# The temporal paradox of Hebbian learning and homeostatic plasticity

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## Abstract

Hebbian plasticity, a synaptic mechanism which detects and amplifies co-activity between neurons, is considered a key ingredient underlying learning and memory in the brain. However, Hebbian plasticity alone is unstable, leading to runaway neuronal activity, and therefore requires stabilization by additional compensatory processes. Traditionally, a diversity of homeostatic plasticity phenomena found in neural circuits are thought to play this role. However, recent modelling work suggests that the slow evolution of homeostatic plasticity, as observed in experiments, is insufficient to prevent instabilities originating from Hebbian plasticity. To remedy this situation, we suggest that homeostatic plasticity is complemented by additional rapid compensatory processes, which rapidly stabilize neuronal activity on short timescales.

*Keywords:* synaptic plasticity, Hebbian plasticity, homeostatic plasticity, rapid compensatory processes

## Introduction

More than half a century ago, Donald Hebb [1] laid down an enticing framework for the neurobiological basis of learning, which can be succinctly summarized in the well-known mantra, "neurons that fire together wire together" [2]. However, such dynamics suffers from two inherent problems. First, Hebbian learning exhibits a positive feedback instability: those neurons that wire together will fire together more, leading to even stronger connectivity. Second, such dynamics alone would lead to all neurons in a recurrent circuit wiring together, precluding the possibility of rich patterns of variation in synaptic strength that can encode, through learning, the rich structure of experience. Two fundamental ingredients required to solve these problems are stabilization [3], which prevents runaway neural activity, and competition [4–6], in which the strengthening of a synapse may come at the expense of the weakening of others.

In theoretical models, competition and stability are often achieved by augmenting Hebbian plasticity with additional constraints [3, 5, 7]. Such constraints are typically implemented by imposing upper limits on individual synaptic strengths, and by enforcing some constraint on biophysical variables, for example, the
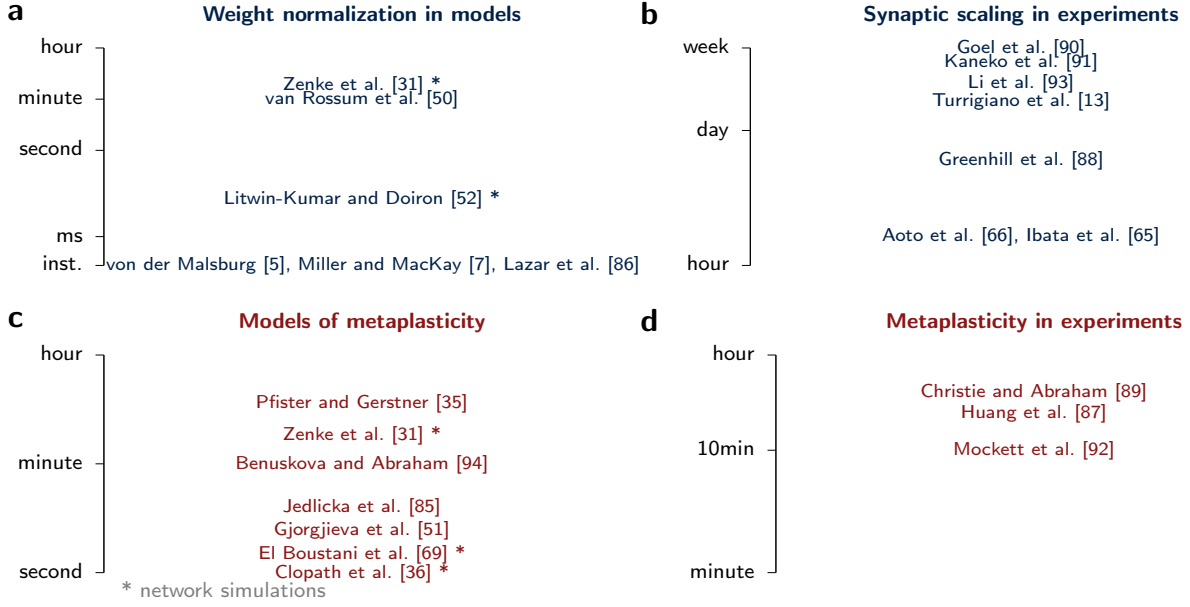
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Figure 1: **Comparison of the typical timescale of different forms of homeostatic plasticity in models and in experiments.** (a) Weight normalization in models. Here we plot the characteristic timescale on which synaptic weights are either normalized or scaled. (b) Synaptic scaling in experiments. Here we plot the typical time at which synaptic scaling is observed. (c) Models of metaplasticity. We plot the characteristic timescale on which the learning rule changes. (d) Metaplasticity in experiments. Here we show the typical timescale at which metaplasticity is observed.

