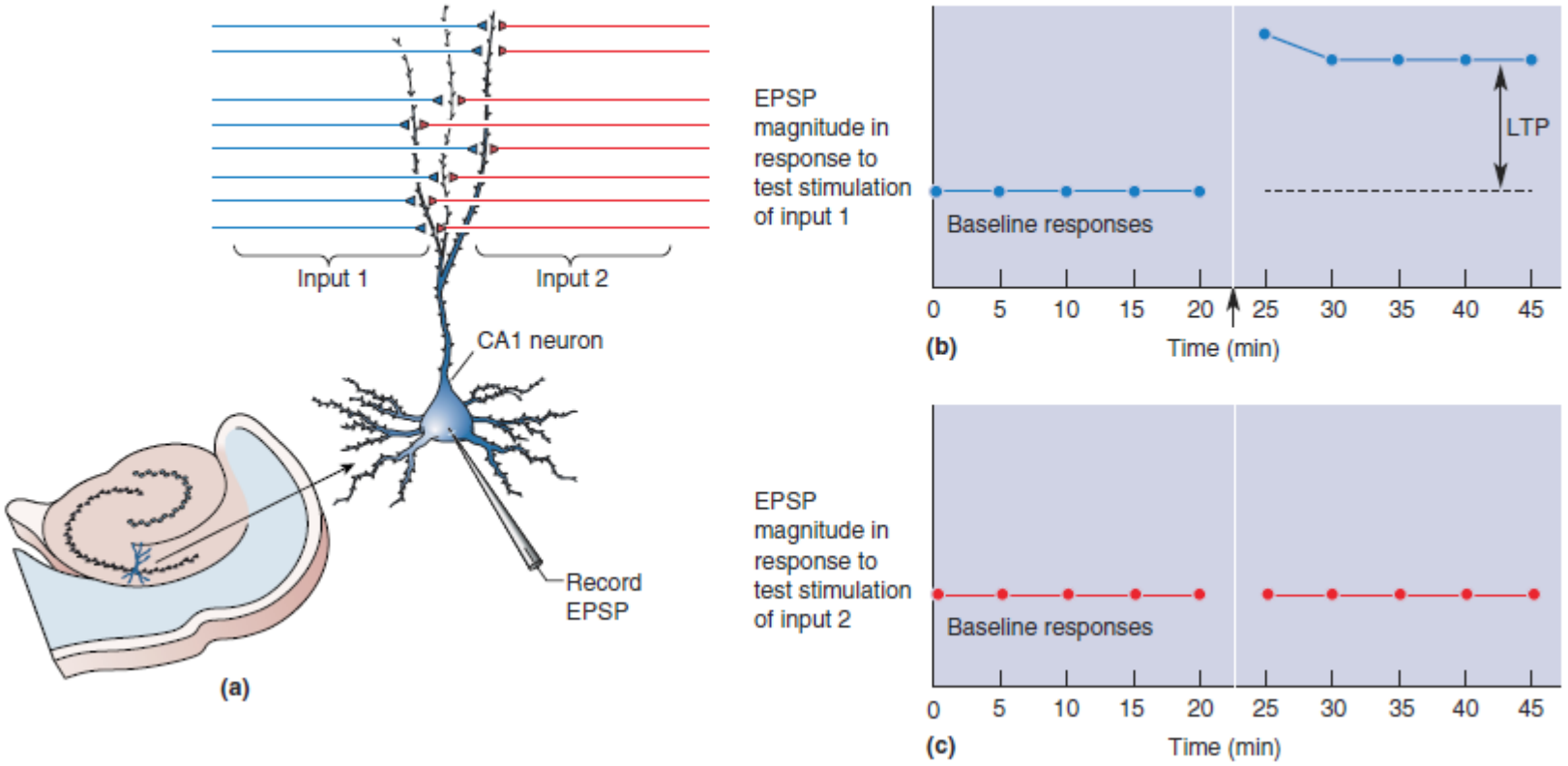


Homosynaptic plasticity

Homosynaptic plasticity is input-specific, meaning changes in synapse strength occur only at post-synaptic targets specifically stimulated by a pre-synaptic target. Therefore, the spread of the signal from the pre-synaptic cell is localized.

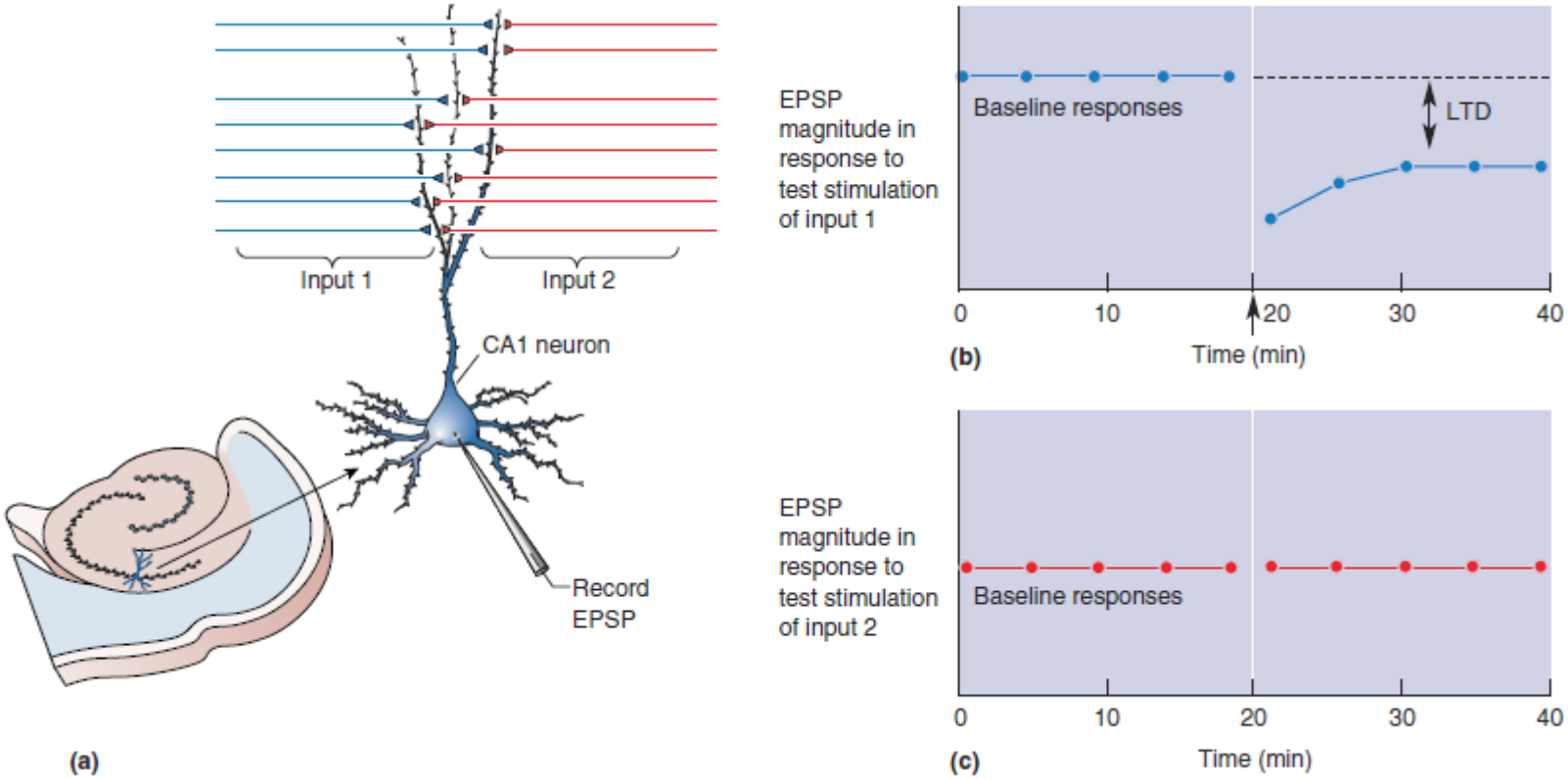


Homosynaptic plasticity in LTP



Long-term potentiation in Cornu Ammonis. The figure b shows a record of the experiment. The tetanus to input 1 (arrow) yields a potentiated response to stimulation of this input. (c) LTP is input-specific, so there is no change in the response to input 2 after a tetanus to input 1.

