

# Spike-timing dependent plasticity beyond synapse – pre- and post- synaptic plastic...

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## Introduction

Long-lasting plasticity of synaptic transmission was for years considered as the favorite mechanism to account for information storage in the brain. Bidirectional long-term synaptic plasticity is indeed computationally appealing, principally because of synapse-specific changes among a large array of inputs. However, this is not the whole story and many additional electrophysiological components of neurons may undergo use-dependent long-term plasticity ( [Kim and Linden, 2007](#) ). Besides electrical synapses and glutamate transporters, voltage-gated ion channels occupy a key position in the persistent filtering of the neuronal message ( [Sjöström et al., 2008](#) ).

### **Plasticity of Intrinsic Neuronal Excitability: A Cellular Mechanism of Learning and Memory**

The postulate that modification in intrinsic excitability could underlie the formation of functional neuronal assemblies and may thus contribute to a specific memory trace has its origin in invertebrate neural systems. The first evidence for intrinsic plasticity came from the pioneering work of Alkon who showed that phototoxic learning in the marine mollusk *Hermissenda crassicornis* involved the reduction of voltage-gated A-type and  $\text{Ca}^{2+}$ -activated  $\text{K}^+$  currents in a photoreceptor ( [Alkon, 1984](#) ). Several forms of learning in *Aplysia* (sensitization and operant conditioning) were also found to be associated with persistent changes in neuronal excitability ( [Scholz and Byrne, 1987](#) ; reviewed in [Mozzachiodi and Byrne, 2010](#) ). Interest in plasticity of intrinsic excitability of mammalian neurons was for a long-time occluded by the challenge of dissecting the mechanisms underlying long-  
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term synaptic potentiation (LTP) and depression (LTD). A new interest in the study of intrinsic plasticity in mammalian brain has flourished only during the 1990s. The search for cellular excitability correlates of learning and memory in the mammalian brain has focused on neurons that are specifically active during learning. Eyeblink conditioning in the cat or the rabbit is well documented in this respect since neurons from active circuits during the conditioning exhibit *in vitro* an excitability that is significantly elevated ([Disterhoft et al., 1986](#); [Aou et al., 1992](#); [Schreurs et al., 1997](#); [Matthews et al., 2008](#)). In several cases, the after-hyperpolarization (AHP) current was found to be depressed. Other forms of learning such as spatial or olfactory learning involve the regulation of neuronal excitability ([Saar et al., 1998](#); [Oh et al., 2003](#)). These observations therefore suggest that the memory trace could not only be supported by selective changes in synaptic strength but that modifications in neuronal excitability might also contribute to the cellular substrate of the memory trace in the brain. This conclusion must be however qualified since in hippocampal and cortical neurons, increased neuronal excitability is no longer detectable 8 days after eyeblink conditioning, although learning is still established ([Moyer et al., 1996](#)). These *in vivo* studies, therefore suggest that synaptic or propagated activity during the induction phase may determine the induction of intrinsic plasticity. However, several key questions regarding the induction and expression mechanisms of intrinsic plasticity cannot be satisfactorily addressed at a cellular and molecular scale in the *in vivo* preparation and the use of *in vitro* assays must be envisaged to accurately control synaptic

activity, membrane potential as well as pharmacologically isolated membrane currents.

