Symposium

Behavioral Timescale Synaptic Plasticity: A Burst in the Field of Learning and Memory

Antoine D. Madar,[1] Aaron D. Milstein,[2] Thomas J. O'Dell,[3] Anant Jain,[4] Claudia Clopath,[5] and Mark E. J. Sheffield[1]

[1]Department of Neurobiology, Neuroscience Institute, University of Chicago, Chicago, Illinois 60637, [2]Department of Neuroscience and Cell Biology and Center for Advanced Biotechnology and Medicine, Rutgers Heath, Rutgers, The State University of New Jersey, Piscataway, New Jersey 08854, [3]Department of Physiology, David Geffen School of Medicine, and Integrative Center for Learning and Memory, Brain Research Institute, University of California, Los Angeles, Los Angeles, California 90095, [4]Centre for High Impact Neuroscience and Translational Applications (CHINTA), TCG CREST, Kolkata, West Bengal 700091, India, and [5]Department of Bioengineering, Imperial College London, London SW72AZ, United Kingdom

Hebbian synaptic plasticity is currently the main framework to relate neuronal activity, network structure, and learning and memory. However, recent experimental and computational modeling studies have revealed a new form of synaptic plasticity termed behavioral timescale synaptic plasticity (BTSP). It is triggered by dendritic plateau potentials associated with somatic burst firing, causes large changes in synaptic strength in a single shot, and operates on the timescale of seconds. Here we review the recent advances in our understanding of the circuit, cellular, and molecular mechanisms of BTSP, its prevalence in the brain, its role in shaping neuronal representations, and the emerging ideas regarding its contribution to different forms of learning.

Introduction

Donald Hebb famously postulated that associative forms of learning, such as pavlovian conditioning, could arise from changes in synaptic strength triggered by coincident pre- and postsynaptic activity (Hebb, 1949). The Hebbian requirement for coactivity is best exemplified by classic spike timing-dependent plasticity (STDP), where causal pairings of pre and postsynaptic action potentials (pre shortly before post) yield long-term potentiation (LTP), whereas acausal pairings (post shortly before pre) produce long-term depression (LTD; Fig. 1*A*; Bi and Poo, 2001; Feldman, 2012). Mounting evidence indicates that Hebbian plasticity is indeed an important mechanism of learning (Clopath et al., 2010; Gilson et al., 2011; D'Albis et al., 2015; Dringenberg, 2020; Magee and Grienberger, 2020). Hebbian learning rules are also widely used in computational models, endowing neuronal networks with self-organizing properties that enable assembly formation, information storage, and recall (Hopfield, 1982; Litwin-Kumar and Doiron, 2014; Ocker et al., 2015; Chenkov et al., 2017; Acker et al., 2019; Boscaglia et al., 2023; Debanne and Inglebert, 2023).

However, some features of Hebbian plasticity make it inappropriate for many forms of learning (Gallistel and Matzel, 2013; Magee and Grienberger, 2020). For example, STDP rules operate on the timescale of milliseconds and thus cannot explain associative learning of events separated by seconds or more, unless additional processes are involved (Frémaux and Gerstner, 2015; Fuchsberger et al., 2022). Moreover, STDP yields robust and significant LTP only after many pairings of input and output spikes; STDP is thus too weak to support one- or even few-shot learning. Finally, because of its correlative nature, Hebbian plasticity is intrinsically an unsupervised process without an explicit mechanism for solving the credit assignment problem, i.e., for identifying which synapses should be modified for learning (Richards and Lillicrap, 2019). As such, it is ill-suited in situations of supervised learning (Sutton and Barto, 1981), when feedback is provided to reach specific learning objectives (Frémaux and Gerstner, 2015; Magee and Grienberger, 2020). In addition to these theoretical limitations, canonical STDP is an incomplete framework that does not account for many determinants of plasticity (Lisman and Spruston, 2005; Wittenberg and Wang, 2006; Abraham, 2008; Shouval et al., 2010; Zenke and Gerstner, 2017; Brzosko et al., 2019; Inglebert et al., 2020) and has little influence on neuronal dynamics in vivo (Graupner et al., 2016; Madar et al., 2025).

A different kind of plasticity called behavioral timescale synaptic plasticity (BTSP) has recently been uncovered in area CA1 of the hippocampus (Bittner et al., 2017; Milstein et al., 2021) and has properties that appear to solve many of the aforementioned limitations of Hebbian plasticity (Magee and Grienberger, 2020). It differs from STDP in three major ways. First, it is triggered by occasional dendritic plateau potentials associated with a burst of firing in the soma. Bursts are sparse but reliable elements of the neural code, distinct from isolated action potentials (Lisman, 1997; Zeldenrust et al., 2018), which makes burst-dependent forms of plasticity potentially useful for credit assignment (Bouvier et al., 2018; Payeur et al., 2021). Second, BTSP operates on the timescale of seconds rather than milliseconds and can therefore support associative learning over temporal delays relevant to behavior. Third, it leads to large

Received May 28, 2025; revised Aug. 8, 2025; accepted Aug. 11, 2025.

Author contributions: A.D. Madar, A.D. Milstein, T.J.O., A.J., C.C., and M.E.J.S. wrote the paper.

This work was supported by the National Institute of Neurological Disorders and Stroke of the NIH (M.E.J.S., R01MH136274), the National Institute of Mental Health of the NIH (M.E.J.S., RF1NS127123; A.D. Madar, F32MH126643; A.D. Milstein, R01MH135576), and ANRF India (A.J., ANRF/ECRG/2024/003420/LS).

The authors declare no competing financial interests.

Correspondence should be addressed to Antoine D. Madar at madar@uchicago.edu.

https://doi.org/10.1523/JNEUROSCI.1332-25.2025

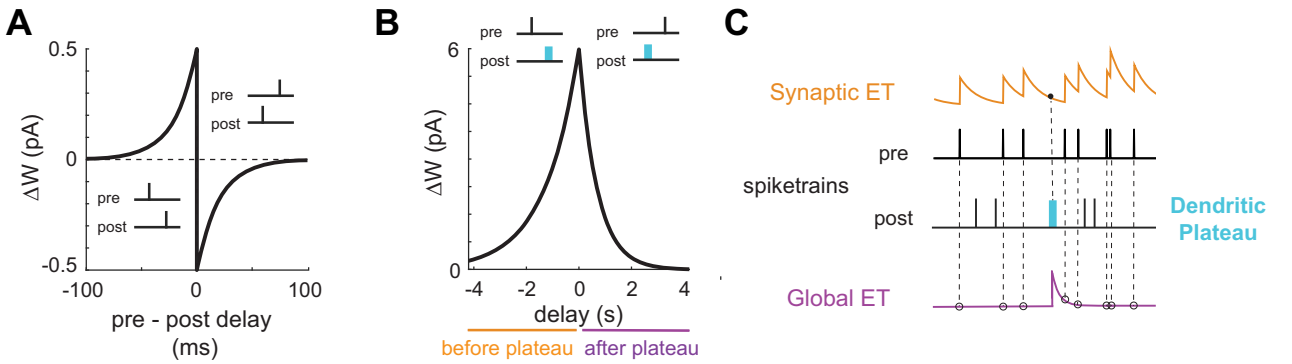

Figure 1. STDP and BTSP. ***A***, Canonical Hebbian STDP rule. ΔW is the synaptic weight change after a single pair of pre- and postsynaptic spikes. Causal pairings yield LTP, acausal pairings yield LTD. ***B***, BTSP rule revealed by in vitro experiments in CA1 from Bittner et al. (2017) using Schaffer collateral stimulations before or after somatic induction of a dendritic plateau. ***A***, ***B***, Amplitudes are indicative (based on estimates from simulations in Madar et al., 2025), to illustrate a major difference between STDP and BTSP. ***C***, Implementation of the BTSP rule shown in ***B*** with eligibility traces. Presynaptic spikes are converted into a local signal at each synapse (orange), whereas the dendritic plateau is converted into a global signal (purple). The combination of both signals determines synaptic weight changes at each synapse. Adapted from Madar et al. (2025).

changes in synaptic strength, enabling fast remodeling of neuronal representations that may support one-shot learning. This review aims to highlight the recent burst of research on BTSP, from the discovery of the phenomenon to our current understanding of its mechanisms of induction and expression, and its functional importance in learning and memory.

Somatic Bursts, Active Dendrites, and the Discovery of BTSP

From the first reports on STDP, it was noted that delays between input and output spikes were not the only determinant of the sign and strength of plasticity and that bursts of spikes were important (Markram et al., 1997). STDP protocols using pre–post pairs of single spikes can induce LTP only at high repetition rates but have little effect when pairings are repeated at low frequency, sometimes even yielding LTD (Wittenberg and Wang, 2006). However, robust LTP is rescued when presynaptic single action potentials are paired with postsynaptic bursts of a few spikes induced by somatic current injections (Debanne et al., 1996; Pike et al., 1999; Kampa et al., 2006; Wittenberg and Wang, 2006; Carlisle et al., 2008; Buchanan and Mellor, 2010). Similarly, STDP protocols have no effect when using physiological levels of extracellular calcium, but plasticity can be induced when using postsynaptic bursts instead of single action potentials (Inglebert et al., 2020). Postsynaptic bursts allow larger weight changes, necessitating fewer pre–post pairings to induce LTP than single-spike STDP protocols (Cepeda-Prado et al., 2022). These effects are generally interpreted as a necessity for dendrites to be sufficiently depolarized during incoming synaptic activity in order to remove the magnesium-block of NMDA receptors (NMDARs) and allow sufficient calcium influx (Wittenberg and Wang, 2006; Graupner and Brunel, 2012). This depolarization can come from backpropagating action potentials (bAPs; Magee and Johnston, 1997), but other kinds of dendritic regenerative events can also be involved and influence synaptic plasticity (Stuart and Spruston, 2015; Bono and Clopath, 2017; Ebner et al., 2019). For instance, it was shown in Layer 5 pyramidal neurons of the neocortex that a three-spike burst can rescue STDP only if bAPs evoke dendritic calcium spikes (Kampa et al., 2006; Letzkus et al., 2006). In CA1 pyramidal cells, bAPs from a somatic burst are not necessary for LTP to occur, as long as sufficiently strong synaptic inputs trigger local dendritic spikes (Golding et al., 2002; Remy and Spruston, 2007; Gambino et al., 2014; Kim et al., 2015). A single dendritic event can even induce robust LTP if it is sufficiently large to cause a burst-inducing depolarization in the soma (Remy and Spruston, 2007).