total synaptic strength or average neuronal activity [6–12]. In neurobiology, forms of plasticity exist which seemingly enforce such limits or constraints through synaptic scaling in response to firing rate perturbations [13, 14], or through stabilizing adjustments of the properties of plasticity in response to the recent synaptic history, a phenomenon known as homeostatic metaplasticity [6, 11, 15, 16]. Overall, synaptic scaling and metaplasticity, as special cases of homeostatic mechanisms that operate over diverse spatiotemporal scales across neurobiology [17–22], are considered key ingredients that contribute both stability and competition to Hebbian plasticity by directly affecting the fate of synaptic strength.

The defining characteristic of homeostatic plasticity is that it drives synaptic strengths so as to ensure a homeostatic set point [42, 43], such as a specific neuronal firing rate or membrane potential. However, it is important that this constraint is implemented only on average, over long timescales, thereby allowing neuronal activity to fluctuate on shorter timescales, so that these neuronal activity fluctuations, which drive learning through Hebbian plasticity, can indeed reflect the structure of ongoing experience. This requisite separation of timescales is indeed observed experimentally; forms of Hebbian plasticity can be induced on the timescale of seconds to minutes [44–47], whilst most forms of homeostatic synaptic plasticity operate over hours or days [14, 43, 48]. This separation of timescales, however, raises a temporal paradox: homeostatic

plasticity then may become too slow to stabilize the fast positive feedback instability of Hebbian learning. Indeed modeling studies that have attempted to use homeostatic plasticity mechanisms to stabilize Hebbian learning [11, 28, 38, 49, 50] were typically required to speed up homeostatic plasticity to timescales that are orders of magnitude faster than those observed in experiments (Fig. 1).

This temporal paradox could have two potential resolutions. First, the timescale of Hebbian plasticity, as captured by recent plasticity models fit directly to data from slice experiments [25, 30, 47, 51, 52], may overestimate the rate of plasticity that actually occurs *in-vivo*. This overestimate could arise from differences in slice and *in-vivo* preps, or because complex nonlinear synaptic dynamics, both present in biological synapses and useful in learning and memory [53–55], are missing in most, but not all [56, 57], data-driven models. While slow plasticity may be a realistic possibility in cortical areas exhibiting plastic changes over days [58, 59], it may not be a realistic resolution in other areas, like the hippocampus, which must rapidly encode new episodic information [60, 61]. The second potential resolution to the paradoxical separation of timescales between Hebbian and homeostatic plasticity may be the existence of as yet unidentified rapid compensatory processes (RCPs) that stabilize Hebbian learning. Below, we explore both the theoretical utility and potential neurobiological instantiations of these putative RCPs.