In central cortical neurons, excitatory postsynaptic potentials (EPSPs) measured at the axon hillock result from a tight interplay between synaptic and intrinsic voltage-gated conductances that either amplify or attenuate excitatory synaptic potentials (review in [Spruston, 2008](#)). Any modifications in this fragile equilibrium may in turn facilitate or diminish the probability that a given synaptic input triggers an action potential. Thus, regulation of ion channel activity represents a powerful means to control the principal neuronal output. For instance, tetanic stimulation of glutamatergic synaptic inputs not only induces LTP in hippocampal neurons but also persistently facilitates the generation of a postsynaptic action potential by the EPSP ([Bliss et al., 1973](#); [Andersen et al., 1980](#)), a phenomenon known as EPSP-Spike potentiation. Notably, this form of plasticity is also observed in the presence of blockers of synaptic inhibition and is bidirectional (reviewed in [Daoudal and Debanne, 2003](#)). Induction of LTD with low frequency stimulation of the Schaffer collateral-CA1 neuron input is associated with a long-lasting decrease in EPSP-Spike coupling ([Daoudal et al., 2002](#)). The three major classes of glutamate receptors (i. e., *N*-methyl- D -aspartate (NMDAR), metabotropic glutamate (mGluR) and kainate (KR) receptors) play a central role in the induction of long-lasting plasticity of intrinsic neuronal excitability. Synaptic activation of NMDAR induces a wide range of pre- and postsynaptic plasticities of neuronal excitability in hippocampal, neocortical and cerebellar neurons ([Aizenman and Linden, 2000](#); [Armano et al., 2000](#); [Ganguly et al., 2000](#); [Wang et al., 2003](#); [Li et al., 2004](#); [Fan et al., 2005](#));

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[Kim et al., 2007](#) ; [Campanac et al., 2008](#) ; [Losonczy et al., 2008](#) ). And many different voltage-gated sodium, potassium and cationic currents are targeted by the stimulation NMDAR. Activation of mGluR also produces many forms of short- and long-term regulation of intrinsic excitability in hippocampal and neocortical and modulate calcium-activated potassium, voltage-gated sodium, and hyperpolarization-activated cationic channels ( [Ireland and Abraham, 2002](#) ; [Sourdet et al., 2003](#) ; [Carlier et al., 2006](#) ; [Brager and Johnston, 2007](#) ). Synaptic activation of KR induces long-term increase in hippocampal neuron excitability that is mediated by a reduction of the slow calcium-activated potassium current /<sub>sAHP</sub> ( [Melyan et al., 2004](#) ; [Ruiz et al., 2005](#) ).

Activation of glutamate receptors is not the only means to induce long-term plasticity of intrinsic excitability in central neurons. Postsynaptic spiking may also induce persistent increases in neuronal excitability of cerebellar and cortical neurons *in vitro* and *in vivo* ( [Aizenman and Linden, 2000](#) ; [Egorov et al., 2002](#) ; [Nelson et al., 2003](#) ; [Cudmore and Turrigiano, 2004](#) ; [Paz et al., 2009](#) ). In most of these cases, the cellular conditioning consists of repeated postsynaptic bursting induced by direct injection of depolarizing current in the neuron that produce a postsynaptic elevation in Ca<sup>2+</sup> concentration, and the subsequent activation of protein kinases and the regulation of voltage-gated ion channels.

## Spike-Timing Dependent Plasticity

Spike-timing dependent plasticity (STDP) has originally been defined for excitatory synaptic transmission in hippocampal and neocortical circuits (

[Levy and Steward, 1983](#) ; [Gustafsson et al., 1987](#) ; [Debanne et al., 1994](#) , [1996](#) , [1998](#) ; [Markram et al., 1997](#) ; [Bi and Poo, 1998](#) ; [Feldman, 2000](#) ; [Sjöström et al., 2001](#) ; reviewed in [Abbott and Nelson, 2000](#) ; [Dan and Poo, 2006](#) ). Synaptic efficacy is persistently and bi-directionally altered depending on the temporal interaction between afferent synaptic activity (EPSP) and postsynaptic spiking (the action potential). In most excitatory synapses positively correlated pre- and postsynaptic activity (EPSP before action potential) leads to synaptic reinforcement whereas negative correlation (action potential before EPSP) induces LTD. Thus, as initially postulated by [Hebb \(1949\)](#) , synapses that repeatedly contribute to the postsynaptic discharge are reinforced whereas synapses that repeatedly fails to participate to the neuronal activity are depressed ( [Stent, 1973](#) ). Since the early 1990's, this rule has been verified in a large number of glutamatergic synapses impinging on glutamate neurons ( [Gustafsson et al., 1987](#) ; [Debanne et al., 1994](#) , [1996](#) , [1998](#) ; [Markram et al., 1997](#) ; [Bi and Poo, 1998](#) ; [Tzounopoulos et al., 2004](#) ; but see [Egger et al., 1999](#) ). However, when the postsynaptic neuron is inhibitory this rule is generally inverted ( [Bell et al., 1997](#) ; [Wang et al., 2000](#) ; [Tzounopoulos et al., 2004](#) ; [Fino et al., 2005](#) ; but see [Pawlak and Kerr, 2008](#) ). A major consequence of STDP is that synaptic inputs compete for postsynaptic spike-timing and short-latency inputs win over long-latency synapses ( [Song et al., 2000](#) ). STDP is also observed in many different *in vivo* preparations in which synaptic activity can be monitored in response to electrical or sensory stimulation ( [Zhang et al., 1998](#) ; [Jacob et al., 2007](#) ). It may account for activity-dependent plasticity of orientation maps in the cat striate cortex (