Large and long-lasting dendritic depolarizations causing somatic burst firing can be induced by combining stimulations of the distal and perisomatic compartments of pyramidal cells (Larkum et al., 1999; Magee and Carruth, 1999). These calcium-mediated dendritic events, lasting at least 50 ms, are often called "plateau potentials" (Antic et al., 2010 ; Larkum et al., 2022) and the resulting somatic burst riding on a large step-like depolarization is called a "complex spike" (CS; Fig. 2*A*,*B*). In CA1 pyramidal cells, CS-associated dendritic plateaus, evoked by a few repetitions of correlated distal and proximal inputs (Fig. 3*A*), lead to strong LTP of the distal pathway (Takahashi and Magee, 2009). However, because these seminal experiments were made in slices with GABAergic transmission blocked and because the induction of dendritic plateaus is tightly controlled by inhibitory circuits (Milstein et al., 2015), it was not immediately clear whether plateau potentials naturally occur in the brain. In vivo recordings confirmed that CSs occasionally occur in CA1 place cells (Epsztein et al., 2011; Bittner et al., 2015; Cohen et al., 2017) and that they arise from large multidendritic calcium events (Grienberger et al., 2014) that shape the spatial modulation of somatic activity (i.e., place fields; Sheffield and Dombeck, 2015).

The crucial finding that led to the discovery of BTSP by the Magee lab is that, in mice running on a treadmill with sensory cues, intracellularly recorded CSs sometimes spontaneously occurred in silent neurons and were always followed by the emergence of a place field on subsequent laps (Bittner et al., 2015). Moreover, somatically induced CS-like depolarizations, repeated at the same location on a few laps, were sufficient to cause previously silent cells to develop a place field near that spot. The firing activity defining the new place field was riding on a long and asymmetric subthreshold ramp of the membrane potential, which indicated potentiation of spatially informative inputs that were active seconds before and after the CS. Computational models showed that STDP could not explain this phenomenon but an asymmetric potentiation rule triggered on the CS and operating over several seconds could (Fig. 1*B*; Bittner et al., 2017). This rule predicts that the width of CS-induced place fields must be correlated with the animal speed at the time of the CS (at faster speeds, inputs coding for farther

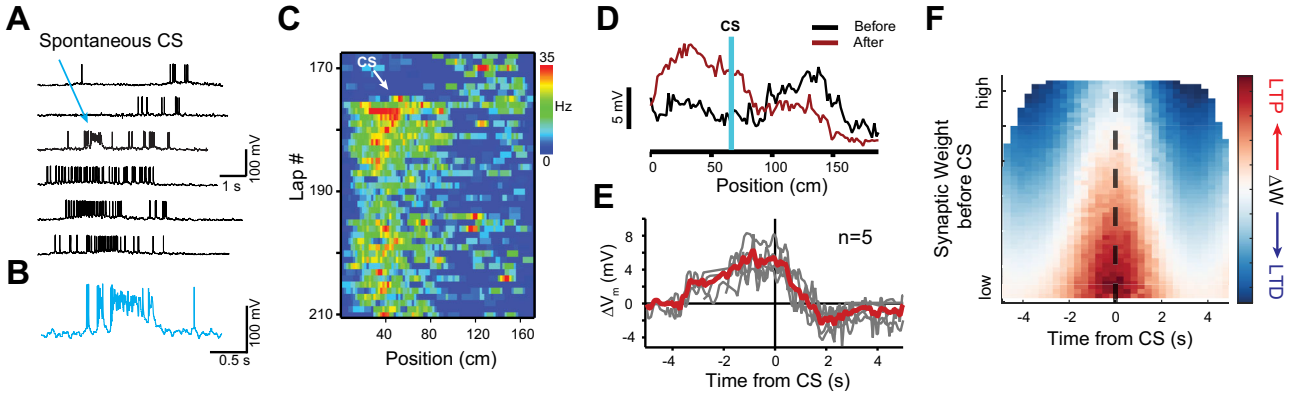

Figure 2. BTSP as a weight-dependent bidirectional rule. ***A–E***, Intracellular current-clamp recordings of a place cell in CA1. A spontaneous dendritic plateau occurred outside of the place field, causing a somatic CS, depotentiation of the place field, and emergence of a new one. ***B***, Zoom on the somatic CS. ***C***, A rate map showing the translocation of the place field after the plateau. ***D***, Averaged subthreshold membrane potential (V_m) before and after the plateau. V_m ramps reflect synaptic weights. ***E***, Difference in V_m before and after a spontaneous plateau, reflecting changes in synaptic weight, in five pyramidal neurons with pre-existing place fields. ***F***, Weight-dependent bidirectional BTSP rule inferred from multiple intracellular recordings with spontaneously or artificially induced plateaus. ΔW is the change in synaptic weight. ***A–E***, adapted from Milstein et al. (2021) and (***F***) from Madar et al. (2025).

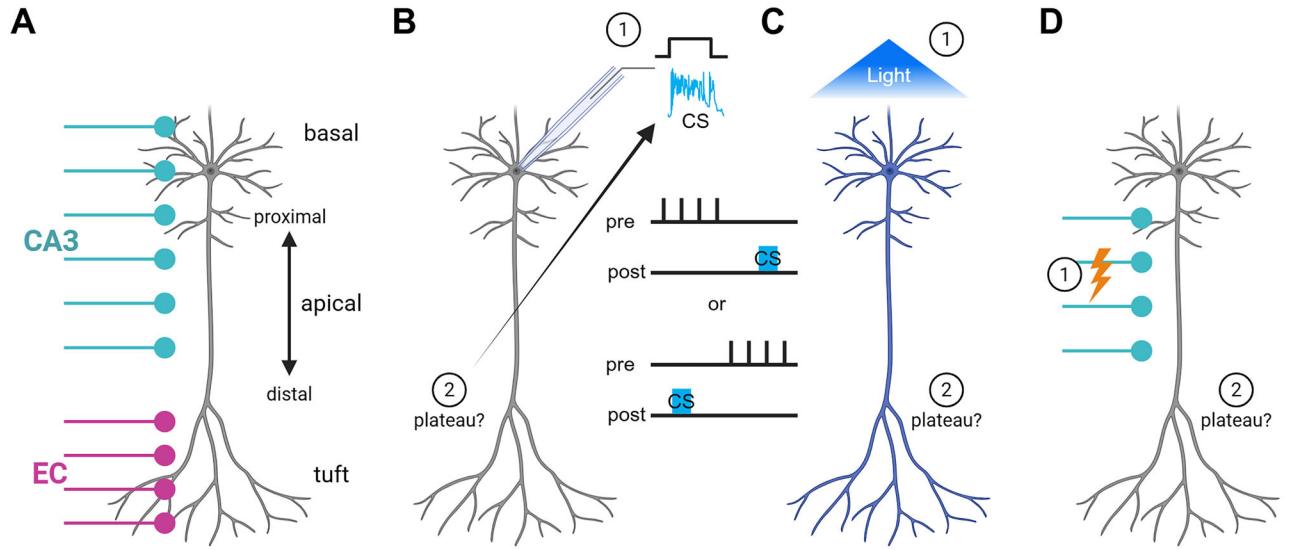

Figure 3. Artificial BTSP induction. ***A***, CA1 pyramidal neuron, its main inputs, and the associated dendritic compartments. Coincident stimulation of the CA3 and EC inputs can trigger a dendritic plateau (Takahashi and Magee, 2009). ***B***, Whole-cell patch–clamp: repeated and large somatic step depolarizations (black) from the intracellular electrode can trigger a CS (cyan). Synaptic plasticity is assessed by comparing, before and after induction, EPSPs evoked by stimulations of CA3 inputs or subthreshold Vm reflecting spontaneous inputs (Bittner et al., 2017; Jain et al., 2024). ***C***, Optogenetic step stimulations of a single neuron expressing a light-sensitive ion channel. ***D***, Theta-paced electrical stimulations of CA3 inputs can trigger a CS and induce BTSP without somatic manipulation (O'Dell, 2022). Created in BioRender. Madar et al. (2025) https://BioRender.com/rsb0xp8.

places fall in the potentiating window around the CS), and this was verified experimentally. Finally, the BTSP rule inferred from in vivo recordings was confirmed in slices, showing that somatically induced CS-like depolarizations can strongly potentiate CA3 inputs over long delays (>1 s), with causal pairings (EPSPs before CS) inducing LTP over longer delays than acausal pairings (CS before EPSPs; Bittner et al., 2017). Importantly, additional in vivo intracellular recordings in cells with pre-existing place fields showed that (1) in-field CSs do not result in much change, indicating that LTP saturates after just a few BTSP induction events and (2) CSs triggered away from the current place field not only produce a new place field but can also depress the initial one (Fig. 2*A–E*; Milstein et al., 2021). This suggests that BTSP is bidirectional, with the amount of LTP or LTD apparently depending on both CS timing and the synaptic weight before CS induction (Fig. 2*F*).

How to Study BTSP?

BTSP was initially deduced from serendipitous observations of CSs in vivo. BTSP was thus defined as a form of plasticity triggered by a single or few repetitions of CS-associated dendritic plateaus and operating over seconds. To study its phenomenology, different strategies have been used, mostly in area CA1 of the hippocampus (Fig. 3).

The main approach, introduced by the Magee lab (Bittner et al., 2017), is to artificially induce BTSP with a long-lasting high–amplitude depolarization of the soma (typically a 300 ms 600 pA current injection) triggering a somatic CS (which is assumed to reflect a dendritic plateau), preceded or followed by synaptic activity within a few seconds time window (Fig. 3*B*). This can be done in vitro (Bittner et al., 2017; Caya-Bissonnette et al., 2023; Jain et al., 2024) or in vivo, where repeated and sufficiently strong somatic depolarizations in

behaving rodents can, relatively reliably, induce place fields in the hippocampus, a process shown to rely on the long timescale of BTSP. In vivo somatic step depolarization can be achieved using whole-cell patch–clamp current injections (Bittner et al., 2017; Milstein et al., 2021; Zhao et al., 2022; Li et al., 2024) or extracellular optogenetic stimulations of single pyramidal cells (Fig. 3*C*; Diamantaki et al., 2018; Geiller et al., 2022; O'Hare et al., 2022; Fan et al., 2023; Gonzalez et al., 2025). Note that synchronous activation of many pyramidal cells does not work due to the recruitment of strong feedback inhibition (McKenzie et al., 2021; Rolotti et al., 2022).

The combination of CS induction with synaptic activity is necessary for BTSP, which is synapse-specific: isolated CSs are not sufficient to potentiate inactive synapses (Bittner et al., 2017; Caya-Bissonnette et al., 2023; Jain et al., 2024). In vivo, there is a constant barrage of spontaneous synaptic inputs, probably making it easier for the somatic depolarization to trigger a dendritic plateau, but because somatic induction is generally repeated five times, it is unclear how reliable a single CS-like somatic depolarization is at triggering a dendritic plateau. In vitro, experimenters have more control over synaptic inputs, but triggering a dendritic plateau somatically is more difficult due to the lack of synaptic barrage. The ability to trigger a plateau likely depends on multiple experimental parameters such as the use of inhibition blockers or the composition of the intracellular solution (e.g., cesium has often been used to block potassium channels, improve space clamp, and increase dendritic excitability). More research is needed to explore these parameters and find better control on the induction of dendritic plateaus as well as their amplitude, spatial extent, and duration (Park et al., 2025).

