

# Memristance can explain Spike-Time-Dependent-Plasticity in Neural Synapses

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Interdisciplinary research broadens the view of particular problems yielding fresh and possibly unexpected insights. This is the case of neuromorphic engineering where technology and neuroscience cross-fertilize each other. For example, consider on one side the recently discovered memristor<sup>1-3</sup>, postulated in 1971<sup>4</sup>, thanks to research in nanotechnology electronics. On the other side, consider the mechanism known as Spike-Time-Dependent-Plasticity<sup>5-11</sup> (STDP) which describes a neuronal synaptic learning mechanism that outperforms the traditional Hebbian synaptic plasticity<sup>12</sup> proposed in 1949. STDP was originally postulated as a computer learning algorithm<sup>5</sup>, and is being used by the machine intelligence and computational neuroscience community<sup>8-11</sup>. At the same time its biological and physiological foundations have been reasonably well established during the past decade<sup>13-20</sup>. If memristance and STDP can be related, then (a) recent discoveries in nanophysics and nanoelectronic principles may shed new lights into understanding the intricate molecular and physiological mechanisms behind STDP in neuroscience, and (b) new neuromorphic-like computers built out of nanotechnology memristive devices could incorporate the biological STDP mechanisms yielding a new generation of self-adaptive ultra-high-dense intelligent machines. Here we show that by combining memristance models with the electrical wave signals of neural impulses (spikes) converging from pre- and post-synaptic neurons into a synaptic junction, STDP behavior emerges naturally. This result serves to understand how neural and memristance parameters modulate STDP, which might bring new insights to neurophysiologists in searching for the ultimate physiological mechanisms responsible for STDP in biological synapses. At the same time, this result also provides a direct mean to incorporate STDP learning mechanisms into a new generation of nanotechnology computers employing memristors.

Memristance was postulated in 1971 by Chua<sup>4</sup> based on circuit theoretical reasonings and has been recently demonstrated in nanoscale two-terminal devices, such as certain titanium-dioxide<sup>1-2</sup> and amorphous Silicon<sup>3</sup> cross-point switches. Memristance arises naturally in nanoscale devices because small voltages can yield enormous electric fields that produce the motion of charged atomic or molecular species changing structural properties of a device (such as its conductance) while it operates. By definition<sup>4</sup> a memristor obeys equations of the form

$$\frac{dw}{dt} = f(w, v_{MR}) \tag{1}$$

$$i_{MR} = g(w, v_{MR})v_{MR} \tag{2}$$

where  $w$  is some physical (structural) parameter,  $i_{MR}$  is the current

through the device,  $v_{MR}$  the voltage drop across it, and  $g$  is its (nonlinear) conductance. In memristive nanoscale devices, function  $f$  may describe ionic drift under electric fields. Although a linear dependence of  $f$  with voltage  $v_{MR}$  yields memristive behavior<sup>1</sup>, it is clear that in reality such dependence is more likely to grow exponentially and/or include a threshold barrier  $v_{th}$ . For our discussions, let's assume the following generic dependence

$$f(v_{MR}) = A \operatorname{sign}(v_{MR}) \left[ e^{|v_{MR}|/v_o} - e^{v_o/v_{MR}} \right] \quad \text{if } |v_{MR}| > v_{th} \tag{3}$$

$$f(v_{MR}) = 0 \quad \text{otherwise}$$

where  $A$  and  $v_o$  are parameters which may or may not depend on  $w$ . The shape of  $f$  is shown in Fig. 1. Many other mathematical formulations can be used, but the bottom line is to describe a thresholding behavior, an exponential behavior beyond threshold, and a bidirectional behavior (symmetric or not).