[Schuett et al., 2001](#)), sensory adaptation ([Yao and Dan, 2001](#)) or shaping of hippocampal place-fields in the rat ([Mehta et al., 2000](#)). STDP rules have also been verified in the human brain with peripheral and transcranial stimulation ([Wolters et al., 2003](#)). However, when functional plasticity is monitored *in vivo*, it is virtually impossible to precisely identify the neuronal substrate and the underlying mechanisms of the observed changes. They may, indeed, equally result from modification of either synaptic strength or intrinsic excitability, or both. Therefore, it is of great importance to know whether protocols based on temporal interactions also produce long-lasting modifications of neuronal excitability in hippocampal and cortical neurons, and the spatial and temporal extent of these modifications.

## Presynaptic Changes in Neuronal Excitability and STDP

Changes in neuronal excitability associated with synaptic plasticity induced by STDP protocols have first been identified at the presynaptic side. Using paired-recordings from synaptically coupled hippocampal neurons, [Ganguly et al. \(2000\)](#) found that correlated pre- and postsynaptic spiking not only induces LTP of synaptic transmission but also increases the excitability of the presynaptic neuron. This presynaptic plasticity of neuronal excitability is rapidly expressed (<2 min) and is long-lasting (> 30 min). Its induction requires activation of NMDA receptors (NMDAR), postsynaptic  $\text{Ca}^{2+}$  elevation and activation of protein kinases C (PKC). The expression mechanisms have been studied at the soma of the presynaptic neuron and involve a shift in the activation curve of the  $\text{Na}^+$  current towards hyperpolarizing values that may account for the lower spike threshold ([Ganguly et al., 2000](#)). In addition, the sodium current exhibits an improved

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recovery from inactivation following correlated pre- and postsynaptic activity.

Mirror effects have been observed presynaptically when LTD is induced by negative correlation in hippocampal and neocortical pyramidal neurons ( [Li et al., 2004](#) ). The excitability of the presynaptic cell is persistently reduced following induction of LTD. This plasticity requires postsynaptic calcium elevation and presynaptic PKA- and PKC-dependent modification of slow-activating K<sup>+</sup> channels. The reduction in excitability is reversible if LTP is induced with positive correlation ( [Li et al., 2004](#) ). It is important to note here that these presynaptic changes in excitability were also observed in pairs of L4 spiny neurons from brain slices, suggesting that this form of intrinsic plasticity might also be present *in vivo* .

The functional consequences of long-lasting plasticity of presynaptic excitability might be of great importance for the dynamics of the neural circuits. For instance, the presynaptic increase in neuronal excitability following correlated spiking activity may facilitate the induction of bursting-like behavior and will enhance the reliability of signal transmission by creating privileged activity pathways in the brain. In addition, enhancement of presynaptic neuronal excitability may affect the plasticity of upstream synapses made onto the presynaptic cell by facilitating the initiation of back-propagated action potentials ( [Tao et al., 2000](#) ). Indeed, retrograde spread of LTP and LTD from developing retinotectal synapses to upstream synapses made by bipolar cells on retinal ganglion cells in the retina has been observed *in vivo* , through cytoplasmic retrograde signaling with the order of

minutes after the induction of LTP/LTD ([Du et al., 2009](#)). Presynaptic modifications of neuronal excitability and efficacy of upstream synapses may be attributed to the actions of common retrograde signals and serve synergistic functions in neuronal circuit plasticity.