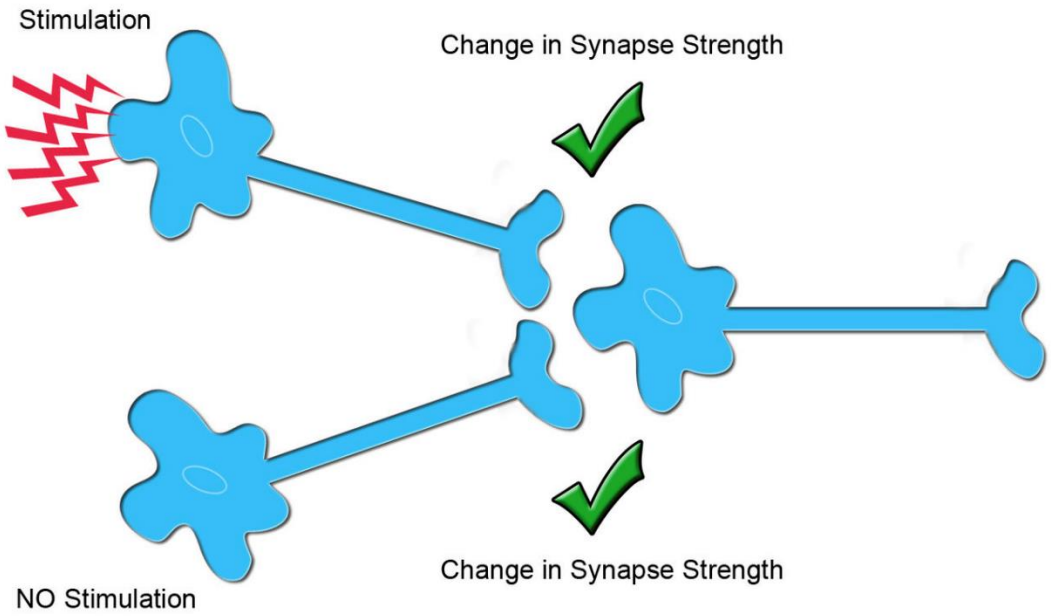
Homosynaptic plasticity in LTD



Homosynaptic LTD in the Cornu Ammonis. The figure b shows a record of the experiment. The tetanus to input 1 (arrow) yields a potentiated response to stimulation of this input. (c) LTD is input-specific, so there is no change in the response to input 2 after a tetanus to input 1.

Heterosynaptic plasticity

In the case of heterosynaptic plasticity, the activity of a particular neuron leads to input unspecific changes in the strength of synaptic connections from other unactivated neurons. These different forms of heterosynaptic plasticity contribute to a variety of neural processes including associative learning, the development of neural circuits, and homeostasis of synaptic input.



Hebbian Rule

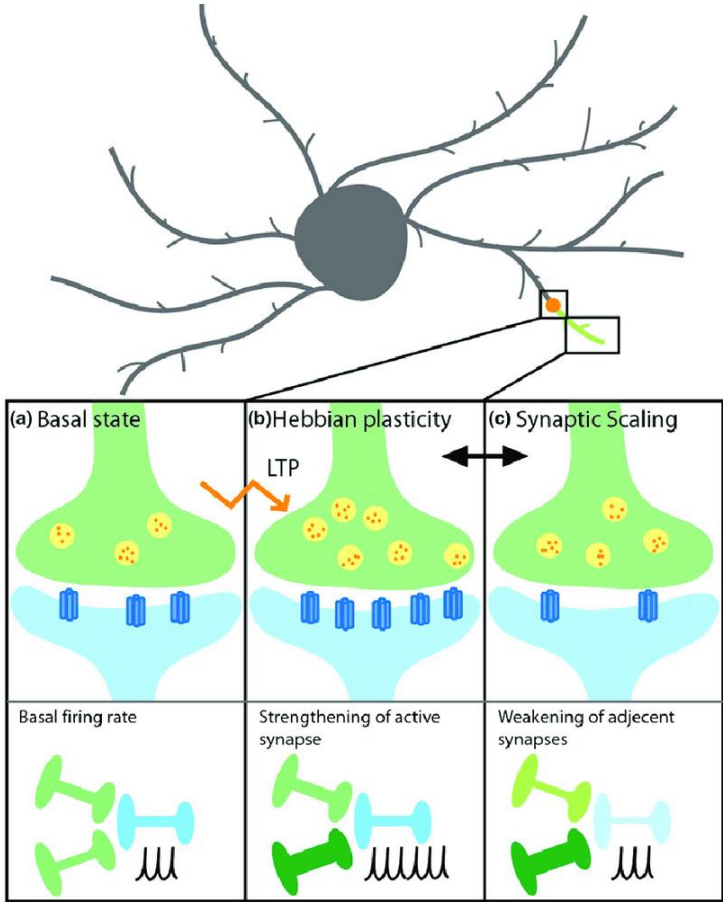
- Hebbian rule is a neuroscientific theory to explain synaptic plasticity, the adaptation of brain neurons during the learning process. It claims that an increase in synaptic efficacy arises from a presynaptic cell's repeated and persistent stimulation of a postsynaptic cell.

- This theory attempts to explain associative or Hebbian learning, in which simultaneous activation of cells leads to pronounced increases in synaptic strength between those cells. It also provides a biological basis for errorless learning methods for education and memory rehabilitation.

- In the study of neural networks in cognitive function, it is often regarded as the neuronal basis of unsupervised learning.