Spike-time-dependent plasticity (STDP) is a learning mechanism originally postulated<sup>5</sup> in the context of artificial machine learning algorithms (or computational neuroscience) exploiting spike-based computations (as in brains). It has been shown to improve Hebbian correlation-based plasticity at explaining cortical phenomena<sup>10-11</sup>, and has been proven successful to learn hidden spiking patterns<sup>8</sup> or to perform competitive spike pattern learning<sup>9</sup>. Astonishingly, experimental evidences of STDP have been reported by several neuroscience groups worldwide during the past decade<sup>13-20</sup>, so that today we can state that the physiological existence of STDP has been reasonably well established. However, the ultimate molecular and electro-chemical principles behind STDP are still under debate<sup>21</sup>. Before describing STDP mathematically, let us first explain how neurons interchange information and what the synaptic connections are. Fig. 2 illustrates two neurons connected by a synapse. The pre-synaptic neuron is sending a pre-synaptic spike  $V_{mem-pre}(t)$  through one of its axons to the synaptic junction. Neural spikes are membrane

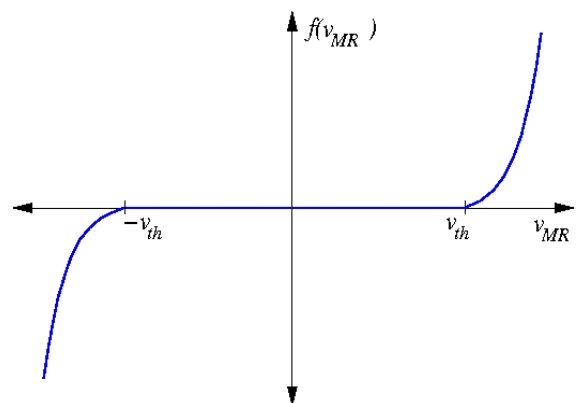
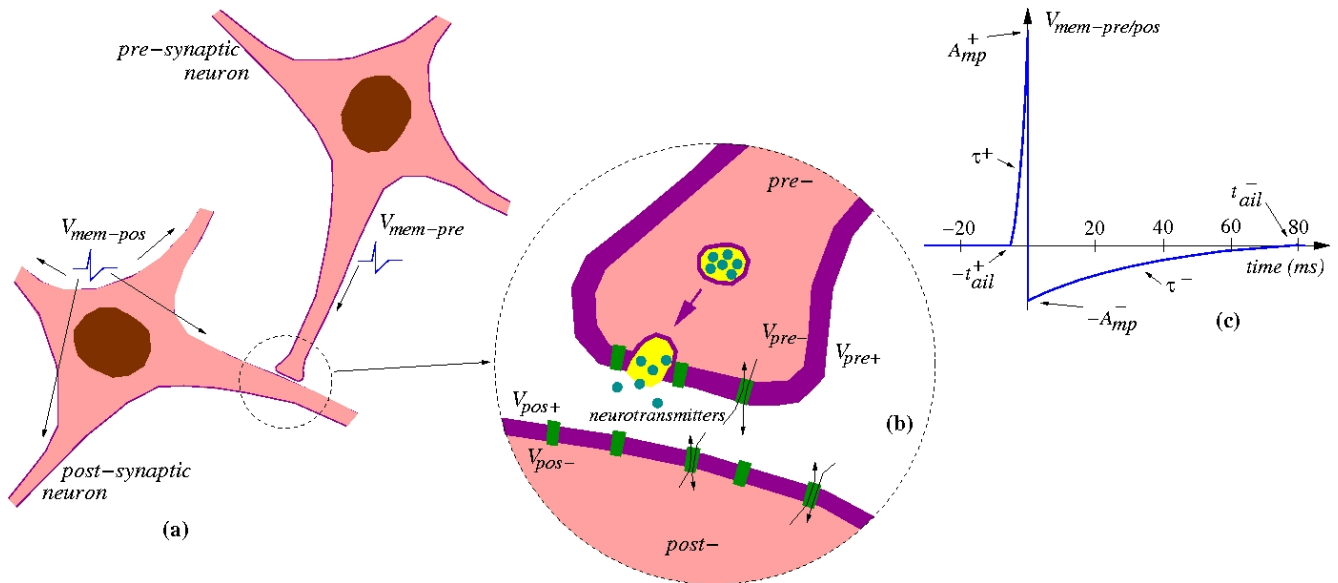


Figure 1 | Memristor characteristic function  $f(v_{MR})$ , as defined by eq. (3).