In vitro, artificial BTSP induction can also be achieved with patterns of synaptic stimulation that trigger CS bursting without the need for somatic step depolarization (O'Dell, 2022, 2025). As previously noted, repeated and nearly coincident stimulations of distal and proximal dendrites of CA1 pyramidal cells can evoke LTP-inducing dendritic plateaus (Takahashi and Magee, 2009), but the potentiation of independent synaptic pathways active within seconds of the CS was not tested: it is thus unclear whether BTSP was involved. Nevertheless, it was recently shown that several seconds of theta-like (5 Hz) stimulation of projection fibers targeting proximal apical dendrites could increase dendritic excitability and lead to CS-like somatic bursts (Figs. 3*D*, 4; O'Dell, 2022). The dendritic nature of these bursts was not directly measured, but they induced high levels of pathway-specific LTP. The magnitude of LTP was correlated with the number of synaptically evoked CSs, and LTP was abolished under low concentrations of TTX (an inhibitor of voltage-gated sodium channels), showing CS bursting was necessary. Finally, the long timescale defining BTSP was revealed by stimulating one group of fibers (S1) to only evoke EPSPs, followed, after a variable delay, by the stimulation of an independent group of fibers (S2) that triggered a few CSs (Fig. 4*C*): S1 was robustly potentiated even for delays up to 4 s, confirming CS-induced plasticity is BTSP.

An alternative approach to artificial BTSP induction, which is limited to small sample sizes, has been to look for indirect signatures of BTSP in large-scale in vivo recordings of hippocampal pyramidal cells. Several studies have, for instance, attempted to detect BTSP-inducing CSs as large somatic calcium transients preceding the emergence of an asymmetric receptive field slightly shifted compared with the putative CS (as predicted from the asymmetry of the CA1 BTSP rule; Grienberger and Magee, 2022; O'Hare et al., 2022, 2025; Priestley et al., 2022; Sumegi et al., 2024; Dorian et al., 2025; Vaidya et al., 2025). A strong correlation between place field width and animal speed at the time of place field onset is a confirmatory sign of the long timescale of the plasticity rule. Others have leveraged the fact that a BTSP-triggering CS can cause a place field to shift if the CS occurs far enough from the current place field center-of-mass

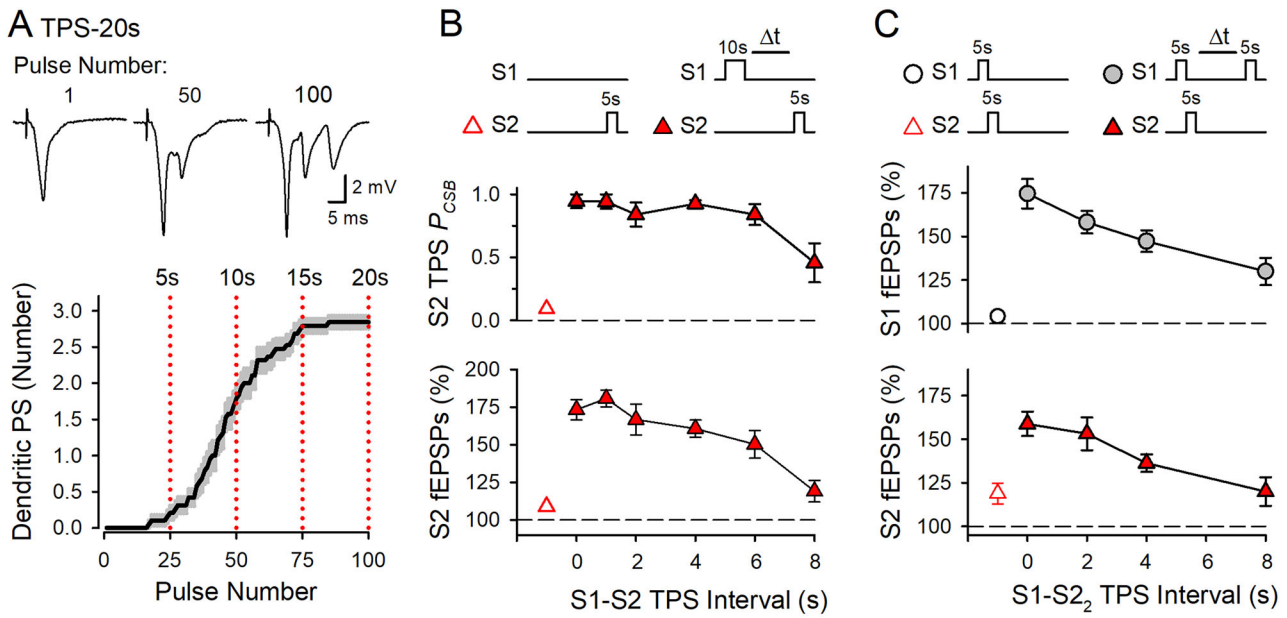

Figure 4. Synaptically induced BTSP through heterosynaptic cooperation. ***A***, 20 s trains of theta-paced stimulations (TPS, 5 Hz) of Schaffer collaterals eventually lead to CS-like bursts of population spikes (PS) in CA1 pyramidal cells. Traces show field potentials recorded in str. radiatum. ***B***, ***C***, Two stimulating electrodes (S1 and S2) were used to activate independent groups of synapses. Top, Steps represent duration of TPS trains. Bottom, LTP is measured as an increase in fEPSP slopes (percentage baseline) 45 min after TPS. ***B***, P_{CSB} is the probability of CS bursting during S2 TPS. TPS trains too short to evoke CSs on their own can lead to CSs through a cooperative increase of dendritic excitability. S2 TPS induces LTP only when P_{CSB} is high. ***C***, A sequence of short trains of TPS yields CS bursting only during the second S1 train. LTP is induced at S2 even with up to 4 s of delay between S2 TPS and S1-evoked CS bursting. Adapted from O'Dell (2022).

(Milstein et al., 2021), inferring from the diversity of lap-to-lap place–field trajectories multiple properties of BTSP (Madar et al., 2025). This last study actually suggests that comparing spatial tuning between the formation lap and the average of the following laps may not be sufficient to detect BTSP, as it does not account for shift-inducing CSs that may occur on laps following place field emergence.

BTSP Induction: Dendritic Plateau Potentials

BTSP-triggering dendritic plateaus are hard to study, so their induction mechanisms and spatiotemporal profile remain unclear. They are defined as multidendritic long-lasting calcium–mediated depolarizations associated with somatic CSs and are dependent on voltage-gated calcium channels and NMDARs (Grienberger et al., 2014; Bittner et al., 2015; Jain et al., 2024; Park et al., 2025). They are considered necessary for BTSP induction because, in behaving animals, high-frequency trains of somatic spikes without an underlying plateau could not induce a new place field, showing that bAPs from somatic bursting are not sufficient to drive BTSP (Bittner et al., 2015). Complementary findings were reported in brain slices (Cepeda-Prado et al., 2022; O'Dell, 2022). However, low concentration or somatic puffs of TTX prevents CS-dependent LTP (Takahashi and Magee, 2009; O'Dell, 2022; Jain et al., 2024), suggesting that bAPs may have a role perhaps in triggering or sustaining the plateau through interactions with local dendritic spikes (Stuart and Spruston, 2015; Park et al., 2025). Importantly, action potential backpropagation is relatively limited in distal dendrites, especially during high-frequency action potential firing (Spruston et al., 1995; Golding et al., 2001). The current emerging picture is that a sufficiently long depolarization of apical dendrites increases dendritic excitability through the slow inactivation of dendritic potassium channels, allowing further properly timed depolarization (from synaptic or bAP origin) to trigger a dendritic sodium spike in the distal apical trunk which can propagate to the tuft (Park et al., 2025). If enough dendritic potassium channels are inactivated, coinciding strong synaptic drive to the distal tuft will initiate a widespread calcium plateau triggering somatic spiking, which can reverberate in the dendrites to prolong their excitability (Larkum et al., 1999; Magee and Carruth, 1999; Takahashi and Magee, 2009; Park et al., 2025). Distal dendritic spikes are necessary to connect the proximal and tuft compartments and induce the plateau, the duration of which depends on the timing of synaptic inputs (Park et al., 2025). However, the precise initiation site of plateaus is not clear: they were thought to be initiated in the tuft, but recent in vivo calcium imaging shows plateau-like events in tuft dendrites occur mostly after signatures of BTSP induction, suggesting that they might originate in the distal apical trunk below (O'Hare et al., 2025).

Overall, dendritic spikes and plateaus require sufficient dendritic depolarization which can theoretically come from bursts of bAPs or simultaneous activation of clustered synapses (Bono and Clopath, 2017; Ujfalussy and Makara, 2020; Park et al., 2025). Such functionally clustered glutamatergic inputs exist in proximal (Adoff et al., 2021; Gonzalez et al., 2025) and perhaps even more in distal compartments (Bloss et al., 2018; Tasciotti et al., 2025). Increases in dendritic excitability triggered by cooperative synaptic interactions may also be important: for example, long trains of theta-paced extracellular stimulations of apical synapses have been shown to facilitate CS and BTSP induction by an independent set of apical inputs (O'Dell, 2022; Fig. 4*B*).

What are the circuit mechanisms and activity patterns causing dendritic plateaus? In CA1, pyramidal cells receive two main excitatory inputs (Basu and Siegelbaum, 2015): proximal dendrites are mainly contacted by axons from CA3 that can initiate somatic spiking, whereas the distal apical tuft receives inputs from the entorhinal cortex (EC) that do not reach the soma unless they initiate boosting dendritic spikes (Ang et al., 2005; Jarsky et al., 2005). Correlated CA3 and EC inputs have been shown to trigger plateaus/CSs in vitro (Takahashi and Magee, 2009; Bittner et al., 2015; Milstein et al., 2015; Park et al., 2025). The critical role of both CA3 and EC inputs for plateau induction and BTSP-related place field emergence in CA1 has been confirmed in vivo with optogenetic silencing (Bittner et al., 2015; Grienberger and Magee, 2022; Fan et al., 2023; Madar et al., 2025). Importantly, during navigation, CA3 and EC inputs generally arrive at different phases of the hippocampal theta rhythm (Mizuseki et al., 2009; Valero and De La Prida, 2018). This slight delay is thought to allow CA3-evoked depolarizations to backpropagate and coincide with EC-evoked dendritic depolarizations to initiate regenerative events leading to a plateau (Ang et al., 2005; Bittner et al., 2015; Park et al., 2025). Note, however, that plateau induction may not always require EC inputs, since strong (Remy and Spruston, 2007) or repeated (O'Dell, 2022) CA3 inputs are sufficient to trigger CSs in vitro. Pyramidal cells must also escape local inhibition to allow both somatic bursting and dendritic spiking (Lovett-Barron et al., 2012; Royer et al., 2012; Milstein et al., 2015; O'Dell, 2025), with specific disinhibitory networks transiently engaged by novelty or explicit learning tasks (Basu et al., 2016; Sheffield et al., 2017; Pedrosa and Clopath, 2020; Bilash et al., 2023; Udakis et al., 2024; Neubrandt et al., 2025).