## **Postsynaptic Changes in Neuronal Excitability and STDP**

Neuronal excitability is also affected on the postsynaptic side when long-term synaptic plasticity is induced with STDP protocols. At least two neuronal functions, namely EPSP summation and EPSP-spike coupling, are modulated after the induction of spike-timing dependent LTP/LTD, through changes in postsynaptic voltage-gated channels.

In hippocampal CA1 pyramidal neurons, summation of EPSPs is determined by voltage-gated conductances that amplify or attenuate synaptic potentials. The property of EPSP summation in these neurons is strongly modulated following induction of LTP or LTD ([Wang et al., 2003](#); [Xu et al., 2006](#)). A gain in EPSP summation is consistently observed when LTP is induced with positively correlated activity whereas a loss of summation is associated with LTD induced by negative correlation. The activity-dependent increase in EPSP summation requires activation of NMDA receptors, PKC and Ca<sup>2+</sup>/calmodulin-dependent protein kinase II (CaMKII), but is independent of GABA A receptor-mediated inhibition ([Wang et al., 2003](#)). Interestingly, blockers of hyperpolarization-activated cationic (h) channels prevent the facilitation in EPSP summation, suggesting that the facilitation results from the down-regulation of h-channel activity in the dendrites.

EPSP-spike coupling is also profoundly affected in CA1 pyramidal neurons when long-term synaptic plasticity is induced with STDP protocols ( [Campanac and Debanne, 2008](#) ). Positive correlation between pre- and postsynaptic activity not only induces LTP but also facilitates EPSP-spike coupling. Conversely, LTD induced by negative correlation is associated with EPSP-spike depression. Both EPSP-spike potentiation and depression require activation of NMDA receptors but are independent of GABA<sub>A</sub> receptor dependent inhibition ( [Campanac and Debanne, 2008](#) ). The amplitude-slope relation of the EPSP was facilitated after LTP but depressed after LTD, indicating that mechanisms of EPSP amplification are regulated (see also [Wang et al., 2003](#) ). Most importantly, the regulation of h-channel activity may constitute a major mechanism of expression. Apparent input resistance measured in the cell body is increased following induction of LTP induced by correlated activity or by high frequency stimulation ( [Campanac et al., 2008](#) ). This change is more pronounced when apical dendrites are recorded. In addition, the local decrease of the dendritic h-conductance with the use of dynamic-clamp techniques mimics EPSP-spike potentiation ( [Campanac et al., 2008](#) ). However, the regulation of A-type K<sup>+</sup> current in the dendrite ( [Frick et al., 2004](#) ; [Kim et al., 2007](#) ) or the fast transient Na<sup>+</sup> current in the cell body ( [Xu et al., 2005](#) ) induced by positive correlation between synaptic activity and postsynaptic spiking may also facilitate EPSP-spike coupling in CA1 pyramidal neurons.