Hebbian Rule

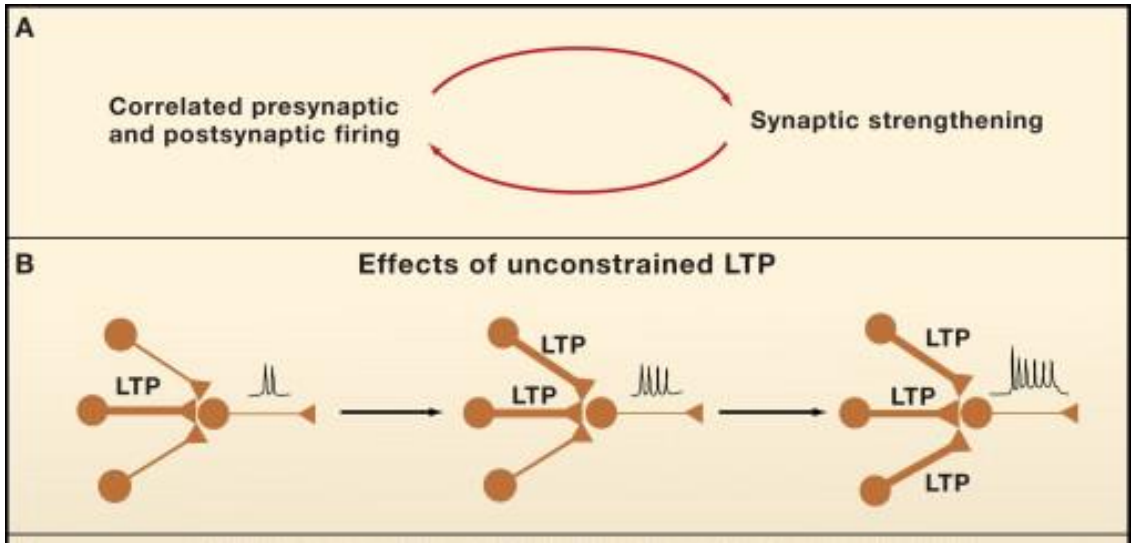
The theory is often summarized as “Neurons that fire together wire together."



“Neurons that fire together wire together."

The Self-Tuning Neuron

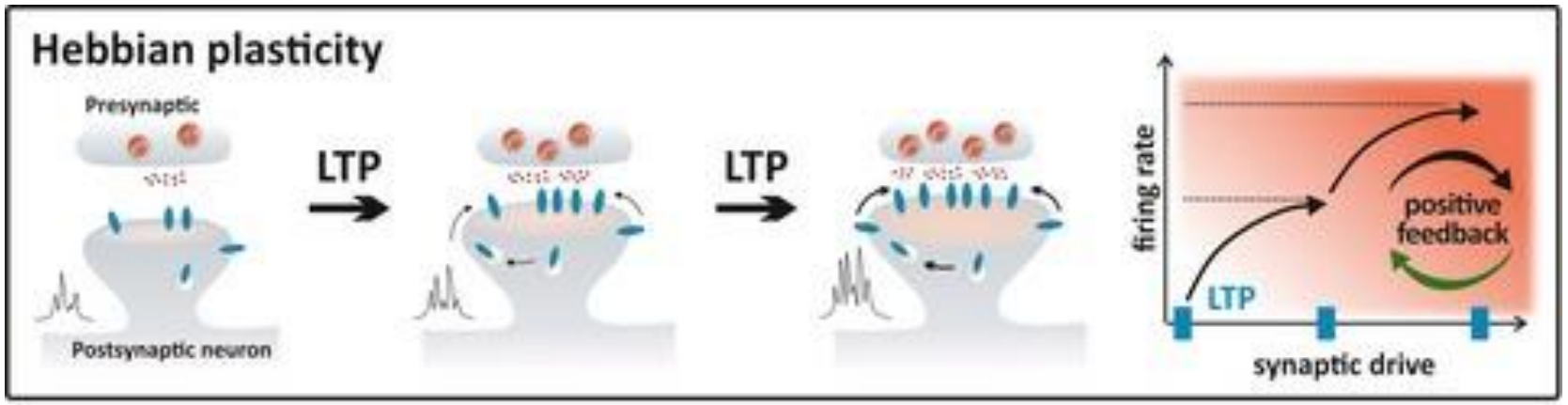
The many forms of plasticity based on correlated activity of presynaptic and postsynaptic neurons that have now been described biologically are each likely to carry with them their own unique destabilizing influences.



(A) Correlated presynaptic and postsynaptic firing induces long-term potentiation (LTP) , which increases the correlation between presynaptic and postsynaptic activation, and drives more LTP so on in an unconstrained positive feedback cycle.
(B) Unconstrained LTP will lose synapse specificity, because when one input undergoes LTP and drives the postsynaptic neuron more strongly, it makes it easier for other inputs to make the postsynaptic neuron fire.

Turrigiano G G. The self-tuning neuron: synaptic scaling of excitatory synapses[J]. Cell, 2008, 135(3): 422-435.

The Self-Tuning Neuron



Hebbian forms of synaptic plasticity, such as long-term potentiation (LTP), induce long-lasting changes in synaptic strength, which can be destabilizing and drive activity to saturation.

The Self-Tuning Neuron

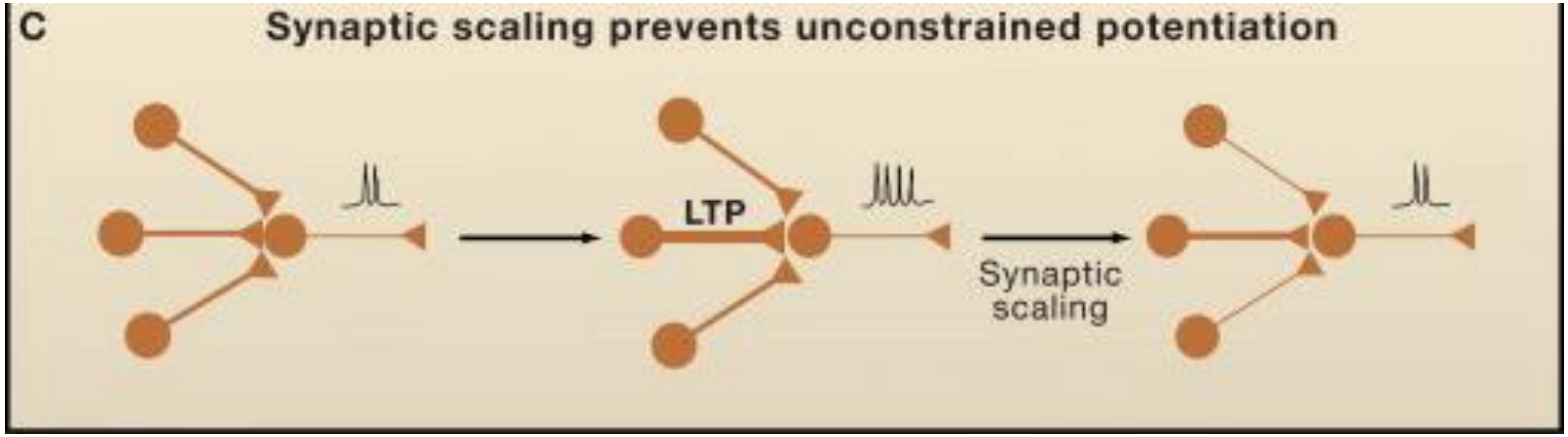
Current evidence suggests that neurons can adjust the strength of all of a neuron's excitatory synapses up or down to stabilize firing by using homeostatic synaptic plasticity.

Homeostatic plasticity refers to the capacity of neurons to regulate their own excitability relative to network activity, a compensatory adjustment that occurs over the timescale of days.

The term homeostatic plasticity derives from two opposing concepts: 'homeostatic' (a product of the Greek words for 'same' and 'state' or 'condition') and plasticity (or 'change'), thus homeostatic plasticity means "staying the same through change".

Homeostatic plasticity is also very important in the context of central pattern generators. In this context, neuronal properties are modulated in response to environmental changes in order to maintain an appropriate neural output.