When and how often do BTSP-inducing plateaus occur in vivo? Despite recent progress, we still don't know how to predict BTSP induction events. Direct measures of spontaneous CS occurrence in single CA1 pyramidal neurons in vivo have been inconsistent, likely due to low sample sizes: some reported an average of 1.8 CSs per 100 spikes in familiar environments (Bittner et al., 2015), some suggested CSs are much rarer (Cohen et al., 2017), and others suggested that they are much more frequent (Fan et al., 2023). An analysis of thousands of hippocampal place fields compared with computational simulations of a model of pyramidal cell with plastic synapses shows that the average probability of BTSP induction is low (at most 0.5 plateau per 100 spikes in a novel environment, 0.2 in a familiar one; Madar et al., 2025). However, the probability of BTSP induction is dynamic: quite high just after place field emergence before it decays to a very low but nonzero value (Fig. 5). In other words, dendritic plateaus occasionally happen long after place field emergence, sometimes outside of the place field, as corroborated by direct examples from other studies (Bittner et al., 2015; Milstein et al., 2021; O'Hare et al., 2025). Reports of high frequency of short CSs (Fan et al., 2023) suggest that not all plateaus induce BTSP or not to the same level, with longer ones being perhaps more impactful (Takahashi and Magee, 2009; Milstein et al., 2020).

Factors regulating the frequency, duration, and spatial extent of plateaus are not well understood. Local inhibition and disinhibitory networks are critical (see above). Increased synaptic drive is another likely contributor, as it has been shown to prolong local NMDA plateaus in basal dendrites (Bono and Clopath, 2017). Neuromodulatory signals of novelty, surprise, reward, or attention (Kaufman et al., 2020; Krishnan et al., 2022; Heer and Sheffield, 2023), which are known to affect synaptic plasticity

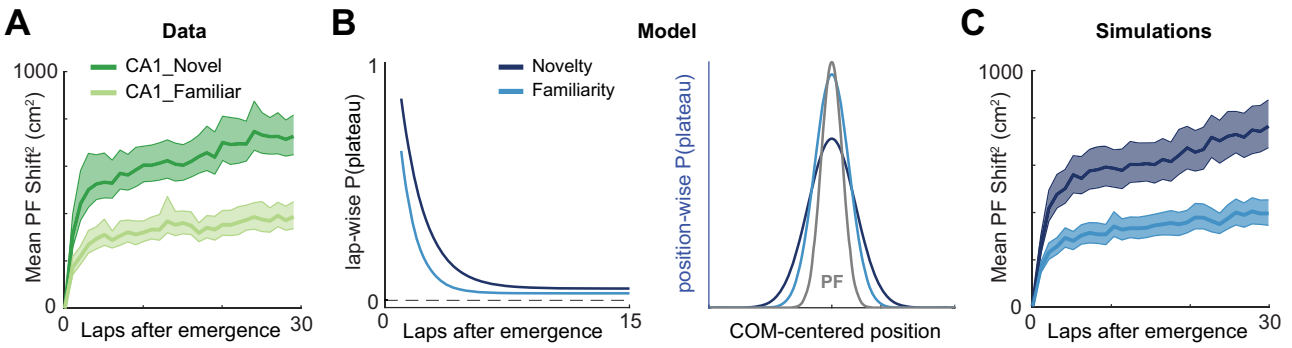

Figure 5. The probability of spontaneous BTSP induction is dynamic. ***A***, Mean squared displacement of place fields (PFs) from lap to lap, recorded in CA1 in mice running in novel ($n = 1,167$ PFs) or familiar environments ($n = 1,068$ PFs). PF shifts result from dendritic plateaus inducing BTSP: the farther the plateau is from the current PF center-of-mass (COM), the larger the shift. ***B***, The data in A suggest a model with the probability of a BTSP-inducing plateau starting high but decaying to a nonzero value after PF emergence. Plateaus are more likely in novel environments, where they are also more likely to occur outside of the current PF (gray Gaussian in right panel). ***C***, Simulations ($n = 500$ PFs) confirm that the model in B explains the shifts observed in ***A***. Adapted from Madar et al. (2025).

(Fuchsberger and Paulsen, 2022), may also play a role. For instance, cholinergic activation can induce very long CSs (Fraser and MacVicar, 1996), and β-adrenergic receptors facilitate dendritic excitability and somatic bursting in CA1 pyramidal cells (Hoffman and Johnston, 1999; Liu et al., 2017) as well as the induction of CS-dependent LTP (O'Dell, 2025). Finally, short-term plasticity can also impact dendritic excitability and constrain the induction of BTSP (O'Dell, 2022).

BTSP Induction: Behavioral Timescale

In addition to being triggered by dendritic plateaus, a defining feature of BTSP is its long timescale: synaptic inputs active within an approximately ±5 s temporal window around the plateau will undergo significant weight changes, in a synaptic-specific manner. Phenomenologically, this can be modeled with eligibility traces (ETs) in two-factor plasticity rules (Fig. 1*C*): each presynaptic spike triggers a local signal decaying with a long time constant, representing a short-term memory of the input, whereas the plateau initiates a multidendritic (perhaps "global") signal decaying with a long but shorter time constant. The local and global time constants define the degree of asymmetry of the BTSP rule (Fig. 1*B*,*C*). Each signal is by definition insufficient to trigger plasticity: it is the combination of both local and global signals at each synapse that determines the change in synaptic weight (Cone and Shouval, 2021; Milstein et al., 2021; Madar et al., 2025). These ETs are abstract variables however, and, despite recent advances, their molecular underpinning is still mysterious.

The notion that local ETs might provide a substrate to store information about events that unfold over behavioral timescales was first proposed >50 years ago (Crow, 1968) and is at the core of modern theories of learning (Redondo and Morris, 2011; Frémaux and Gerstner, 2015; Sutton and Barto, 2018; Magee and Grienberger, 2020; Namboodiri, 2024). Yet, the first physiological evidence for ETs is very recent, and BTSP is one of the prime examples (Gerstner et al., 2018; Shouval and Kirkwood, 2025). How are they triggered? In Hebbian theories, coincidence between presynaptic glutamate release and postsynaptic depolarization is necessary (Gerstner et al., 2018) as it would open NMDARs to let calcium flow in the synaptic spine and initiate molecular cascades that may underlie a local ET. However, BTSP can be induced in neurons with a flat somatic membrane potential and manipulations of the membrane potential have little effect on weight changes, suggesting that local ET induction does not require much global depolarization at the time of synaptic activation (Milstein et al., 2021). Simulations suggest that spine compartmentalization and high spine neck resistance may yield sufficient depolarization locally every time a single spine is activated to open NMDARs (Milstein et al., 2021), allowing sufficient calcium influx to start a local ET (Magee and Grienberger, 2020) but perhaps not enough to express LTP or LTD. Clustered inputs and cooperativity across spines may also help reach the depolarization threshold. Note that despite the clear dependence of BTSP on the ionotropic function of NMDARs (Bittner et al., 2015, 2017; O'Dell, 2022; Caya-Bissonnette et al., 2023; Jain et al., 2024), the role of synaptic NMDARs in local ET induction is not clear because they also play roles in the induction and maintenance of the dendritic plateau initiating the global signal (Park et al., 2025). Nonconventional signaling from NMDARs or activation of metabotropic glutamatergic receptors may also be involved in triggering local ETs (Nabavi et al., 2013; Stein et al., 2021; Jain et al., 2024).

Calcium is an essential effector of synaptic plasticity in general (Graupner and Brunel, 2010; Evans and Blackwell, 2015; Ebner et al., 2019; Inglebert et al., 2020) and both experimental and modeling studies suggest BTSP relies on similar calcium-dependent cascades (Bittner et al., 2017; Caya-Bissonnette et al., 2023; G. Li et al., 2023; Jain et al., 2024; Moldwin et al., 2025). However, calcium transients in spines last only tens of milliseconds (Miyazaki and Ross, 2022), and calcium plateaus are a few hundreds of milliseconds at most: these signals appear too short to explain the long timescale of both local and global ETs. Recent work suggests that the time course of calcium signals is extended by interactions with the endoplasmic reticulum (ER; Caya-Bissonnette et al., 2023), a large organelle acting as a calcium store and present throughout dendrites and spines (Konietzny et al., 2023): the ER may buffer cytosolic calcium from a first event (a synaptic input or a plateau), leading to a long-lasting ER transient, and then restore calcium to the cytosol in response to a new, delayed, calcium event (plateau or synaptic input) through calcium-induced calcium release (CICR; Caya-Bissonnette et al., 2023; Moldwin et al., 2025). Consistent with this scenario, genetic or pharmacologic manipulations of intracellular calcium release have been shown to alter BTSP in vivo (O'Hare et al., 2022) and in vitro (Jain et al., 2024).

In addition to ER-mediated delayed calcium dynamics, the calcium-dependent protein kinase CaMKII may also serve as an integrator of local and global signals supporting the long

timescales of BTSP (G. Li et al., 2023), but its role is less clear. CaMKII is essential for LTP and can remain active for dozens of seconds thanks to its autophosphorylation property (Yasuda et al., 2022). A point mutation impairing this autophosphorylation abolishes BTSP in vitro and in vivo, consistent with an essential role for the expression of LTP (Xiao et al., 2023; Jain et al., 2024). Furthermore, using an improved CaMKII sensor in hippocampal organotypic slices also revealed that CaMKII is activated during spontaneous calcium plateaus and depends on intracellular calcium release (Jain et al., 2024). However, this activity was not localized in the stimulated spines but throughout dendrites, with a spatial profile largely restricted in the stimulated branch. Being neither synapse-specific nor widespread, the CaMKII signal is difficult to reconcile with the notions of independent local and global ETs and suggest that it may be part of a cascade integrating both signals. Note that if the long timescale of BTSP is underpinned by such an integration of local and global calcium (through ER and/or CaMKII), the asymmetry of BTSP (i.e., the difference in local and global time constants) may be explained by complex nonlinear dynamics that need to be clarified (G. Li et al., 2023). The mechanism for synapse specificity also remains to be identified but could come from ER invasions of spines providing delayed and local CICR to activate spatially restricted enzymes. Finally, local protein synthesis may be involved, as it was shown to support local ETs in non-BTSP protocols (Fuchsberger et al., 2022).

BTSP Expression: Locus, Amplitude, and Sign

The final defining feature of BTSP is its large amplitude, which can mediate one-shot LTP and sudden place field emergence. Computational models fitted to in vitro and in vivo data indicate that the maximum synaptic potentiation is 8–16 times higher in BTSP compared with STDP (Madar et al., 2025). The reason for that order of magnitude of difference is not well understood. It may simply be due to the much higher levels of calcium coming from the plateau and CICR. Experiments pairing glutamate-uncaging stimulations of single spines with a CS suggest that the magnitude of LTP is correlated with the delay of CaMKII activity (Jain et al., 2024). Furthermore, BTSP may lead to faster LTP saturation at individual synapses (Milstein et al., 2021), but its strength might also be due to its ability to potentiate clusters of synapses (Gonzalez et al., 2025).