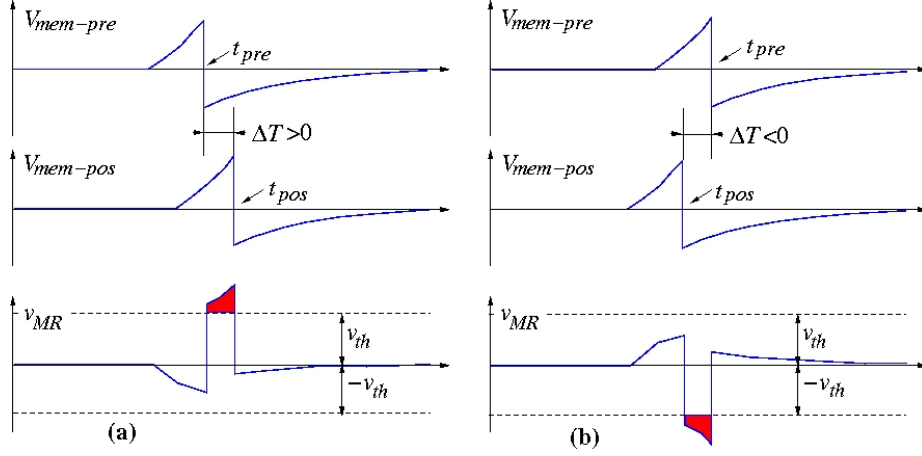


**Figure 2 | Illustration of synaptic action.** **a.** A synapse is where a pre-synaptic neuron “connects” with a post-synaptic neuron. The pre-synaptic neuron sends an action potential  $V_{mem-pre}$  travelling through one of its axons to the synapse. The cumulative effect of many pre-synaptic action potentials, generates a post-synaptic action potential at the membrane of the post-synaptic neuron, which propagates through all neuron’s terminations. **b.** Detail of synaptic junction. The cell membrane has many membrane channels of varying nature which open and close with changes of the membrane voltage. During a pre-synaptic action potential vesicles containing neurotransmitters are released into the synaptic cleft. **c.** Details of membrane voltage action potential, as by eq. (4).

voltages from the outside of the cellular membrane  $V_{pre}^+$  with respect to the inside  $V_{pre}^-$  (see inset at Fig. 2(b)). Thus  $V_{mem-pre} = V_{pre}^+ - V_{pre}^-$  and  $V_{mem-pos} = V_{pos}^+ - V_{pos}^-$ . The large membrane voltages during a spike (in the order of hundreds of  $mV$ ) cause a variety of selective molecular membrane channels to open and close allowing for many ionic and molecular substances to flow or not through the membrane. At the same time, synaptic vesicles inside the pre-synaptic cell containing “packages” of neurotransmitters fuse with the membrane in such a way that these “packages” are released into the synaptic cleft (the inter cellular space between both neurons at the synaptic junction). Neurotransmitters are collected in part by the post-synaptic membrane contributing to a change in its membrane conductivity. The cumulative effect of pre-synaptic spikes (coming from this or other pre-synaptic neurons) will eventually trigger the generation of a new spike at the post-synaptic neuron. Each synapse is characterized by a “synaptic strength” (or weight) “ $w$ ” which determines the efficacy of a pre-synaptic spike in contributing to this cumulative action at the post-synaptic neuron. This weight  $w$  could well be interpreted as the size and/or number of neurotransmitter packages released during a pre-synaptic spike. However, for our analyses, we will interpret  $w$  more generally as some structural parameter of the synapse (like the amount of one or more substances) that controls directly the efficacy of this synapse per spike (like the amount of neurotransmitter released per spike). The synaptic weight  $w$  is considered to be non-volatile and of analog nature, but changes in time as a function of the spiking activity of pre- and post-synaptic neurons. This phenomenon was originally observed and reported by Hebb in 1949, who introduced his hebbian learning postulate<sup>12</sup>: “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”. Traditionally, this has been described by computational neuroscientists and machine learning computer engineers as producing an increment in synaptic weight  $\Delta w$  proportional to the product of the mean firing rates of pre- and post-synaptic neurons. STDP is a refinement of this 1949 rule which takes into account the precise relative timing of