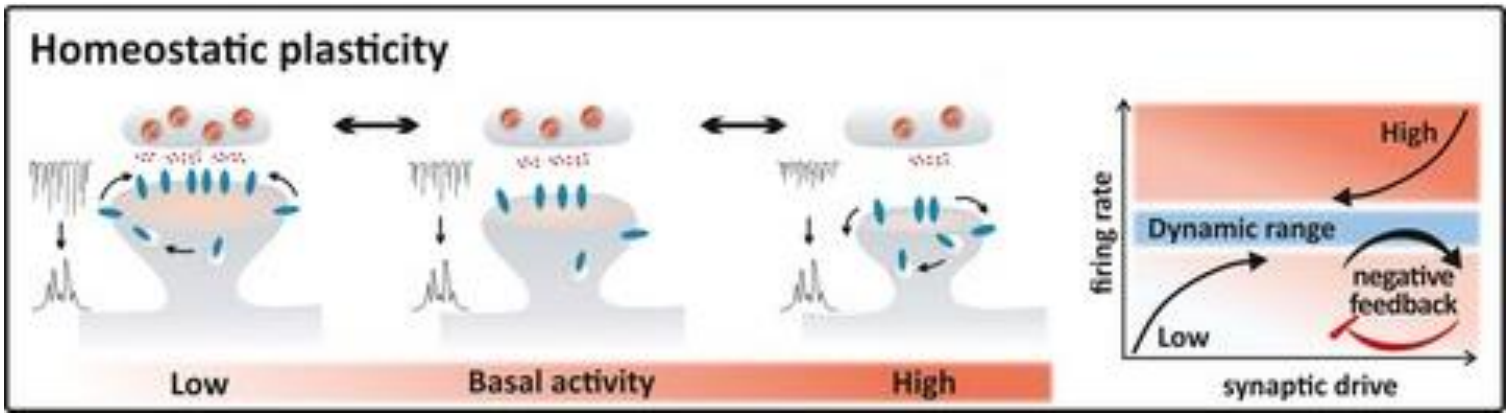
The Self-Tuning Neuron



Homeostatic plasticity prevents this runaway potentiation. When LTP of one input increases postsynaptic firing, synaptic scaling will reduce the strength of all synaptic inputs until the firing rate returns to control levels.

Turrigiano G G. The self-tuning neuron: synaptic scaling of excitatory synapses[J]. Cell, 2008, 135(3): 422-435.

The Self-Tuning Neuron



Homeostatic plasticity operates to compensate for prolonged activity changes, stabilizing neuronal firing within a dynamic physiological range.

The Self-Tuning Neuron

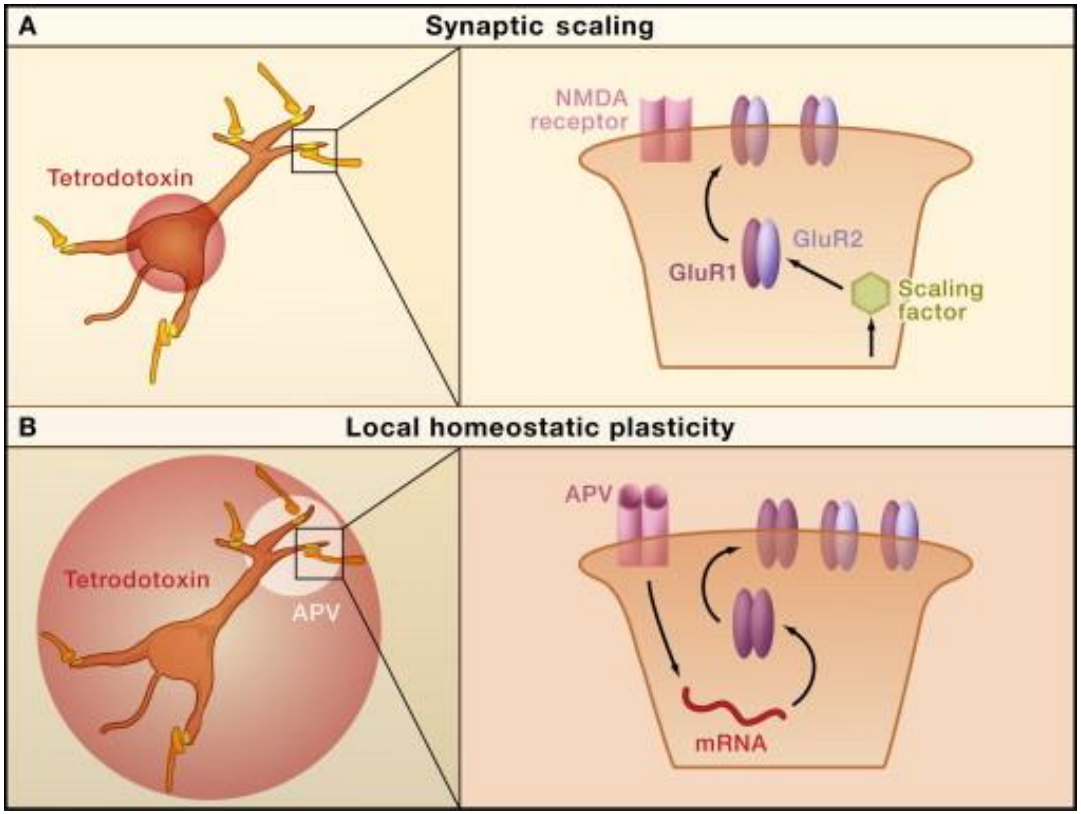


Figure 3. Distinct Mechanisms of Global and Local Changes in Synaptic Strength

(A) Blocking postsynaptic firing while leaving presynaptic and network activity intact scales up synaptic strengths in the dendrites, via a mechanism that results in increased accumulation of both GluR1 and GluR2 subunits of AMPA receptors.
(B) When action potential firing is blocked and NMDA receptor activation is locally blocked with the antagonist APV, there is a local increase in synaptic GluR1 accumulation that requires local dendritic protein synthesis.

Global and local homeostatic plasticity are likely to interact with forms of plasticity such as LTP and LTD rather differently..

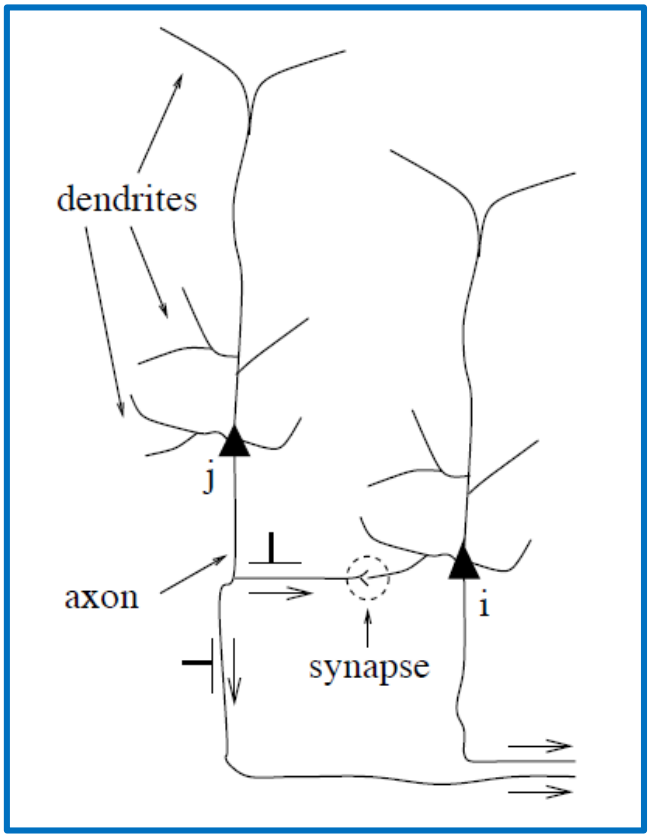
可塑性与学习规则: 计算模型

Plasticity and Learning Rules

什么是学习?