Where do these synaptic changes occur? BTSP protocols in hippocampal slices have generally investigated plasticity of inputs to proximal apical dendrites, i.e., from CA3 (Bittner et al., 2017; O'Dell, 2022; Jain et al., 2024), but robust plateau-induced LTP of distal tuft inputs, i.e., mostly from EC, has also been shown (Takahashi and Magee, 2009). In vivo, optogenetic stimulations directly confirmed that contralateral CA3 inputs are potentiated after CS induction and that this was not due to intrinsic plasticity (Fan et al., 2023). In vitro experiments testing paired-pulse facilitation before and after BTSP induction also demonstrated that the locus of expression is mainly postsynaptic (Bittner et al., 2017). This was confirmed in vivo by directly measuring presynaptic glutamate release, which did not change much after place field induction, in contrast to corresponding spine-restricted calcium transients, which increased (Gonzalez et al., 2025). Intriguingly, no structural changes were detected in otherwise potentiated spines. This study also confirmed that spatially tuned spines undergo LTP and LTD according to a rule qualitatively similar to the weight-dependent BTSP rule previously inferred (Fig. 2*F*; Milstein et al., 2021). Most importantly, the expression of BTSP was not uniform across the dendritic tree: BTSP was observed mostly on proximal apical dendrites, not in the basal compartment. This confirms in vitro findings (Jain et al., 2024) and is in alignment with the growing body of work showing a functional dichotomy between the two compartments in pyramidal cells (Piskorowski and Chevaleyre, 2011; Stuart and Spruston, 2015; O'Hare et al., 2022; Wright et al., 2025).

An unresolved matter is how the quick saturation of LTP and the expression of LTD are orchestrated after BTSP induction. BTSP was initially reported as a purely potentiating rule (Fig. 1*B*), but a CS has little effect if it occurs in a pre-existing place field and can depotentiate it if triggered at a different location (Fig. 2). This phenomenon can be explained by a homosynaptic rule where the sign and amplitude of plasticity depend on the interval between CS and synaptic inputs as well as the initial weight of active synapses (Milstein et al., 2021). In that case, LTP and LTD may be the result of a single local ET with two different thresholds and expression cascades or the result of two different ETs with different dynamics (Cone and Shouval, 2021). BTSP may also not be entirely homosynaptic: an alternative model suggests that the observed bidirectional changes could also be the result of a homosynaptic potentiation rule combined with fast homeostatic heterosynaptic plasticity (Madar et al., 2025). Future in vitro and in vivo experiments and more biophysically realistic models are needed to test each model's predictions and specify the mechanisms. Interestingly, a study recently reported that, after place field induction, LTP occurs in clusters whereas LTD is broader across a dendritic branch (Gonzalez et al., 2025). This suggests two independent processes and is potentially consistent with fast and local heterosynaptic effects (El-Boustani et al., 2018; Moldwin et al., 2023).

There are many outstanding questions. For instance, the kinetics of BTSP expression and maintenance are not well understood (Madar et al., 2025). They may not be different from what is known of LTP in general, but the effects of plateau-induced plasticity have not been investigated beyond the early phase (Reymann and Frey, 2007; Bliss et al., 2018). Whether BTSP impacts inhibitory synapses is also largely unknown, although it likely supports a functional reorganization participating in place field emergence (Geiller et al., 2022) and computational modeling suggests that plateau-driven plasticity of inhibitory inputs is important for efficient learning (Galloni et al., 2025).

Localization: Where Does BTSP Occur?

BTSP was first discovered in CA1 pyramidal cells, and most of the research on BTSP has focused on that region of the hippocampus. However, any neuron able to trigger dendritic plateaus may in theory undergo BTSP.

For example, CA3 pyramidal cells can exhibit CS-associated plateaus, but this property is dependent on the septotemporal position, which correlates with gradients of specific dendritic conductances (Balind et al., 2019)—such gradients also exist in CA1 (Soltesz and Losonczy, 2018) and may underlie some heterogeneity in BTSP induction experiments. Recent studies have confirmed that BTSP occurs in CA3, albeit with phenomenological differences from CA1: in familiar environments, the BTSP kernel is symmetric (Li et al., 2024; Madar et al., 2025) but highly asymmetric in novel contexts (Madar et al., 2025). This switch may be due to changes in the duration of dendritic plateaus: CSs are longer in CA3 than CA1 in familiar environments (Bittner et al., 2015; Li et al., 2024), but a CA3-specific short dendritic calcium spike (Magó et al., 2021) could be more prevalent during novelty. Spontaneous BTSP induction also appears less

frequent in CA3 than CA1 and happens less often outside of the current place field (Madar et al., 2025). Finally, optogenetic experiments showed that BTSP in CA3 potentiates recurrent CA3 inputs (Li et al., 2024). The effect on DG and EC inputs and the role of these synaptic sources in BTSP induction are unclear.

Dendritic plateaus have also been recorded in pyramidal cells from various regions of the neocortex (Larkum et al., 1999; Xu et al., 2012) and have been related to plasticity in vivo (Gambino et al., 2014). Consistently, in vitro experiments in slices of the prefrontal cortex suggest that a form of long-timescale synaptic plasticity depending on protracted ER-mediated dendritic calcium signaling can be induced by CS-like somatic depolarizations (Caya-Bissonnette et al., 2023). Intriguingly, the plasticity kernel uncovered is very different from hippocampal BTSP. It is unclear if this is due to experimental or physiological differences with CA1.

It is not currently known whether BTSP occurs in nonpyramidal cells, but dendritic plateaus have been reported in Purkinje cells of the cerebellum (Davie et al., 2008) as well as in spiny neurons of the striatum, amygdala, and cortex (Oikonomou et al., 2014). Little is known about dendritic integration in GABAergic interneurons, but dendritic spikes may occur and play an important role in some cell types (Larkum et al., 2022; Tzilivaki et al., 2022).

Functions of BTSP

The discovery of BTSP was intimately linked to its capacity to shape hippocampal representations (Bittner et al., 2017). Indeed, the magnitude and long timescale of BTSP make the rapid emergence of spatial tuning possible with a single dendritic plateau. Moreover, BTSP is sufficient to induce remapping (Milstein et al., 2021) and the emergence of mixed selectivity (Zhao et al., 2022; Cone and Clopath, 2024). However, does BTSP naturally support neuronal representations in the brain? Because of methodological variations (e.g., place field detection criteria), estimates of the prevalence of BTSP-induced formation of place fields in CA1 vary across studies: overall, BTSP does not appear necessary, but a substantial proportion of newly formed place fields shows signatures of BTSP around onset (Cohen et al., 2017; Grienberger and Magee, 2022; Priestley et al., 2022; Sumegi et al., 2024; Madar et al., 2025; Vaidya et al., 2025), and this is also the case for the emergence of nonspatial tuning (Dorian et al., 2025). Furthermore, simulations show that BTSP can explain the diverse and nonlinear lap-to-lap shifting dynamics observed in hippocampal place fields, but STDP cannot (Madar et al., 2025). Thus, BTSP is an important determinant of neuronal activity in vivo that underlies a rapid form of representational drift (Dong et al., 2021; Zheng et al., 2024) perhaps to support time coding and pattern separation (Madar, 2018; Mau et al., 2020).

In addition to shaping neuronal representations, BTSP has been suggested as a better candidate than Hebbian plasticity to support one-shot or delayed associative learning (Magee and Grienberger, 2020). However, establishing a convincing link between a particular form of plasticity and behavioral learning is difficult and, as suggested by others (Takeuchi et al., 2014; Dringenberg, 2020), requires verifying that (1) it correlates with learning performance, (2) preventing it blocks learning, and (3) inducing it causes learning. These questions have just started to be addressed. Some correlative studies have, for instance, shown that BTSP-triggering events are more frequent when exploring a new environment than a familiar one, suggesting that BTSP induction increases with higher needs for learning (Priestley et al., 2022; Madar et al., 2025). BTSP signatures also correlate with the stabilization of spatial representations while familiarizing to a context over several days (Vaidya et al., 2025) and with the development of the over-representation of reward zones (Grienberger and Magee, 2022). Moreover, in silico simulations showed that BTSP can cause the reorganization of place fields around rewards (Milstein et al., 2021). The impact of experimentally reducing BTSP on learning behavior is unclear (Grienberger and Magee, 2022), but a recent report demonstrated that BTSP can be used to enable a virtual agent to rapidly learn an efficient path to a reward (Galloni et al., 2024). The role of BTSP in delayed or one-shot memory remains to be tested experimentally, in part because methods to directly manipulate the induction of dendritic plateaus are lacking.

Nevertheless, there is a growing body of theoretical work showing that BTSP efficiently supports learning and memory in artificial neural networks. Importantly, sparse and stochastic induction of a symmetric form of BTSP, as found in CA3 in familiar environments, is able to rapidly form stable attractors and increases memory capacity compared with Hebbian learning rules (P. Y. Li and Roxin, 2023; Wu and Maass, 2025) in part thanks to the weight-dependent nature of BTSP that enforces pattern separation by depressing large synaptic weights overlapping with previously encoded patterns (Li et al., 2024). Interestingly, a symmetric BTSP-like rule has also been shown to enhance the ability of recurrent networks to learn and replay sequences of neuronal activity offline (Ecker et al., 2022; Milstein et al., 2023), a phenomenon linked to memory recall and consolidation (Buzsáki, 2015; van der Meer and Bendor, 2025).

Finally, BTSP has been proposed as a biological mechanism of supervised learning. The critical aspect of BTSP for this function is that it is triggered by dendritic plateaus that can be evoked by the coincidence of two input pathways, a bottom-up pathway targeting proximal dendrites (e.g., CA3) and a top–down pathway targeting distal dendrites (e.g., EC; Magee and Grienberger, 2020). This allows the substrate for "reading out" stored memories—somatic spikes—to be decoupled from the substrate for "writing in" new memories, dendritic plateaus. If the top–down input pathway is required to trigger plateaus and carries information about reward or other behavioral consequences, then this also enables plasticity to be shaped by learning targets rather than by spurious "Hebbian" correlations. However, it is still unclear how the credit assignment problem is solved, i.e., how numerous EC inputs determine which sparse subset of pyramidal neurons is selected to evoke plasticity at a given time during learning. Building on previous biology-inspired approaches to credit assignment in multilayer networks (Guerguiev et al., 2017; Sacramento et al., 2018; Richards and Lillicrap, 2019; Payeur et al., 2021; Greedy et al., 2022), current theory proposes that dendrite-targeting feedback inhibition could prevent BTSP by suppressing dendritic spikes, suggesting that a dynamic balance between top–down excitation and local inhibition gates plasticity (Milstein et al., 2015, 2021; Grienberger and Magee, 2022; Rolotti et al., 2022). In this theory, EC inputs provide a learning target, and BTSP-inducing plateaus constitute an error signal, computed as the difference between dendritic excitation and inhibition. An implementation of this theory solves the credit assignment problem and results in image classification performance comparable with "backprop," the standard but biologically implausible machine learning algorithm (Galloni et al., 2025; Fig. 6).