individual pre- and post-synaptic spikes, and not their average rates over time. In STDP the change in synaptic weight  $\Delta w$  is expressed as a function of the time difference between the post-synaptic spike at  $t_{pos}$  and the pre-synaptic spike at  $t_{pre}$ . Specifically,  $\Delta w = \xi(\Delta T)$ , with  $\Delta T = t_{pos} - t_{pre}$  (see Fig. 3). The shape of the STDP function  $\xi$  can be interpolated from experimental data from Bi and Poo as shown in Fig. 4(a)<sup>14-15</sup>. For positive  $\Delta T$  (which means, the pre-synaptic spike has a highly relevant role in producing the post-synaptic spike) there will be a potentiation of synaptic weight  $\Delta w > 0$ , which will be stronger as  $|\Delta T|$  reduces. For negative  $\Delta T$  (which means, the pre-synaptic spike is highly irrelevant for the generation of the post-synaptic spike), there will be a depression of synaptic weight  $\Delta w < 0$ , which will be stronger as  $|\Delta T|$  reduces. Bi and Poo concluded they observed an asymmetric critical window for  $\Delta T$  of size about  $40ms$  for synaptic modification to take place.

How can one relate STDP with memristance? The key is to consider carefully the shape of the electric neural spikes. The exact shape of neural spikes, usually called “action potentials” among neuroscientists, is difficult to measure precisely since the experimental setup influences strongly. Furthermore, different action potential shapes have been recorded for different types of neurons, although in general they keep a certain resemblance among them. For our discussion it suffices to assume a generic action potential shape with the following properties (see Fig. 2(c)). During spike onset, which happens during a time  $t_{ail}^+$ , membrane voltage increases exponentially until a positive peak amplitude  $A_{mp}^+$ . After this, it changes quickly to a peak negative amplitude  $-A_{mp}^-$  and returns smoothly to its resting potential during a time  $t_{ail}^-$ . A shape of the type shown in Fig. 2(c) can be expressed mathematically, for example, as



**Figure 3 | Membrane and memristor waveforms.** Pre- and post-synaptic membrane voltages for the situations of positive  $\Delta T$  (a) and negative  $\Delta T$  (b). The voltage at the memristor  $v_{MR}$  is the difference between the post-synaptic membrane voltage  $V_{mem-pos}$  and the pre-synaptic membrane voltage  $V_{mem-pre}$ . When the signs are opposite, voltages add up, and may exceed the memristor threshold voltage  $v_{th}$  of function  $f()$ . When this happens, the area in red (amplified exponentially) contributes to a change in synaptic efficacy. If  $\Delta T$  is positive synaptic efficacy is increased, while if it is negative synaptic efficacy will be decreased.

$$spk(t) = \begin{cases} A_{mp}^+ \frac{e^{t/\tau^+} - e^{-t_{ail}^+/\tau^+}}{1 - e^{-t_{ail}^+/\tau^+}} & \text{if } -t_{ail}^+ < t < 0 \\ -A_{mp}^- \frac{e^{-t/\tau^-} - e^{-t_{ail}^-/\tau^-}}{1 - e^{-t_{ail}^-/\tau^-}} & \text{if } 0 < t < t_{ail}^- \\ 0 & \text{otherwise} \end{cases} \quad (4)$$