## The temporal paradox of Hebbian and homeostatic plasticity

To understand the theoretical necessity for RCPs to stabilize Hebbian plasticity, it is useful to view a diversity of synaptic learning models through the unifying lens of control theory (Fig. 2). Here we can view the "fire together, wire together" interplay of neuronal activity and Hebbian synaptic plasticity as an unstable dynamical system. Also, we can view a compensatory process as a feedback controller that observes some aspect of either neuronal activity or synaptic strength, and uses this observation to compute a feedback control signal which then directly affects synaptic strength so as to stabilize global circuit activity. Indeed homeostatic plasticity is often thought of as a negative feedback control process [42, 43, 62, 63]. In general, the delay in any feedback control loop must be fast relative to the time-scale over which the unstable system exhibits run-away activity [64]. If the loop is slightly slow, the run-away will start before the stabilizing feedback arrives, generating oscillations within the system. In some cases, if the loop is even slower, the unstable runaway process might escape before stabilization is even possible.

Such oscillations and run-away are demonstrated in Fig. 3 for several compensatory mechanisms with feedback timescales that are chosen to be too slow, including the Bienenstock-Cooper-Munro (BCM) rule [6], and triplet spike-timing-dependent plasticity (STDP) [30] with either synaptic scaling [13, 29] or a metaplastic sliding threshold, as stabilizing controllers. For example, the BCM rule (Fig. 3a) can be thought of as a feedback control system where the controller observes a recent average of the postsynaptic output firing rate of a neuron, and uses this information to control both the sign and amplitude of associative plasticity; if
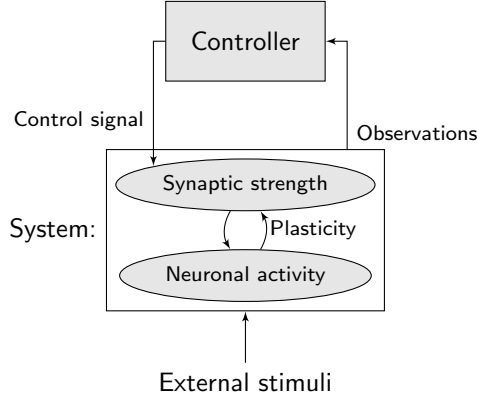
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Figure 2: **A control theoretic view of homeostatic plasticity and Hebbian learning.** The coupled dynamics of Hebbian plasticity and neural activity constitutes an unstable dynamical system. Homeostatic plasticity, and more generally any compensatory mechanism, can be viewed as a controller that observes aspects of neuronal activity and synaptic strengths, and uses these observations to compute a feedback control signal that acts on synaptic dynamics so as to stabilize circuit properties.

the recent average is high (low), plasticity is modulated to be anti-Hebbian (Hebbian). However, to achieve stability, the BCM controller must average recent output-activity over a short enough time-scale to modulate plasticity before this activity itself runs away (Fig. 3a; [11, 28, 38]). This result is not limited to BCM like rate models, but applies equally to STDP models which rely on similar metaplasticity processes to ensure stability ( Fig. 3bc; [25, 27, 28, 30]). These modified STDP rules can be thought of as employing feedback controllers which also observe a recent average of output firing rate, but use this information to modulate the STDP window, thereby stabilizing the system by changing relative rates of long-term potentiation (LTP) and long-term depression (LTD).

Another class of models relies on re-normalization of afferent synaptic weights to stabilize Hebbian plasticity. We distinguish between models with instantaneous algorithmic re-normalization of the weights [4, 5, 7, 24] and models which proportionally scale synaptic weights in an activity dependent way [8, 28, 65]. In the former case the controller observes total synaptic strength and uses this information to adjust synapses to keep this total strength constant. In the latter case, the controller observes neuronal firing rate, or recent average thereof, and uses this information to proportionally adjust afferent synaptic weights to enforce either a specified target output, or a total synaptic strength. Just as in the case of metaplasticity discussed above, the temporal average of the activity sensor has to be computed over a short timescale, related to the timescale over synaptic strengths and neuronal activity change, to ensure stability (Fig. 3d [28, 50]). Moreover, the rate at which the synaptic scaling process itself causes synaptic strengths to scale must be finely tuned to a narrow parameter regime that is neither too fast nor too slow [28, 29, 50]; if too slow, then stabilization is not possible, while if too fast, the stabilization process overshoots, causing oscillations.