## Spatial and Temporal Selectivity of STDP-Induced Intrinsic Plasticity

### Spatial Selectivity

The storage of information through the regulation of synaptic strength in models of memory is particularly attractive because of the property of input specificity. Given the large number of synapses per neuron (> 10000) and the huge number of neurons found in the brain ( $10^{11}$ ), a memory storage system that uses input-specific changes in synaptic transmission has a potentially enormous storage capacity. Although chemical synapses represent the major functional interface between neurons, following dendritic integration neuronal information is conveyed by action potentials. Thus, the activity-dependent regulation of ion channels located near the spike generation zone (the axon initial segment or the cell body) would change the throughput of all synapses that the neuron receives ([Daoudal and Debanne, 2003](#); [Zhang and Linden, 2003](#); [Mozzachiodi and Byrne, 2010](#)), possibly leading to a loss in the information stored at specific synapses. In this case, the specificity might be observed at the cellular level. Such global changes in neuronal excitability have been observed following brief episodes of synaptic ([Aizenman and Linden, 2000](#); [Sourdet et al., 2003](#)) or spiking activity ([Cudmore and Turrigiano, 2004](#); [Paz et al., 2009](#)) and in the presynaptic neuron when LTP or LTD is induced in hippocampal cells with spike-timing dependent protocols ([Ganguly et al., 2000](#); [Li et al., 2004](#)). In these studies, neuronal excitability was assessed only in the cell body. Whether and to what extent intrinsic excitability changes in axonal and dendritic membranes remains unclear. Global changes have also been reported on the postsynaptic side when LTP is induced with correlated <https://assignbuster.com/spike-timing-dependent-plasticity-beyond-synapse-pre-and-post-synaptic-plasticity-of-intrinsic-neuronal-excitability/>

activity in CA1 pyramidal neurons ([Xu et al., 2005](#)). However, several lines of evidence indicate that postsynaptic changes in intrinsic excitability induced by spike-timing dependent protocols are restricted to a localized region of the dendrites. Enhanced EPSP summation associated with synaptic potentiation is dependent on the position of the synaptic inputs ([Wang et al., 2003](#)). A-type K<sup>+</sup> channels are regulated in the dendrites of CA1 pyramidal neurons after induction of LTP ([Frick et al., 2004](#)). Potentiation and depression of dendritic integration observed in parallel with induction of spike-timing dependent synaptic plasticity are input specific ([Campanac and Debanne, 2008](#)). Finally, dendritic h-channel activity is locally reduced following induction of LTP ([Campanac et al., 2008](#)). Thus, intrinsic excitability changes associated with STDP-related protocols are localized in the dendrites of CA1 pyramidal neurons and may thus preserve the input specificity property for high-capacity information storage.

## Temporal Selectivity

The temporal windows for synaptic potentiation and depression induced by correlated activity are generally not wider than a few tens of millisecond ([Markram et al., 1997](#); [Bi and Poo, 1998](#); [Debanne et al., 1998](#); [Feldman, 2000](#)). They are usually precisely defined (at the millisecond scale) and depend on both the number of postsynaptic spikes ([Debanne et al., 1994](#); [Sourdet and Debanne, 1999](#)) and dendritic location of the synaptic input ([Froemke et al., 2005](#)). Similar plasticity windows are observed for intrinsic plasticity. Presynaptic changes in neuronal excitability follow precisely the STDP rule ([Li et al., 2004](#)). At the postsynaptic side, EPSP-spike plasticity also obeys the STDP learning rule ([Campanac and Debanne, 2008](#)). In these

two examples, no changes in excitability at the pre- or postsynaptic side are observed out of the LTP and LTD time windows (i. e., for long positive or long negative intervals), suggesting common induction mechanisms for plasticity in synaptic efficacy and intrinsic excitability.

## Functional Synergy Between Synaptic and Intrinsic Plasticity Induced by STDP

A functional synergy has been demonstrated for synaptic and intrinsic plasticity in central neurons at the pre- and postsynaptic side. LTP is generally associated with an increase in neuronal excitability ([Armano et al., 2000](#); [Ganguly et al., 2000](#); [Daoudal et al., 2002](#); [Wang et al., 2003](#); [Xu et al., 2005](#); [Campanac and Debanne, 2008](#); [Campanac et al., 2008](#)) whereas synaptic depression is usually accompanied by a reduced intrinsic excitability ([Daoudal et al., 2002](#); [Wang et al., 2003](#); [Campanac and Debanne, 2008](#)). Thus, plasticity in intrinsic excitability cannot be simply considered as an additional level of plasticity that makes understanding brain storage mechanisms more complex but rather, it can be incorporated into a general framework in which synaptic and intrinsic plasticity interact coherently ([Daoudal and Debanne, 2003](#)). This functional synergy insures significant changes in the neuronal output when synaptic plasticity is induced.