- In neural network model, each synapse is characterized by a single constant parameter **wij** that determines the **amplitude of the postsynaptic response to an incoming action potential**.
- The process of parameter adaptation is called **learning** and the algorithm for adjusting the weights is referred to as a **learning rule**.
- The weight wij of a connection from neuron j to i is considered as **a parameter that can be adjusted so as to optimize the performance of a network for a given task.**

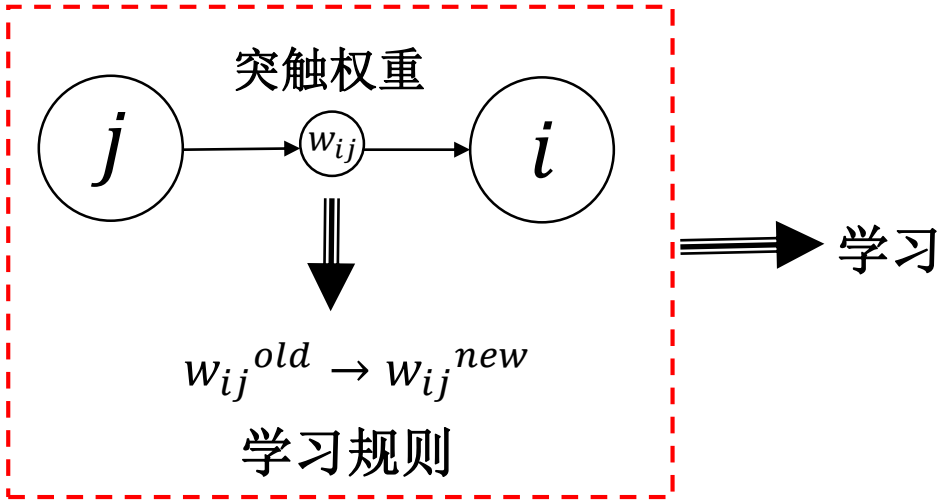
Learning Rule → **Optimize the performance of a network**

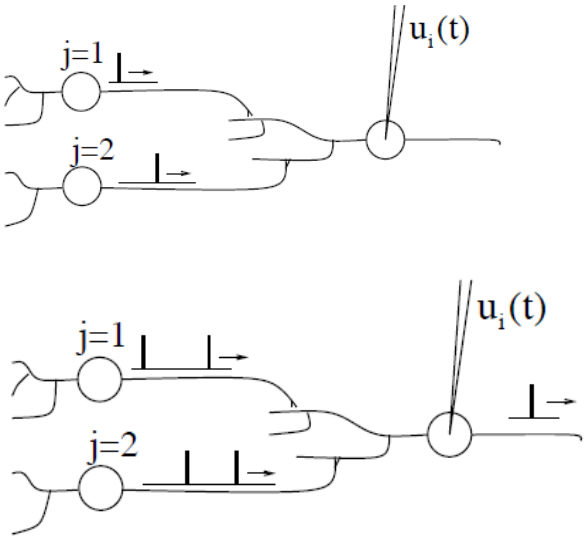


什么是学习?

- **突触权重/效能**(synaptic weight/ efficacy):神经网络中，从神经元j到i的突触连接由权重参数 $\boldsymbol{w_{ij}}$ 表示，该参数决定了传入动作电位（action potential）的突触后响应幅度，可以调整以优化给定任务的网络性能。

- **学习&学习规则:**
  - ✓ 参数自适应过程称为学习;
  - ✓ 调整权重的过程称为学习规则。





Hebb 规则

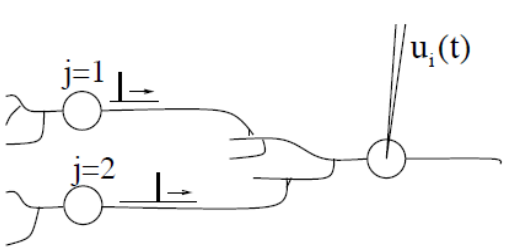
- Hebb假说（Hebb's postulate）：

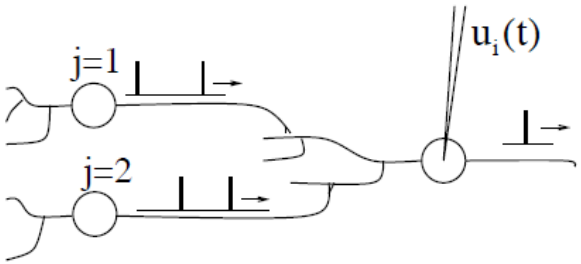
When an axon of cell A is near enough to excite a cell B and repeatedly or persistently takes part in firing it, some growth process or metabolic change takes place in one or both cells such that A's efficiency, as one of the cells firing B, is increased.

——D. O. Hebb, 1949

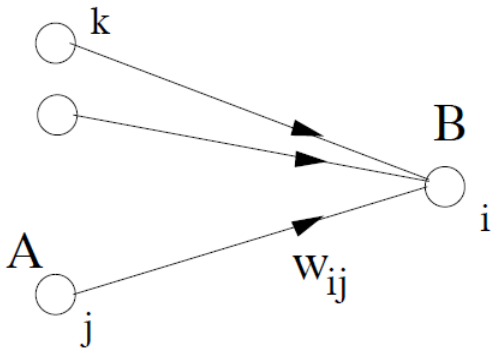
✓ Hebb假说描述了一种根据突触前（presynaptic）神经元A和突触后（postsynaptic）神经元B的相关活动修改突触连接的基本准则：

"突触传递效能（synaptic transmission efficacy）的改变是由突触前和突触后神经元的放电活动的相关性（correlations）驱动的"





Hebb 规则



突触权重w_{ij}的变化取决于突触前神经元j和突触后神经元i的状态以及当前的权值w_{ij}，但对其他神经元的状态没有影响。

如何刻画相关性？

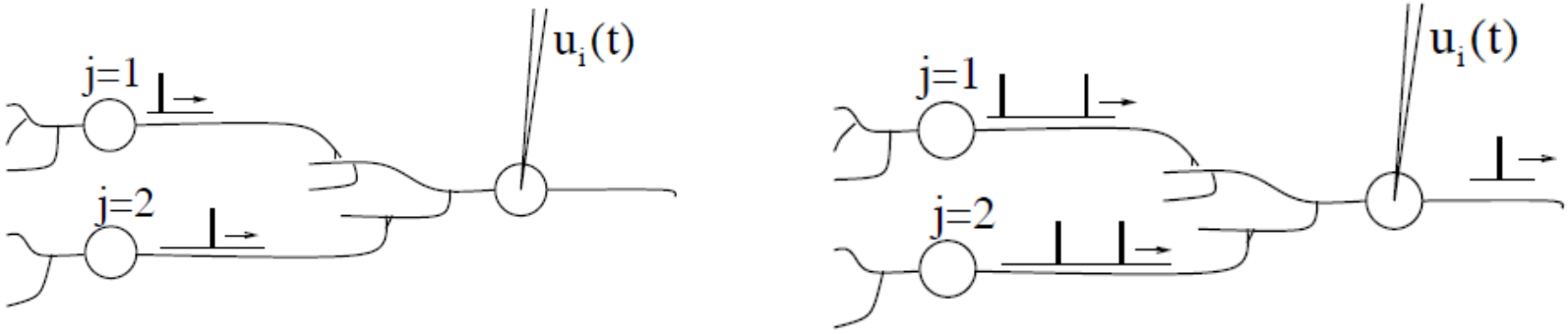
长时程增强
Long-Term Potentiation (LTP)