Finally, some STDP models can be intrinsically stable, especially in feedforward circuits. One example are pair-based STDP models in which the integral of the STDP window is slightly biased toward depression. When weights are additionally limited by hard bounds, this can lead to bimodal weight distributions and firing rate stabilization [9, 66]. However, these learning rules typically require fine-tuning and become unstable when input correlations are non-negligible. Other stable models arise from a weight dependence
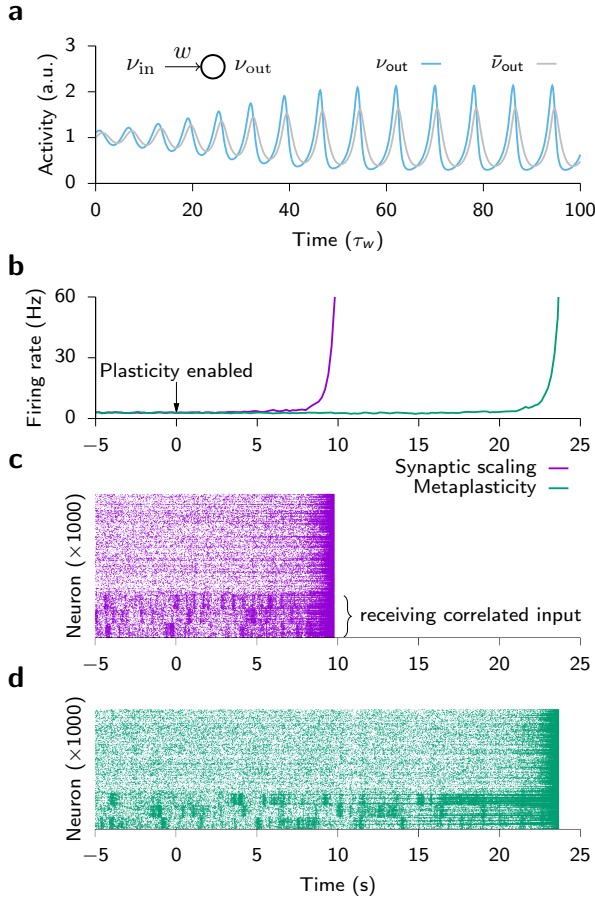
Figure 3: **Instability in different plasticity models.** (a) Unstable oscillations in the BCM model for a simple feed-forward circuit [6, 11]. Model: $\tau_w \frac{dw}{dt} = \alpha \nu_{\mathrm{in}} \nu_{\mathrm{out}} (\nu_{\mathrm{out}} - \beta \bar{\nu}_{\mathrm{out}}^2)$, $\tau_c \frac{d}{dt} \bar{\nu}_{\mathrm{out}} = \nu_{\mathrm{out}} - \bar{\nu}_{\mathrm{out}}$ with $\nu_{\mathrm{out}} = w \nu_{\mathrm{in}}$, $\tau_w = 0.9 \tau_c$, $\nu_{\mathrm{in}} \equiv 1$ in arbitrary units (a.u.) and $\alpha$ and $\beta$ are dimensionful scalar constants that ensure correct units; we take simply $\alpha = \beta = 1$. Here $\bar{\nu}_{\mathrm{out}}$ can be thought of as the observation of a controller, corresponding to an average of output neuronal activity $\nu_{\mathrm{out}}$ over time-scale $\tau_c$. Moreover, the multiplicative term in parenthesis in the weight dynamics can be interpreted as a control signal that modulates both the sign and amplitude of associative plasticity, dictating a stabilizing anti-Hebbian rule if the recent average $\bar{\nu}_{\mathrm{out}}$ is too large. If the control dynamics $\tau_c$ is too slow relative to the synaptic plasticity dynamics $\tau_w$, unstable oscillations arise. (b–d) Runaway activity in a recurrent neural network simulation consisting of 25000 excitatory and inhibitory integrate-and-fire neurons and plastic excitatory synapses using a minimal triplet STDP model [30] with different homeostatic mechanisms such as sliding threshold metaplasticity (violet) and synaptic scaling (green), both described in detail in [28]. (b) Population firing rate as a function of time. (c,d) Raster plot of spiking activity. The bottom 3 groups of 100 neurons received rate modulated spiking input with 100ms correlation time constant to emulate sensory input to a small set of neurons. The timescale of sliding threshold metaplasticity was $\tau_c = 3$mins. The timescale for the rate detector and the scaling dynamics for synaptic scaling were $\tau_c = 10$s and $\tau_{\mathrm{scl}} = 1$h respectively. Because of these slow stabilization dynamics, the fast interplay between Hebbian plasticity and recurrent network dynamics leads to rapid population firing rate destabilization within 10 to 20 seconds for both learning rules.