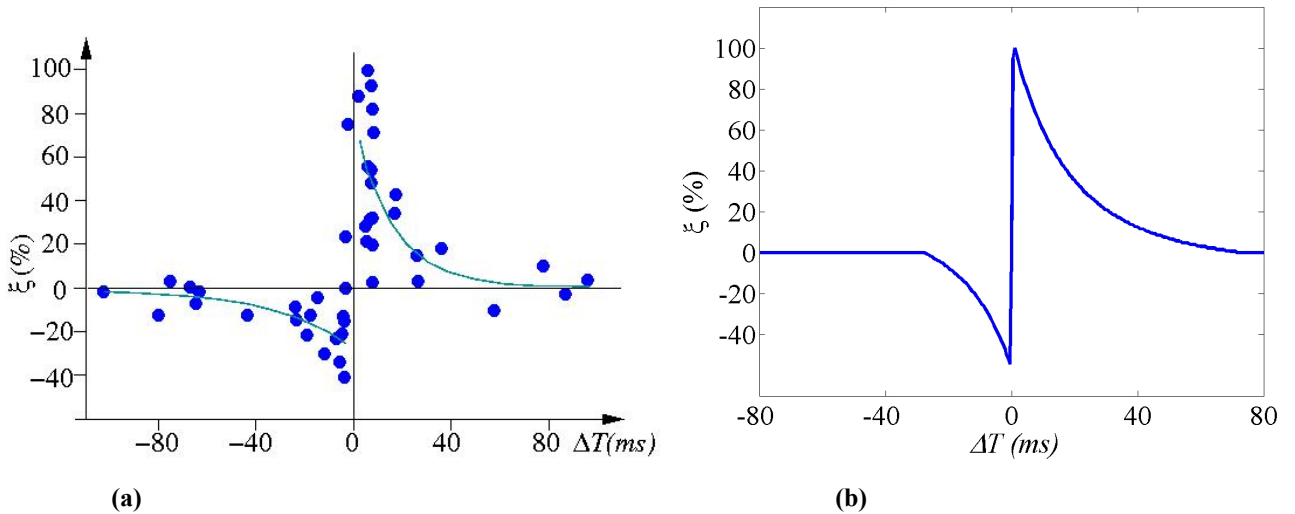
Parameters  $\tau^+$  and  $\tau^-$  control the curvature of the on-set and off-set sides of the action potential. Consider the case of pre- and post-synaptic neurons in Fig. 2 being of the same type, and thus generating the same action potential shape,  $spk(t)$  of eq. (4), when they fire. Axons and dendrites operate as transmission lines, so it is reasonable to expect some attenuation when the spikes arrive at the respective synapses. Let  $\alpha_{pre}$  be the attenuation for the pre-synaptic spike  $V_{mem-pre}(t) = \alpha_{pre} spk(t - t_{pre})$ , and  $\alpha_{pos}$  for the post-synaptic spike  $V_{mem-pos}(t) = \alpha_{pos} spk(t - t_{pos})$ . When both spikes are more or less simultaneously present at the two cell membranes of the synapse,

then channels on both membranes are open. Consequently, in principle, it makes sense to assume that during such time there could be a path for substances in the inside of one cell to move directly to the inside of the other cell and vice versa. Furthermore, let us assume now that such motion of substances obeys a memristive law similar to those described by eqs. (1-3). This means, that we would have a two-terminal memristive device between the inside sides of the two cells. More specifically, between  $V_{pos}^-$  and  $V_{pre}^-$  in Fig. 2(b). Consequently, the memristor voltage would be  $v_{MR} = V_{pre}^- - V_{pos}^-$ . On the other hand, since the outside nodes of both membranes  $V_{pos}^+$  and  $V_{pre}^+$  are very close together, both voltages will be approximately equal, yielding

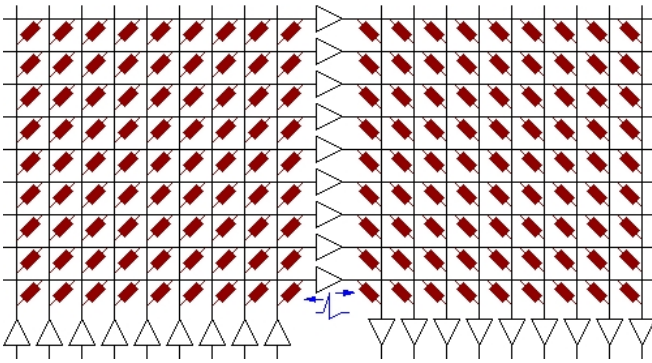
$$v_{MR}(t') \approx V_{mem-pos}(t') - V_{mem-pre}(t') = \alpha_{pos} spk(t' - t_{pos}) - \alpha_{pre} spk(t' - t_{pre}) \quad (5)$$

Doing a simple change of variables  $t = t' - t_{pos}$  and recalling that  $\Delta T = t_{pos} - t_{pre}$ , results in

$$v_{MR}(t, \Delta T) = \alpha_{pos} spk(t) - \alpha_{pre} spk(t + \Delta T) \quad (6)$$



**Figure 4 | STDP function  $\xi(\Delta T)$ .** a. Measured experimentally on biological synapses (data from Bi and Poo<sup>14,15</sup>). b. Predicted using the memristance-based model and spike shapes from eqs. (1-7).