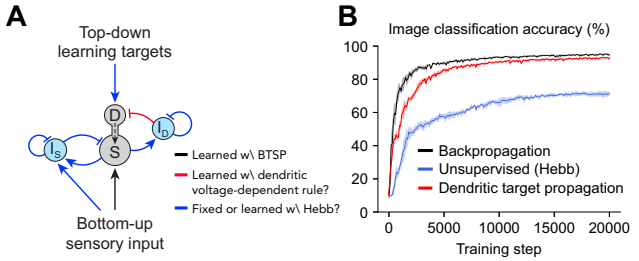

Figure 6. BTSP can support supervised learning. ***A***, Model microcircuit based on CA1 network. Excitatory neurons with two compartments (soma S, dendrite D) receive CA3-like excitatory inputs providing sensory information to S and EC-like top–down inputs providing learning targets to D. The network includes somatic (like PV + basket cells) and dendritic inhibition (like SST + OLM interneurons). Sensory weights (black) are learned with BTSP, triggered when the sum of top–down excitation and lateral inhibition crosses a threshold. Adapting the strength of top–down and lateral inputs to dendrites improves performance during target-directed learning. ***B***, Multilayer artificial neural networks were trained to classify images (MNIST dataset) using different algorithms. In contrast to a Hebbian rule, gating of BTSP by dendritic inhibition as shown in ***A*** (red) approximates backprop (black). Adapted from Galloni et al. (2025).

BTSP is indeed able to solve several unsupervised and supervised learning tasks, but the long timescale of BTSP makes fine temporal credit assignment more challenging, especially in deep recurrent networks (Cone et al., 2025). Moreover, if BTSP enables one-shot learning, its saturability is not well suited to extract regularities from the environment over numerous repetitions. The opposite is true for STDP, suggesting that complementary forms of plasticity may allow the brain to flexibly achieve both episodic memory and statistical learning (Schapiro et al., 2017).

Conclusion

Early studies of synaptic plasticity recognized the limitations of Hebbian STDP based on spike timing correlations and found that LTP could be induced faster and more robustly by pairing synaptic inputs with bursts of somatic action potentials or with dendritic spikes. However, they failed to recognize the striking effect on delayed synaptic inputs, either because long intervals were not tested (Pike et al., 1999; Wittenberg and Wang, 2006; Remy and Spruston, 2007; Takahashi and Magee, 2009) or because simple bursts are not sufficient to trigger dendritic plateaus (Cepeda-Prado et al., 2022; Park et al., 2025). The discovery of BTSP represents a paradigm shift: unlike Hebbian plasticity, the properties of BTSP—its seconds-long timescale, its large amplitude, and its induction by dendritic plateaus—position it as a good physiological solution to several fundamental challenges in neuroscientific theories of learning, such as delayed associations, one-shot learning, and credit assignment.

After the initial discovery of BTSP (Bittner et al., 2017), the field is now undergoing an exciting boom of research across many labs, not unlike what happened after the discovery of STDP 20 years before (Magee and Johnston, 1997; Markram et al., 1997). The phenomenology and circuit, cellular, and molecular mechanisms of BTSP are being explored in vitro and in behaving animals with an expanding array of techniques, and its role in shaping neural networks has initiated an ongoing effervescence of theory and modeling to study how it can support episodic memory, goal-directed navigation, and supervised learning, with implications and potential applications to the field of machine learning and neurocomputing (Galloni et al., 2025; Yang et al., 2025).

This is still the beginning and major questions remain. The precise mechanisms governing when and where BTSP-triggering dendritic plateaus occur, the biological underpinnings of long ETs, the phenomenology of BTSP in different brain regions, and the causal role of BTSP in specific learning behaviors all require further investigation. Additionally, understanding how BTSP relates to other forms of plasticity and homeostatic mechanisms will be crucial for developing a comprehensive theory of neural learning. In a way, BTSP, STDP, neuromodulated Hebbian plasticity, and other plastic phenomena are all facets of the same object, reflecting different aspects of a common neurophysiology that could support learning and memory in a complementary manner (Redondo and Morris, 2011; Gerstner et al., 2018; Fuchsberger et al., 2022; O'Donnell, 2023; Shouval and Kirkwood, 2025).

References

Abraham WC (2008) Metaplasticity: tuning synapses and networks for plasticity. Nat Rev Neurosci 9:387.
Acker D, Paradis S, Miller P (2019) Stable memory and computation in randomly rewiring neural networks. J Neurophysiol 122:66–80.
Adoff MD, Climer JR, Davoudi H, Marvin JS, Looger LL, Dombeck DA (2021) The functional organization of excitatory synaptic input to place cells. Nat Commun 12:3558.
Ang CW, Carlson GC, Coulter DA (2005) Hippocampal CA1 circuitry dynamically gates direct cortical inputs preferentially at theta frequencies. J Neurosci 25:9567–9580.
Antic SD, Zhou W-L, Moore AR, Short SM, Ikonomu KD (2010) The decade of the dendritic NMDA spike. J Neurosci Res 88:2991–3001.
Balind SR, Magó Á, Ahmadi M, Kis N, Varga-Németh Z, Lőrincz A, Makara JK (2019) Diverse synaptic and dendritic mechanisms of complex spike burst generation in hippocampal CA3 pyramidal cells. Nat Commun 10:1859.
Basu J, Siegelbaum SA (2015) The corticohippocampal circuit, synaptic plasticity, and memory. Cold Spring Harb Perspect Biol 7:a021733.
Basu J, Zaremba JD, Cheung SK, Hitti FL, Zemelman BV, Losonczy A, Siegelbaum SA (2016) Gating of hippocampal activity, plasticity, and memory by entorhinal cortex long-range inhibition. Science 351:aaa5694.
Bi G, Poo M (2001) Synaptic modification by correlated activity: Hebb's postulate revisited. Annu Rev Neurosci 24:139–166.
Bilash OM, Chavlis S, Johnson CD, Poirazi P, Basu J (2023) Lateral entorhinal cortex inputs modulate hippocampal dendritic excitability by recruiting a local disinhibitory microcircuit. Cell Rep 42:111962.
Bittner KC, Grienberger C, Vaidya SP, Milstein AD, Macklin JJ, Suh J, Tonegawa S, Magee JC (2015) Conjunctive input processing drives feature selectivity in hippocampal CA1 neurons. Nat Neurosci 18:1133–1142.
Bittner KC, Milstein AD, Grienberger C, Romani S, Magee JC (2017) Behavioral time scale synaptic plasticity underlies CA1 place fields. Science 357:1033–1036.
Bliss TVP, Collingridge GL, Morris RGM, Reymann KG (2018) Long-term potentiation in the hippocampus: discovery, mechanisms and function. Neuroforum 24:A103–A120.
Bloss EB, Cembrowski MS, Karsh B, Colonell J, Fetter RD, Spruston N (2018) Single excitatory axons form clustered synapses onto CA1 pyramidal cell dendrites. Nat Neurosci 21:353–363.
Bono J, Clopath C (2017) Modeling somatic and dendritic spike mediated plasticity at the single neuron and network level. Nat Commun 8:706.
Boscaglia M, Gastaldi C, Gerstner W, Quian Quiroga R (2023) A dynamic attractor network model of memory formation, reinforcement and forgetting. PLoS Comput Biol 19:e1011727.
Bouvier G, Aljadeff J, Clopath C, Bimbard C, Ranft J, Blot A, Nadal J-P, Brunel N, Hakim V, Barbour B (2018) Cerebellar learning using perturbations. Elife 7:e31599.
Brzosko Z, Mierau SB, Paulsen O (2019) Neuromodulation of spike-timing-dependent plasticity: past, present, and future. Neuron 103:563–581.
Buchanan KA, Mellor JR (2010) The activity requirements for spike timing-dependent plasticity in the hippocampus. Front Synaptic Neurosci 2:11.
Buzsáki G (2015) Hippocampal sharp wave-ripple: a cognitive biomarker for episodic memory and planning. Hippocampus 25:1073–1188.