in the learning rule such that high (low) synaptic strength makes LTP (LTD) weaker [29, 67–71]. Such a stabilizing weight dependence is advantageous because it can lead to more plausible unimodal weight distributions as observed empirically [69, 71]. However, the unimodal weight distribution, by precluding multi-stability in the configurations of synaptic strengths, typically leads to the rapid erasure of synaptic memory traces in the presence of background activity driving plasticity [70, 72]. Moreover, when such weight dependent synaptic learning rules are embedded in a recurrent neuronal circuit without any additional control mechanisms, they can succumb to runaway neural activity as experience dependent neural correlations emerge in the recurrent circuit [70, 73, 74].

In summary, empirical findings and control-theoretic considerations suggest that compensatory mechanisms capable of stabilizing Hebbian plasticity must operate on tightly constrained timescales. In practice, such compensatory mechanisms must act on similar or even faster timescales than Hebbian plasticity itself [24, 25, 27, 28, 30, 65]. STDP, as one of the most common manifestations of Hebbian plasticity in the brain, can be induced in a matter of seconds to minutes [44–46, 75, 76]. Homeostatic plasticity, on the other hand, acts on much longer timescales of hours to days [14, 16, 19, 32, 33, 48, 77]. This separation of timescales poses a temporal paradox as it renders most data-driven STDP models unstable [3, 78]. In spiking network models, this instability has severe consequences (Fig. 3b–d); it precludes the emergence of stable synaptic structures or memory engrams [28, 70, 73], unless the underlying plasticity models are augmented by RCPs [24, 26, 28, 65, 79]. Overall, these considerations suggest that one or more RCPs exist in neurobiological systems, which are missing in current plasticity models.

**Putative rapid compensatory processes**

What putative RCPs could augment Hebbian plasticity with the requisite stability and competition? Here we focus on several possibilities, operating at either the network, the neuronal or the dendritic level (Fig. 4). However, we note that these possibilities are by no means exhaustive.

At the network level, recurrent or feedforward synaptic inhibition could influence and potentially stabilize plasticity at excitatory synapses directly. For instance, [80] demonstrated via dynamic-clamp that a simulated increase in total excitatory and inhibitory background conductance, which could originate from elevated levels of network activity, rapidly reduces the amplitude of LTP, but not LTD, of the STDP window. This rapid effect may be mediated through changes in calcium dynamics in dendritic spines and could constitute an RCP. Another study [81] showed that global inhibition in a rate-based network model is sufficient to stabilize plasticity at excitatory synapses with a sliding presynaptic and fixed postsynaptic plasticity threshold. Other modeling studies have suggested a role for inhibitory synaptic plasticity (ISP) [82, 83], instead of non-plastic inhibition, in stabilizing Hebbian plasticity. It has been suggested, for instance, that ISP in conjunction with a fixed plasticity threshold at the excitatory synapse could have a similar effect as the sliding threshold in
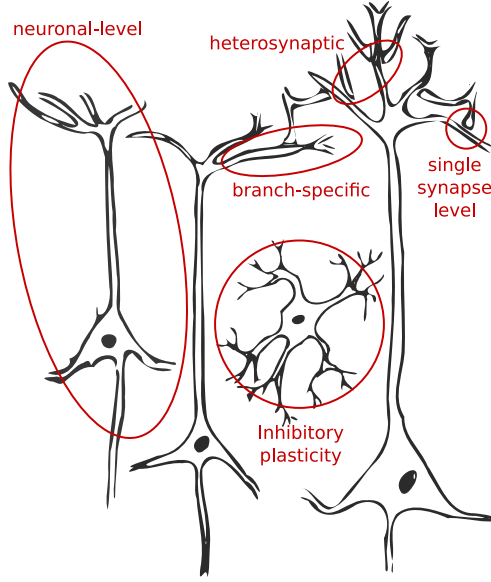
Figure 4: Schematic representation of the many potential loci of rapid compensatory processes.