**Figure 5 | Neuromorphic memristive computer equipped with STDP.** Neuromorphic computing structure with three layers of neurons (made in CMOS technology) and two fully-connected inter-layer meshes of memristors (made with nanowires on top of a CMOS substrate). STDP is implemented by having neurons generate action potentials with shapes similar to that of Fig. 2(c), and sending them forward without attenuation and backward with a slight attenuation.

This memristor voltage is shown in Fig. 3 for the cases of  $\Delta T$  being positive or negative. According to eq. (3), memristive action will take place only if  $v_{MR}$  exceeds threshold  $v_{th}$ , as indicated by the red shaded area in Fig. 3. As we postulated before, during this memristive action some amount of synaptic structural substance(s)  $\Delta w$  would be interchanged among both sides of the synapse. This amount of substance  $\Delta w$  will ultimately affect the synaptic strength of this synapse. If this amount of synaptic structural substance interchanged between the two synaptic terminations obeys a memristive law as in eqs. (1-3), then

$$\Delta w(\Delta T) = \int f(v_{MR}(t, \Delta T)) dt \quad (7)$$

Which is the red area of the shaded regions in Fig. 3, previously amplified exponentially through function  $f()$  of eq. (3). Positive areas (above  $v_{th}$ , when  $\Delta T > 0$ ) yield increments for  $w$  ( $\Delta w > 0$ ), while negative areas (below  $-v_{th}$ , when  $\Delta T < 0$ ) result in decrements for  $w$  ( $\Delta w < 0$ ). As  $|\Delta T|$  approaches zero, the peak of the red area in  $v_{MR}$  is higher. Since this peak is amplified exponentially, the contribution for incrementing/decrementing  $w$  will be more pronounced as  $|\Delta T|$  is reduced. The resulting function  $\Delta w(\Delta T)$ , computed using the memristor model through eq. (7) is shown in Fig. 4(b). It follows indeed the behavior of the STDP function  $\xi$  obtained by Bi and Poo from physiological experiments, which is shown in Fig. 4(a). For this numerical computation we used the following parameters:  $\alpha_{pos} = 1$ ,  $\alpha_{pre} = 0.9$ ,  $v_{th} = A_{mp}^+ = 1$ ,  $A_{mp}^- = 0.25$ ,  $v_o = 1/7$ ,  $t_{ail}^+ = 5ms$ ,  $t_{ail}^- = 75ms$ ,  $\tau^+ = 40ms$ ,  $\tau^- = 3ms$ . Making  $\alpha_{pos} \neq \alpha_{pre}$  breaks the symmetry of function  $\xi(\Delta T)$ , and making them very distinct removes one of the branches in  $\xi(\Delta T)$ .

This result shows that a memristive type of mechanism could be behind the biological STDP phenomenon. On the other hand, for a neuromorphic engineer, this result provides clear hints on how STDP learning rules could be implemented in nanotechnology based neuro-inspired computers using memristors. For example, one can fabricate very high density memristor crossbar structures<sup>1-2</sup> which connect neural layers, as shown in Fig. 5. Memristive crossbars can be fabricated using very dense nanowire fabric, while inter-synaptic neurons can be made using conventional CMOS technology underneath the memristor fabric. In order to equip the system with STDP learning, CMOS neurons could generate fully asynchronous action potentials similar to those shown in Fig. 2(c) and not only propagate them forward but also backwards with some attenuation.

Other proposals have been made recently where sequences of square voltage pulses are propagated forward and backward within precise synchronous global time windows<sup>22</sup>.

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