Carlisle HJ, Fink AE, Grant SGN, O'Dell TJ (2008) Opposing effects of PSD-93 and PSD-95 on long-term potentiation and spike timing-dependent plasticity. J Physiol 586:5885–5900.
Caya-Bissonnette L, Naud R, Béïque J-C (2023) Cellular substrate of eligibility traces (p. 2023.06.29.547097). bioRxiv.
Cepeda-Prado EA, Khodaie B, Quiceno GD, Beythien S, Edelmann E, Lessmann V (2022) Calcium-permeable AMPA receptors mediate timing-dependent LTP elicited by low repeat coincident pre- and postsynaptic activity at Schaffer collateral-CA1 synapses. Cereb Cortex 32:1682–1703.
Chenkov N, Sprekeler H, Kempter R (2017) Memory replay in balanced recurrent networks. PLoS Comput Biol 13:e1005359.
Clopath C, Büsing L, Vasilaki E, Gerstner W (2010) Connectivity reflects coding: a model of voltage-based STDP with homeostasis. Nat Neurosci 13:344–352.
Cohen JD, Bolstad M, Lee AK (2017) Experience-dependent shaping of hippocampal CA1 intracellular activity in novel and familiar environments. Elife 6:e23040.
Cone I, Clopath C (2024) Latent representations in hippocampal network model co-evolve with behavioral exploration of task structure. Nat Commun 15:687.
Cone I, Shouval HZ (2021) Behavioral time scale plasticity of place fields: mathematical analysis. Front Comput Neurosci 15:640235.
Cone I, Clopath C, Costa RP (2025) Credit assignment via behavioral timescale synaptic plasticity: theoretical frameworks (p. 2025.06.12.659336). bioRxiv.
Crow TJ (1968) Cortical synapses and reinforcement: a hypothesis. Nature 219:736–737.
D'Albis T, Jaramillo J, Sprekeler H, Kempter R (2015) Inheritance of hippocampal place fields through Hebbian learning: effects of theta modulation and phase precession on structure formation. Neural Comput 27:1624–1672.
Davie JT, Clark BA, Häusser M (2008) The origin of the complex spike in cerebellar Purkinje cells. J Neurosci 28:7599–7609.
Debanne D, Inglebert Y (2023) Spike timing-dependent plasticity and memory. Curr Opin Neurobiol 80:102707.
Debanne D, Gähwiler BH, Thompson SM (1996) Cooperative interactions in the induction of long-term potentiation and depression of synaptic excitation between hippocampal CA3-CA1 cell pairs in vitro. Proc Natl Acad Sci U S A 93:11225–11230.
Diamantaki M, Coletta S, Nasr K, Zeraati R, Laturnus S, Berens P, Preston-Ferrer P, Burgalossi A (2018) Manipulating hippocampal place cell activity by single-cell stimulation in freely moving mice. Cell Rep 23:32–38.
Dong C, Madar AD, Sheffield MEJ (2021) Distinct place cell dynamics in CA1 and CA3 encode experience in new environments. Nat Commun 12:2977.
Dorian CC, Taxidis J, Arac A, Golshani P (2025) Behavioral timescale synaptic plasticity in the hippocampus creates non-spatial representations during learning and is modulated by entorhinal inputs (p. 2024.08.27.609983). bioRxiv.
Dringenberg HC (2020) The history of long-term potentiation as a memory mechanism: controversies, confirmation, and some lessons to remember. Hippocampus 30:987–1012.
Ebner C, Clopath C, Jedlicka P, Cuntz H (2019) Unifying long-term plasticity rules for excitatory synapses by modeling dendrites of cortical pyramidal neurons. Cell Rep 29:4295–4307.e6.
Ecker A, et al. (2022) Hippocampal sharp wave-ripples and the associated sequence replay emerge from structured synaptic interactions in a network model of area CA3. Elife 11:e71850.
El-Boustani S, Ip JPK, Breton-Provencher V, Knott GW, Okuno H, Bito H, Sur M (2018) Locally coordinated synaptic plasticity of visual cortex neurons in vivo. Science 360:1349–1354.
Epsztein J, Brecht M, Lee AK (2011) Intracellular determinants of hippocampal CA1 place and silent cell activity in a novel environment. Neuron 70:109–120.
Evans RC, Blackwell KT (2015) Calcium: amplitude, duration, or location? Biol Bull 228:75–83.
Fan LZ, et al. (2023) All-optical physiology resolves a synaptic basis for behavioral timescale plasticity. Cell 186:543–559.e19.
Feldman DE (2012) The spike-timing dependence of plasticity. Neuron 75:556–571.
Fraser DD, MacVicar BA (1996) Cholinergic-dependent plateau potential in hippocampal CA1 pyramidal neurons. J Neurosci 16:4113–4128.
Frémaux N, Gerstner W (2015) Neuromodulated spike-timing-dependent plasticity, and theory of three-factor learning rules. Front Neural Circuits 9:85.
Fuchsberger T, Paulsen O (2022) Modulation of hippocampal plasticity in learning and memory. Curr Opin Neurobiol 75:102558.
Fuchsberger T, Clopath C, Jarzebowski P, Brzosko Z, Wang H, Paulsen O (2022) Postsynaptic burst reactivation of hippocampal neurons enables associative plasticity of temporally discontiguous inputs. Elife 11:e81071.
Gallistel CR, Matzel LD (2013) The neuroscience of learning: beyond the Hebbian synapse. Annu Rev Psychol 64:169–200.
Galloni AR, Yuan Y, Zhu M, Yu H, Bisht RS, Wu C-TM, Grienberger C, Ramanathan S, Milstein AD (2024) Neuromorphic one-shot learning utilizing a phase-transition material. Proc Natl Acad Sci U S A 121:e2318362121.
Galloni AR, Peddada A, Chennawar Y, Milstein AD (2025) Cellular and subcellular specialization enables biology-constrained deep learning. bioRxiv: The Preprint Server for Biology, 2025.05.22.655599.
Gambino F, Pagès S, Kehayas V, Baptista D, Tatti R, Carleton A, Holtmaat A (2014) Sensory-evoked LTP driven by dendritic plateau potentials in vivo. Nature 515:116–119.
Geiller T, et al. (2022) Local circuit amplification of spatial selectivity in the hippocampus. Nature 601:105–109.
Gerstner W, Lehmann M, Liakoni V, Corneil D, Brea J (2018) Eligibility traces and plasticity on behavioral time scales: experimental support of neoHebbian three-factor learning rules. Front Neural Circuits 12:53.
Gilson M, Masquelier T, Hugues E (2011) STDP allows fast rate-modulated coding with Poisson-like spike trains. PLoS Comput Biol 7:e1002231.
Golding NL, Kath WL, Spruston N (2001) Dichotomy of action-potential backpropagation in CA1 pyramidal neuron dendrites. J Neurophysiol 86:2998–3010.
Golding NL, Staff NP, Spruston N (2002) Dendritic spikes as a mechanism for cooperative long-term potentiation. Nature 418:326–331.
Gonzalez KC, et al. (2025) Synaptic basis of feature selectivity in hippocampal neurons. Nature 637:1152–1160.
Graupner M, Brunel N (2010) Mechanisms of induction and maintenance of spike-timing dependent plasticity in biophysical synapse models. Front Comput Neurosci 4:136.
Graupner M, Brunel N (2012) Calcium-based plasticity model explains sensitivity of synaptic changes to spike pattern, rate, and dendritic location. Proc Natl Acad Sci U S A 109:3991–3996.
Graupner M, Wallisch P, Ostojic S (2016) Natural firing patterns imply low sensitivity of synaptic plasticity to spike timing compared with firing rate. J Neurosci 36:11238–11258.
Greedy W, Zhu HW, Pemberton J, Mellor J, Ponte Costa R (2022) Single-phase deep learning in cortico-cortical networks. In: *Proceedings of the 36th international conference on neural information processing systems*, NIPS'22, pp 24213–24225.
Grienberger C, Magee JC (2022) Entorhinal cortex directs learning-related changes in CA1 representations. Nature 611:554–562.
Grienberger C, Chen X, Konnerth A (2014) NMDA receptor-dependent multidendrite Ca(2+) spikes required for hippocampal burst firing in vivo. Neuron 81:1274–1281.
Guerguiev J, Lillicrap TP, Richards BA (2017) Towards deep learning with segregated dendrites. Elife 6:e22901.
Hebb DO (1949) The organization of behavior: a neuropsychological theory.
Heer CM, Sheffield MEJ (2023) Distinct catecholaminergic pathways projecting to hippocampal CA1 transmit contrasting signals during behavior and learning.
Hoffman DA, Johnston D (1999) Neuromodulation of dendritic action potentials. J Neurophysiol 81:408–411.
Hopfield JJ (1982) Neural networks and physical systems with emergent collective computational abilities. Proc Natl Acad Sci U S A 79:2554–2558.
Inglebert Y, Aljadeff J, Brunel N, Debanne D (2020) Synaptic plasticity rules with physiological calcium levels. Proc Natl Acad Sci U S A 117:33639–33648.
Jain A, et al. (2024) Dendritic, delayed, stochastic CaMKII activation in behavioural time scale plasticity. Nature 635:151–159.
Jarsky T, Roxin A, Kath WL, Spruston N (2005) Conditional dendritic spike propagation following distal synaptic activation of hippocampal CA1 pyramidal neurons. Nat Neurosci 8:1667–1676.
Kampa BM, Letzkus JJ, Stuart GJ (2006) Requirement of dendritic calcium spikes for induction of spike-timing-dependent synaptic plasticity. J Physiol 574:283–290.