the BCM model [84]. Finally, in some experiments excitatory and inhibitory plasticity are not integrated as independent events, but can influence each other. For instance, there are cases in which induction of ISP alone can flip the sign of subsequent plasticity at excitatory synapses [85]. While inhibition and ISP may constitute RCPs, a clear picture of how these elements tie together has not yet emerged. Further experimental and theoretical work is required to understand their potential for acting as RCPs.

A more well studied possibility for an RCP is heterosynaptic plasticity, which operates at the level of individual neurons or potentially dendritic branches. Heterosynaptic plasticity refers to a non input-specific change at *other* synapses onto a neuron that are not directly activated ([49, 86–89]). Its viability as putative RCP arises from the fact that some forms of heterosynaptic plasticity can be induced rapidly, and moreover, similar to synaptic scaling, can show aspects of weight normalization [87]. For instance, a rapid form of heterosynaptic plasticity, in which neuronal bursting causes bi-directional weight-dependent changes in afferent synapses, has been observed recently [88, 90]. While the observed weight-dependence is reminiscent of Oja's rule [8], as strong synapses weaken, it is not identical because weak synapses can also strengthen. Nevertheless, this form of heterosynaptic plasticity has been shown to prevent runaway of LTP in models of feedforward circuits [49] and has been demonstrated to co-occur with Hebbian plasticity in experiments [90].

Recently, the utility in stabilizing runaway LTP has also been demonstrated in a recurrent network model of spiking neurons [79], in which a similarly burst dependent form of heterosynaptic plasticity is crucial to ensure stable formation and recall of Hebbian cell assemblies. Moreover, the work suggests a potential role of heterosynaptic plasticity in triggering the reversal of LTP and LTD [91].

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At the level of dendritic branches, a recently described form of structural heterosynaptic plasticity [89], involves local dendritic competition between synapses. Specifically, glutamate induced structural synaptic potentiation of a set of clustered dendritic spines causes shrinkage of nearby, but unstimulated spines. Interestingly, even when structural potentiation was switched off through inhibition of $Ca^{2+}$/calmodulin-dependent protein kinase II (CaMKII), the heterosynaptic effect persists, suggesting a model in which spines send and receive shrinkage signals instead of competing for limited resources. Moreover, the observed dendritic locality is consistent with work on local or branch specific conservation of total synaptic conductance [87, 92].

Finally, in some cases heterosynaptic plasticity might not actually be heterosynaptic, as it may still depend on low levels of spontaneous synaptic activity in unstimulated synapses [93]. A recent biophysical model derived from synaptic plasticity data [94] actually requires such low-levels of activity to match the data. Interestingly, this model suggests such "heterosynaptic" plasticity could arise as a consequence of a rapid (timescale $\sim 12s$) homeostatic sliding threshold possibly related to autophosphorylation of CaMKII.