- 这种基于**相关性的学习**通常被称为 Hebbian learning

Hebb 规则

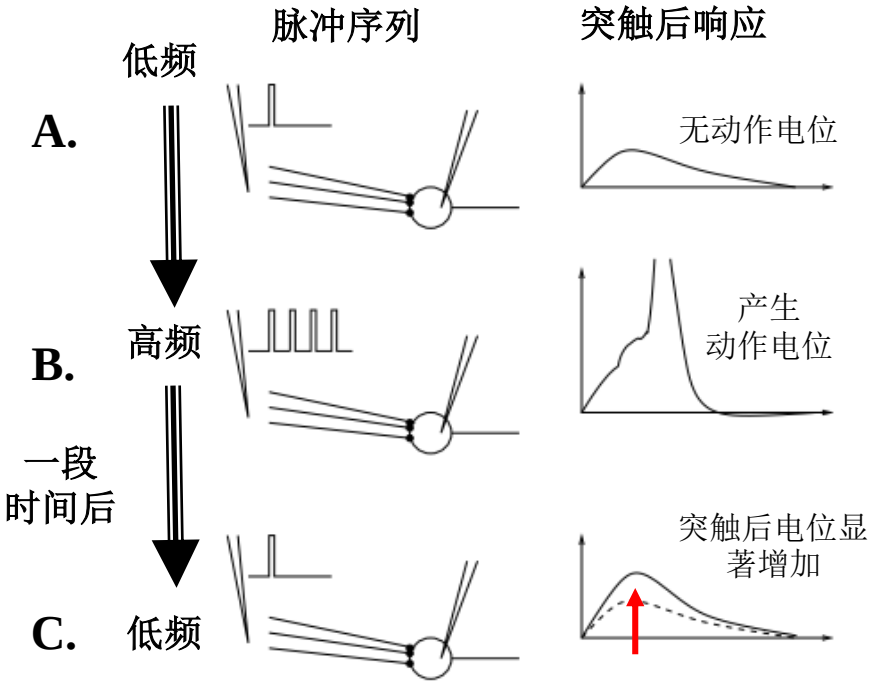
- Hebb规则和脉冲神经元活动的对应

“突触传递效能(synaptic transmission efficacy)的改变是由突触前和突触后神经元的放电活动的相关性(correlations)驱动的”



长时程增强 Long-Term Potentiation (LTP)

- **长时程增强 (Long-Term Potentiation, LTP):**突触前后神经元的联合活动引起突触强度持续增加（几个小时）。

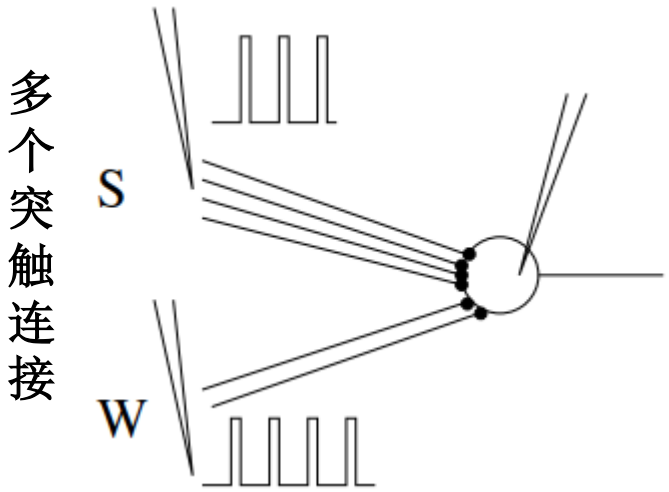


- ✓ 经过高频脉冲刺激一段时间后，再次施加测试脉冲会比初始响应（A）产生更大的突触后响应（C）；
- ✓ **表明突触出现了LTP，与Hebb假设一致**

长时程增强 Long-Term Potentiation (LTP)

强刺激通路S:
可单独激活突触后神经元发放脉冲

弱刺激通路W:
不可单独激活突触后神经元发放脉冲



- ✓ W和S通道单独输入100Hz刺激不会在W的突触上引起LTP，如果两种刺激同时发生，W突触才会增强；
- ✓ **表明LTP诱导具有协同性：突触前和突触后活动都是诱导LTP所必须的。**

基于速率的Hebb规则

Rate-Based Hebb Rules

Rate-Based Hebbian Learning

➢ Hebbian学习规则公式：

$$\frac{\mathrm{d}}{\mathrm{d}t}w_{ij} = F(w_{ij};\nu_i,\nu_j)\,.$$

$$\frac{\mathrm{d}}{\mathrm{d}t}w_{ij} = c_2^{\mathrm{corr}}\,\nu_i\,\nu_j\,.$$

➢ 两点特性：

✓ **局部性：**突触效能的变化只能依赖于局部变量(突触前后发放率v_i, v_j）和突触效能的实际值w_{ij}，而不依赖于其他神经元的活动。

✓ **协同性：**突触前和突触后神经元必须同时活跃，才能发生突触权重变化。

Cells that fire together, wire together

$$\begin{cases} c_2^{\mathrm{corr}} > 0 & \text{Hebbian} \\ c_2^{\mathrm{corr}} < 0 & \text{Anti-Hebbian（反Hebb规则）} \end{cases} \Longrightarrow \text{同时激活时，突触削弱}$$

Rate-Based Hebbian Learning

➢ 突触权重约束：

$$\frac{\mathrm{d}}{\mathrm{d}t}w_{ij} = F(w_{ij};\nu_i,\nu_j)\,.$$

$$\frac{\mathrm{d}}{\mathrm{d}t}w_{ij} = c_2^{\mathrm{corr}}\,\nu_i\,\nu_j\,.$$

如果仅依赖协同性，突触强度会无限增大，或始终处于饱和值！

协同性：突触前和突触后神经元必须同时活跃，才能发生突触权重变化。

$$c_2^{\mathrm{corr}}(w_{ij}) = \gamma_2\,(1 - w_{ij})$$

突触权重饱和：

w_{ij}不可独立于F, 否则持续施加相同的增强刺激，权重会无限增长；需保证w_{ij}达到最大值（$w^{max} = 1$）时， c_2^{corr}趋近于0.