Kaufman AM, Geiller T, Losonczy A (2020) A role for the locus coeruleus in hippocampal CA1 place cell reorganization during spatial reward learning. Neuron 105:1018–1026.e4.
Kim Y, Hsu C-L, Cembrowski MS, Mensh BD, Spruston N (2015) Dendritic sodium spikes are required for long-term potentiation at distal synapses on hippocampal pyramidal neurons. Elife 4:e06414.
Konietzny A, Wegmann S, Mikhaylova M (2023) The endoplasmic reticulum puts a new spin on synaptic tagging. Trends Neurosci 46:32–44.
Krishnan S, Heer C, Cherian C, Sheffield MEJ (2022) Reward expectation extinction restructures and degrades CA1 spatial maps through loss of a dopaminergic reward proximity signal. Nat Commun 13:6662.
Larkum ME, Zhu JJ, Sakmann B (1999) A new cellular mechanism for coupling inputs arriving at different cortical layers. Nature 398:338–341.
Larkum ME, Wu J, Duverdin SA, Gidon A (2022) The guide to dendritic spikes of the mammalian cortex in vitro and in vivo. Neuroscience 489:15–33.
Letzkus JJ, Kampa BM, Stuart GJ (2006) Learning rules for spike timing-dependent plasticity depend on dendritic synapse location. J Neurosci 26:10420–10429.
Li G, McLaughlin DW, Peskin CS (2023) A biochemical description of postsynaptic plasticity – with timescales ranging from milliseconds to seconds (p. 2023.07.09.548255). bioRxiv.
Li PY, Roxin A (2023) Rapid memory encoding in a recurrent network model with behavioral time scale synaptic plasticity. PLoS Comput Biol 19: e1011139.
Li Y, Briguglio JJ, Romani S, Magee JC (2024) Mechanisms of memory-supporting neuronal dynamics in hippocampal area CA3. Cell 187: 6804–6819.
Lisman JE (1997) Bursts as a unit of neural information: making unreliable synapses reliable. Trends Neurosci 20:38–43.
Lisman J, Spruston N (2005) Postsynaptic depolarization requirements for LTP and LTD: a critique of spike timing-dependent plasticity. Nat Neurosci 8:839–841.
Litwin-Kumar A, Doiron B (2014) Formation and maintenance of neuronal assemblies through synaptic plasticity. Nat Commun 5:5319.
Liu Y, Cui L, Schwarz MK, Dong Y, Schlüter OM (2017) Adrenergic gate release for spike timing-dependent synaptic potentiation. Neuron 93: 394–408.
Lovett-Barron M, Turi GF, Kaifosh P, Lee PH, Bolze F, Sun X-H, Nicoud J-F, Zemelman BV, Sternson SM, Losonczy A (2012) Regulation of neuronal input transformations by tunable dendritic inhibition. Nat Neurosci 15: 423–430.
Madar AD (2018) *Pattern separation in the hippocampus, in health and epilepsy*. Madison: The University of Wisconsin-Madison.
Madar AD, Jiang A, Dong C, Sheffield MEJ (2025) Synaptic plasticity rules driving representational shifting in the hippocampus. Nat Neurosci 28: 848–860.
Magee JC, Carruth M (1999) Dendritic voltage-gated ion channels regulate the action potential firing mode of hippocampal CA1 pyramidal neurons. J Neurophysiol 82:1895–1901.
Magee JC, Grienberger C (2020) Synaptic plasticity forms and functions. Annu Rev Neurosci 43:95–117.
Magee JC, Johnston D (1997) A synaptically controlled, associative signal for Hebbian plasticity in hippocampal neurons. Science 275:209–213.
Magó Á, Kis N, Lükő B, Makara JK (2021) Distinct dendritic Ca2+ spike forms produce opposing input-output transformations in rat CA3 pyramidal cells. Elife 10:e74493.
Markram H, Lübke J, Frotscher M, Sakmann B (1997) Regulation of synaptic efficacy by coincidence of postsynaptic APs and EPSPs. Science 275:213–215.
Mau W, Hasselmo ME, Cai DJ (2020) The brain in motion: how ensemble fluidity drives memory-updating and flexibility. Elife 9:e63550.
McKenzie S, Huszár R, English DF, Kim K, Christensen F, Yoon E, Buzsáki G (2021) Preexisting hippocampal network dynamics constrain optogenetically induced place fields. Neuron 109:1040–1054.e7.
Milstein AD, Bloss EB, Apostolides PF, Vaidya SP, Dilly GA, Zemelman BV, Magee JC (2015) Inhibitory gating of input comparison in the CA1 microcircuit. Neuron 87:1274–1289.
Milstein AD, Li Y, Bittner KC, Grienberger C, Soltesz I, Magee JC, Romani S (2020) Bidirectional synaptic plasticity rapidly modifies hippocampal representations independent of correlated activity (p. 2020.02.04.934182). bioRxiv.
Milstein AD, Li Y, Bittner KC, Grienberger C, Soltesz I, Magee JC, Romani S (2021) Bidirectional synaptic plasticity rapidly modifies hippocampal representations. Elife 10:e73046.
Milstein AD, Tran S, Ng G, Soltesz I (2023) Offline memory replay in recurrent neuronal networks emerges from constraints on online dynamics. J Physiol 601:3241–3264.
Miyazaki K, Ross WN (2022) Fast synaptically activated calcium and sodium kinetics in hippocampal pyramidal neuron dendritic spines. eNeuro 9:6.
Mizuseki K, Sirota A, Pastalkova E, Buzsáki G (2009) Theta oscillations provide temporal windows for local circuit computation in the entorhinal-hippocampal loop. Neuron 64:267–280.
Moldwin T, Kalmenson M, Segev I (2023) Asymmetric voltage attenuation in dendrites can enable hierarchical heterosynaptic plasticity. eNeuro 10:7.
Moldwin T, Azran LS, Segev I (2025) A generalized mathematical framework for the calcium control hypothesis describes weight-dependent synaptic plasticity. J Comput Neurosci 53:333–357.
Nabavi S, Kessels HW, Alfonso S, Aow J, Fox R, Malinow R (2013) Metabotropic NMDA receptor function is required for NMDA receptor-dependent long-term depression. Proc Natl Acad Sci U S A 110:4027–4032.
Namboodiri VM (2024) "But why?" Dopamine and causal learning. Curr Opin Behav Sci 60:101443.
Neubrandt M, Lenkey N, Vervaeke K (2025) VIP interneurons control hippocampal place cell remapping through transient disinhibition in novel environments. Research Square.
Ocker GK, Litwin-Kumar A, Doiron B (2015) Self-organization of microcircuits in networks of spiking neurons with plastic synapses. PLoS Comput Biol 11:e1004458.
O'Dell TJ (2022) Behavioral timescale cooperativity and competitive synaptic interactions regulate the induction of complex spike burst-dependent long-term potentiation. J Neurosci 42:2647–2661.
O'Dell TJ (2025) β-adrenergic receptor activation modulates the induction of complex spike burst-dependent LTP by regulating multiple forms of heterosynaptic plasticity (p. 2025.04.28.651102). bioRxiv.
O'Donnell C (2023) Nonlinear slow-timescale mechanisms in synaptic plasticity. Curr Opin Neurobiol 82:102778.
O'Hare JK, et al. (2022) Compartment-specific tuning of dendritic feature selectivity by intracellular Ca2+ release. Science 375:eabm1670.
O'Hare JK, Wang J, Shala MD, Polleux F, Losonczy A (2025) Distal tuft dendrites predict properties of new hippocampal place fields. Neuron 113: 1969–1982.e7.
Oikonomou KD, Singh MB, Sterjanaj EV, Antic SD (2014) Spiny neurons of amygdala, striatum, and cortex use dendritic plateau potentials to detect network UP states. Front Cell Neurosci 8:292.
Park P, et al. (2025) Dendritic excitations govern back-propagation via a spike-rate accelerometer. Nat Commun 16:1333.
Payeur A, Guerguiev J, Zenke F, Richards BA, Naud R (2021) Burst-dependent synaptic plasticity can coordinate learning in hierarchical circuits. Nat Neurosci 24:1010–1019.
Pedrosa V, Clopath C (2020) The interplay between somatic and dendritic inhibition promotes the emergence and stabilization of place fields. PLoS Comput Biol 16:e1007955.
Pike FG, Meredith RM, Olding AWA, Paulsen O (1999) Postsynaptic bursting is essential for 'Hebbian' induction of associative long-term potentiation at excitatory synapses in rat hippocampus. J Physiol 518:571–576.
Piskorowski RA, Chevaleyre V (2011) Synaptic integration by different dendritic compartments of hippocampal CA1 and CA2 pyramidal neurons. Cell Mol Life Sci 69:75–88.
Priestley JB, Bowler JC, Rolotti SV, Fusi S, Losonczy A (2022) Signatures of rapid plasticity in hippocampal CA1 representations during novel experiences. Neuron 110:1978–1992.e6.
Redondo RL, Morris RGM (2011) Making memories last: the synaptic tagging and capture hypothesis. Nat Rev Neurosci 12:17–30.
Remy S, Spruston N (2007) Dendritic spikes induce single-burst long-term potentiation. Proc Natl Acad Sci U S A 104:17192–17197.
Reymann KG, Frey JU (2007) The late maintenance of hippocampal LTP: requirements, phases, 'synaptic tagging', 'late-associativity' and implications. Neuropharmacology 52:24–40.
Richards BA, Lillicrap TP (2019) Dendritic solutions to the credit assignment problem. Curr Opin Neurobiol 54:28–36.
Rolotti SV, et al. (2022) Local feedback inhibition tightly controls rapid formation of hippocampal place fields. Neuron 110:783–794.e6.
Royer S, Zemelman BV, Losonczy A, Kim J, Chance F, Magee JC, Buzsáki G (2012) Control of timing, rate and bursts of hippocampal place cells by dendritic and somatic inhibition. Nat Neurosci 15:769–775.

Sacramento J, Ponte Costa R, Bengio Y, Senn W (2018) Dendritic cortical microcircuits approximate the backpropagation algorithm. In: *Proceedings of the 32nd international conference on neural information processing systems*, NIPS'18, pp 8735–8746.

Schapiro AC, Turk-Browne NB, Botvinick MM, Norman KA (2017) Complementary learning systems within the hippocampus: a neural network modelling approach to reconciling episodic memory with statistical learning. Philos Trans R Soc Lond B Biol Sci 372:20160049.

Sheffield MEJ, Dombeck DA (2015) Calcium transient prevalence across the dendritic arbour predicts place field properties. Nature 517:200–204.

Sheffield MEJ, Adoff MD, Dombeck DA (2017) Increased prevalence of calcium transients across the dendritic arbor during place field formation. Neuron 96:490–504.e5.

Shouval HZ, Kirkwood A (2025) Eligibility traces as a synaptic substrate for learning. Curr Opin Neurobiol 91:102978.

Shouval HZ, Wang S, Wittenberg GM (2010) Spike timing dependent plasticity: a consequence of more fundamental learning rules. Front Comput Neurosci 4:19.

Soltesz I, Losonczy A (2018) CA1 pyramidal cell diversity enabling parallel information processing in the hippocampus. Nat Neurosci 21:484–493.

Spruston N, Schiller Y, Stuart G, Sakmann B (1995) Activity-dependent action potential invasion and calcium influx into hippocampal CA1 dendrites. Science 268:297–300.

Stein IS, Park DK, Claiborne N, Zito K (2021) Non-ionotropic NMDA receptor signaling gates bidirectional structural plasticity of dendritic spines. Cell Rep 34:108664.

Stuart GJ, Spruston N (2015) Dendritic integration: 60 years of progress. Nat Neurosci 18:1713–1721.

Sumegi M, Olah G, Lukacs IP, Blazsek M, Makara JK, Nusser Z (2024) Diverse calcium dynamics underlie place field formation in hippocampal CA1 pyramidal cells. Elife 13:RP103676.

Sutton RS, Barto AG (1981) Toward a modern theory of adaptive networks: expectation and prediction. Psychol Rev 88:135–170.

Sutton RS, Barto AG (2018) *Reinforcement learning: an introduction*, Ed 2. Cambridge: MIT Press; 1(2), 25.

Takahashi H, Magee JC (2009) Pathway interactions and synaptic plasticity in the dendritic tuft regions of CA1 pyramidal neurons. Neuron 62: 102–111.

Takeuchi T, Duszkiewicz AJ, Morris RGM (2014) The synaptic plasticity and memory hypothesis: encoding, storage and persistence. Philos Trans R Soc Lond B Biol Sci 369:20130288.

Tasciotti S, Iascone DM, Chavlis S, Hammond L, Katz Y, Losonczy A, Polleux F, Poirazi P (2025) From morphology to computation: how synaptic organization shapes place fields in CA1 pyramidal neurons (p. 2025.05.30.657022). bioRxiv.

Tzilivaki A, Kastellakis G, Schmitz D, Poirazi P (2022) GABAergic interneurons with nonlinear dendrites: from neuronal computations to memory engrams. Neuroscience 489:34–43.

Udakis M, Claydon M, Zhu HW, Mellor JR (2024) Hippocampal OLM interneurons regulate CA1 place cell plasticity and remapping (p. 2024.11.11.622941). bioRxiv.

Ujfalussy BB, Makara JK (2020) Impact of functional synapse clusters on neuronal response selectivity. Nat Commun 11:1413.

Vaidya SP, Li G, Chitwood RA, Li Y, Magee JC (2025) Formation of an expanding memory representation in the hippocampus. Nat Neurosci 28:1510–1518.

Valero M, De La Prida LM (2018) The hippocampus in depth: a sublayer-specific perspective of entorhinal–hippocampal function. Curr Opin Neurobiol 52:107–114.

van der Meer MAA, Bendor D (2025) Awake replay: off the clock but on the job. Trends Neurosci 48:257–267.

Wittenberg GM, Wang SS-H (2006) Malleability of spike-timing-dependent plasticity at the CA3-CA1 synapse. J Neurosci 26:6610–6617.

Wright WJ, Hedrick NG, Komiyama T (2025) Distinct synaptic plasticity rules operate across dendritic compartments in vivo during learning. Science 388:322–328.

Wu Y, Maass W (2025) A simple model for behavioral time scale synaptic plasticity (BTSP) provides content addressable memory with binary synapses and one-shot learning. Nat Commun 16:342.

Xiao K, Li Y, Chitwood RA, Magee JC (2023) A critical role for CaMKII in behavioral timescale synaptic plasticity in hippocampal CA1 pyramidal neurons. Sci Adv 9:eadi3088.

Xu N, Harnett MT, Williams SR, Huber D, O'Connor DH, Svoboda K, Magee JC (2012) Nonlinear dendritic integration of sensory and motor input during an active sensing task. Nature 492:247–251.

Yang Y, Stöckl C, Maass W (2025) A surprising link between cognitive maps, successor-relation based reinforcement learning, and BTSP (p. 2025.04.22.650046). bioRxiv.

Yasuda R, Hayashi Y, Hell JW (2022) CaMKII: a central molecular organizer of synaptic plasticity, learning and memory. Nat Rev Neurosci 23:666–682.

Zeldenrust F, Wadman WJ, Englitz B (2018) Neural coding with bursts-current state and future perspectives. Front Comput Neurosci 12:48.

Zenke F, Gerstner W (2017) Hebbian plasticity requires compensatory processes on multiple timescales. Philos Trans R Soc Lond B Biol Sci 372:20160259.

Zhao X, Hsu C-L, Spruston N (2022) Rapid synaptic plasticity contributes to a learned conjunctive code of position and choice-related information in the hippocampus. Neuron 110:96–108.e4.

Zheng ZS, Huszár R, Hainmueller T, Bartos M, Williams AH, Buzsáki G (2024) Perpetual step-like restructuring of hippocampal circuit dynamics. Cell Rep 43:114702.