Functional synergy between synaptic and intrinsic plasticities might, however, be broken if the magnitude of synaptic plasticity is excessive and comes out of the physiological range ([Debanne et al., 2003](#)). This form of compensatory plasticity has been first studied theoretically in multi-compartment model of CA1 pyramidal neurons where intrinsic plasticity <https://assignbuster.com/spike-timing-dependent-plasticity-beyond-synapse-pre-and-post-synaptic-plasticity-of-intrinsic-neuronal-excitability/>

restored optimal firing range after synaptic potentiation ( [Siegel et al., 1994](#) ). In biological CA1 pyramidal neurons, synaptic potentiation of very large amplitude (~300% of the control EPSP) is accompanied by a *decrease* in excitability of CA1 pyramidal neurons ( [Fan et al., 2005](#) ). Conversely, nearly maximal synaptic depression (~50%) is associated with an *increase* in excitability ( [Brager and Johnston, 2007](#) ). Both changes globally affect neuronal excitability, involve the regulation of h-channel activity, and are aimed to stabilize the overall neuronal activity by preventing excessive synaptic excitation. Therefore, these regulations are homeostatic in nature and compensate for excessive increases or decreases in synaptic excitation ( [Turrigiano and Nelson, 2004](#) ).

## Concluding Remarks

Temporal interactions of pre- and postsynaptic spiking in neural circuits bidirectionally regulate not only synaptic strength but also intrinsic excitability of pyramidal cells at both pre- and postsynaptic sides. At the postsynaptic side this plasticity is largely localized within the dendrites – involving the regulation of A-type K<sup>+</sup> and h-type cationic channels – and respects the input specificity of synaptic modifications, thus preserving high capacity of information storage. However, intrinsic plasticity is probably not synapse specific but rather may alter the excitability of dendritic branches over a few tens of micrometers ( [Losonczy et al., 2008](#) ). Additional experimental investigations will be necessary to precisely determine the spatial extent of intrinsic plasticity in branched neurons.

Synaptic activation of glutamate receptors is critical in the induction of long-term plasticity of intrinsic excitability in central neurons but one cannot exclude the contribution of other factors. Indeed, in most of the experiments reported so far (except the studies of [Li et al., 2004](#); [Losonczy et al., 2008](#)), intrinsic plasticity is induced by stimulation of a large bundle of glutamatergic inputs and one cannot exclude stimulation of neuromodulators containing varicosities. In fact, neuromodulators are potent regulators of back-propagating action potentials ([Hoffman and Johnston, 1999](#)), ion channel activity ([Cantrell and Catterall, 2001](#)) and spike-timing dependent synaptic plasticity ([Lin et al., 2003](#); [Seol et al., 2007](#); [Zhang et al., 2009](#)). There are good reasons to believe that neuromodulators may also modulate induction of spike-timing dependent intrinsic plasticity. In addition, the gliotransmitter D-serine that is thought to be released by astrocytes upon neuronal stimulation ([Mothet et al., 2005](#); review in [Hamilton and Attwell, 2010](#)) play a critical role in the induction of long-term synaptic plasticity ([Henneberger et al., 2010](#)). One cannot exclude that such new actors in interneuronal signaling may also play a major role in the induction of spike-timing dependent intrinsic plasticity.

Plasticity of intrinsic excitability is synergistic to synaptic plasticity in modulating neuronal output, thus enhancing the functional significance of synaptic modifications. However, compensatory mechanisms acting both at intrinsic and synaptic currents concur to maintain neuronal activity within physiological bounds ([Siegel et al., 1994](#); [Rumsay and Abbott, 2004](#); [Rabinowitch and Segev, 2006](#)). The synergy between synaptic and intrinsic plasticity might be broken outside this physiological range ([Fan et al., 2005](#));  
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[Campanac et al., 2008](#)). While learning rules for STDP of synaptic efficacy and intrinsic excitability are coordinated in pyramidal neurons, additional work is still needed to define the corresponding properties in GABAergic interneurons ([Kullmann and Lamsa, 2007](#)).

## Conflict of Interest Statement

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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