Rate-Based Hebbian Learning

- **突触权重饱和：**
  - ✓ 最初Hebbian学习不包含突触权重减少的规则，因此权重最终都会在最高值处饱和。
- **突触抑制/衰减（synaptic depression）：**
  - ✓ 突触抑制是任何有用学习规则的必要条件，可以通过权重衰减来实现：

$$c_0(w_{ij}) = -\gamma_0 w_{ij} .$$

γ_0是较小正值，描述了在没有刺激的情况下突触衰减回零的速率。

$$\frac{\mathrm{d}}{\mathrm{d}t} w_{ij} = \gamma_2 (1 - w_{ij}) \nu_i \nu_j - \gamma_0 w_{ij} .$$

突触权重“软”边界（soft bounds）：

- $w_{ij} = 1$:持续刺激时饱和；
- $w_{ij} = 0$:无刺激时指数衰减至0。

TIPs：突触权重“硬”边界（hard bounds）：将突触权重限制在有限的区间内，即，只有权重保持在其限制范围内，才应用具有权重无关参数（ weight-independent parameters ）的学习规则。

Hebb 学习规则变体

- 之前给出的Hebb学习规则公式只是一种可能，可以**指定突触重量的增长和衰减规则给出多种基于Hebb学习规则的变体**。

post ν_i	pre ν_j	$dw_{ij}/dt \propto \nu_i \nu_j$	$dw_{ij}/dt \propto \nu_i \nu_j - c_0$	$dw_{ij}/dt \propto (\nu_i - \nu_\theta)\nu_j$	$dw_{ij}/dt \propto \nu_i (\nu_j - \nu_\theta)$	$dw_{ij}/dt \propto (\nu_i - \langle\nu_i\rangle)(\nu_j - \langle\nu_j\rangle)$
ON	ON	+	+	+	+	+
ON	OFF	0	−	0	−	−
OFF	ON	0	−	−	0	−
OFF	OFF	0	−	0	0	+

✓ Standard Hebb

✓ Hebb with decay

✓ presynaptic gating

✓ postsynaptic gating

✓ covariance rule

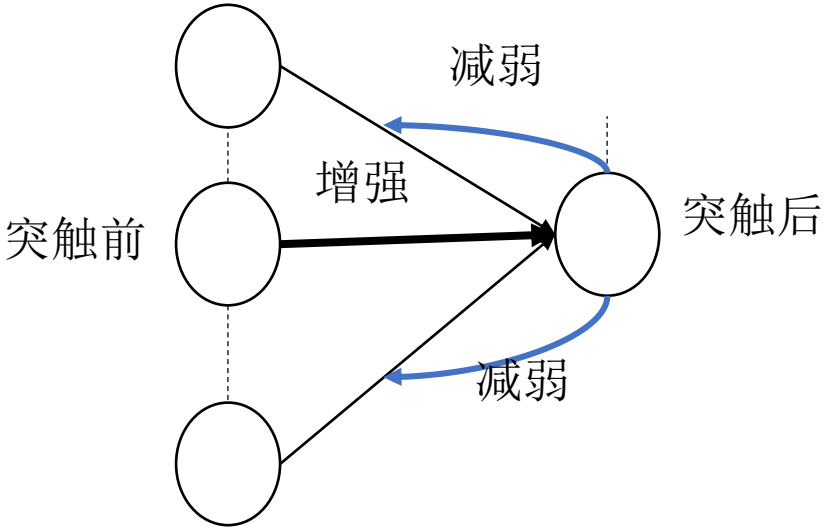
competitive learning

竞争机制 Competition

Hebb学习规则具有协同性，学习规则中另一个重要机制是竞争

- **竞争（competition）：**

突触权重增强只能以牺牲其他突触为代价。如果某一组突触得到加强，那么连接同一突触后神经元的其他突触就必须削弱。



- ✓ 竞争对于任何形式的自组织（self-organization）和模式形成至关重要，可通过将连接到同一突触后神经元上的所有**权重之和标准化来实现**。

竞争机制引入到Hebb学习中，催生出Pre- and Postsynaptic gating两种竞争学习的Hebb变体。

竞争学习规则 competitive learning

➢ **突触后门控（Postsynaptic gating）：**

✓ 权重变化是由突触后神经元“门控”的，也就是只有当突触后神经元活跃时 $v_i > 1$，权重才会发生变化：

$$\frac{\mathrm{d}}{\mathrm{d}t} w_{ij} = \gamma \nu_i \left[\nu_j - \nu_\theta(w_{ij})\right]$$

式中，γ为正常数，且v_θ是取决于的当前值w_{ij}的参考值；权重变化的方向取决于矩形括号中表达式的符号。

突触后门控假设突触后神经元由一组高度活跃的突触前神经元驱动（$v_i > 0$及$v_j > v_\theta$）。

✓ **同突触长时程增强 (homosynaptic LTP)**：高度活跃的突触前神经元与突触后神经元的连接突触权重LTP；

✓ **异突触长时程抑制 (heterosynaptic LTD)**：其他未被激活的突触前神经元与突触后神经元的连接突触权重LTD。

实现了一种竞争学习

✓ 一种常见设置$v_\theta(w_{ij}) = w_{ij}$

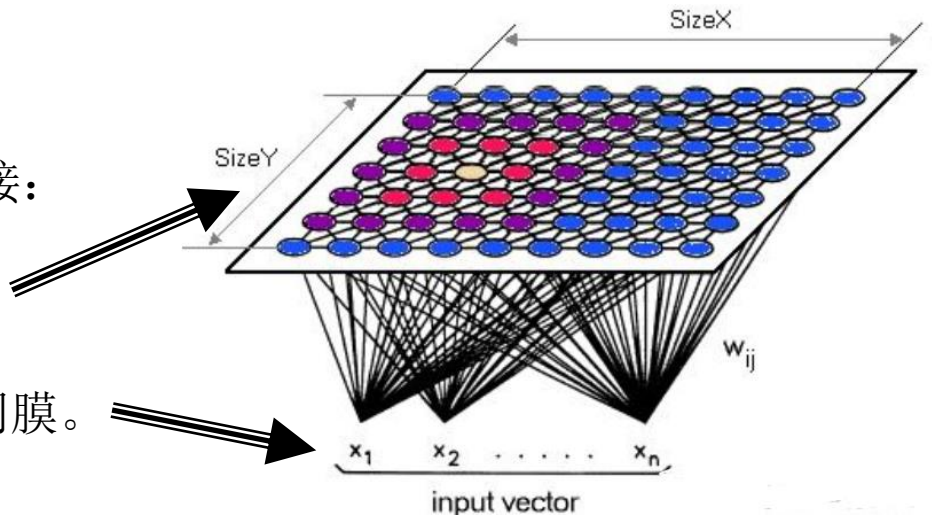
$$\frac{\mathrm{d}}{\mathrm{d}t} w_{ij} = \nu_i \left[\nu_j - w_{ij}\right].$$

- 突触后神经元活跃时，突触权重接近固定点$w_{ij} = v_j$
- 在静止状态下，权重值的集合w_{ij}反映突触前放电模式v_j

竞争学习规则 competitive learning

- 自组织网络（Kohonen Self-Organizing Map Model）

✓ SOM Model是一种运用**无监督的竞争学习规则**，依靠神经元之间**互相竞争逐步优化**的神经网络。



✓ 典型SOM网络共有两层,两层双向连接：

竞争层：模拟做出响应的大脑皮层；

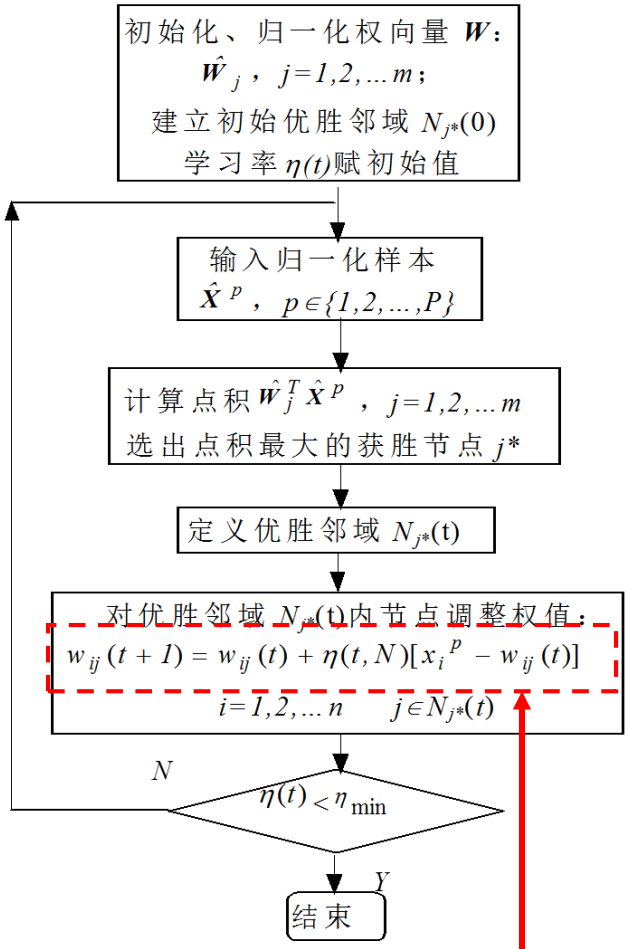
输入层：模拟感知外界输入信息的视网膜。

✓ **基本思想：**网络的竞争层各神经元竞争对输入模式响应的机会，竞争获胜的神经元表示对输入模式的分类。**获胜神经元对其邻近神经元的影响是由近及远，**对附近神经元产生**兴奋影响逐渐变为抑制**。获胜神经元与它周围的神经元都要进行不同程度的权值调整。

竞争学习规则 competitive learning

- SOM的竞争学习过程：

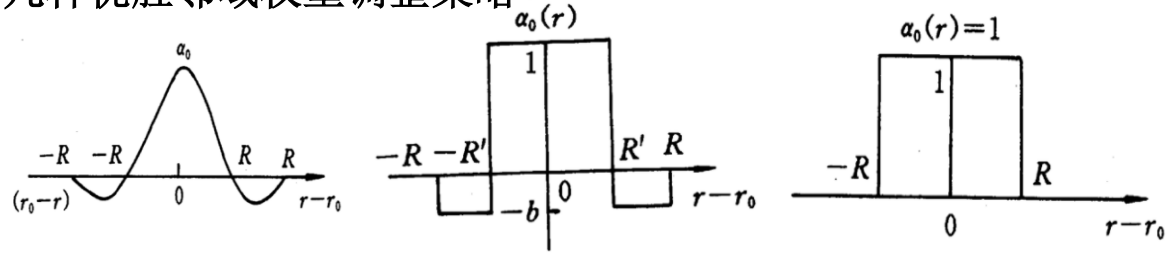
1. **网络初始化**：对竞争层各神经元权重随机初始并进行归一化处理；建立初始优胜领域与学习率；
2. **输入归一化**：假设有P个数据；
3. **寻找获胜神经元**：从与所有的点积中找到最大；
4. **根据优胜邻域确定权值调整域**；
5. **调整权重**：对优胜邻域内所有神经元调整权重。



（Postsynaptic gating）

- **优胜邻域**:获胜神经元为中心设定一个邻域半径R的范围。
  - ✓ 优胜邻域内的所有神经元均按其与获胜神经元的距离远近不同程度地调整权值。
  - ✓ 优胜邻域开始定得很大，但其大小随着训练次数的增加不断收缩，最终收缩到半径为零。

几种优胜邻域权重调整策略



竞争学习规则 competitive learning

- **突触前门控（Presynaptic gating）：**
  - ✓ 权重变化是由突触前神经元“门控”的（与突触后门控相反）

$$\frac{\mathrm{d}}{\mathrm{d}t}w_{ij} = \gamma\left(v_i - v_\theta\right)v_j$$

只有当突触前神经元活跃时$(v_j > 0)$，突触权重才会发生变化，**变化的方向由突触后神经元的活动决定**。

  - ✓ $\gamma > 0$：突触后神经元高度活跃$(v_i > v_\theta)$则突触增强；否则突触削弱。
  - ✓ $\gamma < 0$：

- **Anti-Hebbian learning：对突触后放电率有稳定作用**
  - ✓ 突触前发放率v_j保持不变，突触后发放率$v_i = g(\sum_j w_{ij}\, v_j)$收敛到$v_\theta$；

全局吸引子

$v_i < v_\theta$：所有突触增强，**增强输入**，突触后发放率v_j也增加；

$v_i > v_\theta$：所有突触减弱，**抑制输入**，突触后发放率v_i也减弱。

其他Hebb规则

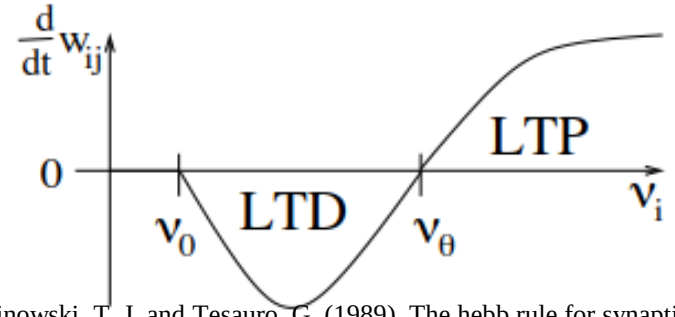
- **协方差规则（covariance rule）[1]：**
  - ✓ 将速率$v_i(t)$和$v_j(t)$围绕平均发放率$\langle v_i \rangle$和$\langle v_j \rangle$上下波动，即取最近发放历史上的运行平均值。

$$\frac{\mathrm{d}}{\mathrm{dt}} w_{ij} = \gamma \left(\nu_i - \langle \nu_i \rangle\right) \left(\nu_j - \langle \nu_j \rangle\right)$$

  - ✓ 平均发放率$\langle v_i \rangle$和$\langle v_j \rangle$需要在时间上恒定。

- **BCM规则（Bienenstock-Cooper-Munroe rule）[2]：**
  - ✓ 突触前门控规则推广：非线性函数$\emptyset$ +平均输出速率$\langle v_i \rangle$替代参考速率v_θ

$$\frac{\mathrm{d}}{\mathrm{dt}} w_{ij} = \eta\, \phi(\nu_i - \nu_\theta)\, \nu_j - \gamma\, w_{ij}$$



BCM以突触后活动的两个阈值v_0和v_θ为特征。
- ✓ 在v_0以下：未发生突触修改，
- ✓ 在v_0和v_θ之间：突触抑制，
- ✓ 超过v_θ：突触增强。

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[2] Bienenstock, E. L., Cooper, L. N., and Munroe, P. W. (1982). Theory of the devel-opment of neuron selectivity: orientation specificity and binocular interaction in visual cortex. J. Neurosci., 2:32–48. reprinted in Anderson and Rosenfeld, 1990. 371, 386, 393


Thank You!