While heterosynaptic plasticity, like synaptic scaling, can have a stabilizing effect, it is distinct from synaptic scaling in two ways. First, heterosynaptic plasticity need not multiplicatively scale all weights in the same manner. Second, it is unclear whether heterosynaptic plasticity in general drives neuronal activity variables to a specific set point, like synaptic scaling does [43]. Thus, while heterosynaptic plasticity has the rapidity to act as a putative RCP, its functional utility in storing memories requires further empirical and theoretical study, as it may lack the ability to precisely preserve ratios of synaptic strengths. However, its clear functional utility in preventing instability (Fig. 3d), along with even an approximate preservation in ratios of strengths, could potentially endow the interaction of heterosynaptic plasticity and Hebbian learning with the ability to stably learn and remember memories. Further network modeling, building on promising heterosynaptic plasticity models [49, 79, 94], could be highly instructive in elucidating the precise properties, beyond rapidity, a putative RCP must obey in order to provide appropriate competition and stability to Hebbian plasticity.

**Conclusion**

The trinity of Hebbian plasticity, competition and stability are presumed to be crucial for effective learning and memory. However, a detailed theoretical and empirical understanding of how these diverse elements conspire to functionally shape neurobiological circuits is still missing. Here we have focused on one striking difference between existing models and neurobiology: the paradoxical separation of timescales between Hebbian and homeostatic plasticity. In models, such a separation of timescales typically leads to instability, unless plasticity is constrained by RCPs that act much faster than observed forms of homeostatic plasticity. In principle, RCPs could be implemented at various spatial scales. Here we have discussed

different forms of heterosynaptic plasticity and processes involving synaptic inhibition as possible candidates. However, identifying the key neurobiological processes that provide stability and competition to Hebbian learning rules remains an important direction for future research.

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**Papers of special interest**

1. [4] (●): Of historical interest as probably the earliest modeling study — the authors show that Hebbian LTP needs to be complemented by two mechanisms: (i) LTD formulated as an early version of a covariance rule and (ii) and a rapid heterosynaptic mechanism formulated as normalization of the sum over all weights of afferent synapses.

2. [6] (●): This classic modeling paper for metaplasticity via a sliding threshold between LTP and LTD links thorough mathematical analysis to experimental data available at that time.

3. [7] (●): A careful and transparent mathematical study on the role of synaptic constraints in Hebbian learning.

4. [43] (●): A broad review of synaptic homeostatic plasticity which also addresses open questions regarding timescales of plasticity and homeostasis.

5. [28] (●): Mathematical analysis of network dynamics in rate neurons indicating the need for RCP implemented through either weight normalization or metaplastic sliding threshold.

6. [38] (●): The authors show that the time course drop of synaptic strength observed in ocular deprivation experiments cannot be captured by a standard BCM model. Instead the authors suggest a model in which plasticity is intrinsically bistable whereas homeostasis is multiplicative.

7. [25] (●): Voltage-based plasticity model with parameters extracted from experimental data (see also [94]).

8. [24] (●): This article demonstrates stable learning and transient recall ($\sim$ 100ms) of Hebbian cell assemblies in a recurrent network model through the combination of a plausible model of STDP with two homeostatic processes: a fast one that is implemented as a renormalization of the sum of all synaptic weights onto a given neuron every 20ms, plus a rapid firing rate homeostasis (FRH) implemented by plasticity ISP following a model by [82] (see also [79]).

9. [79] (●) Here the authors demonstrate stable learning and recall of Hebbian cell assemblies in a recurrent network model by combining a plausible STDP model with heterosynaptic and transmitter-induced plasticity which both act as RCPs to prevents runaway activity (see also [24]).

10. [88] (●): A comprehensive review of heterosynaptic plasticity in experiments and its putative role as a RCP.

11. [89] (●): This article shows that when a cluster of spines is stimulated through glutamate uncaging, which under control conditions leads to structural growth which is presumably related to Hebbian plasticity, nearby unstimulated spines shrink which corresponds to heterosynaptic depression.

12. [90] (●): A recent experimental study with additional modelling demonstrating that heterosynaptic plasticity co-occurs with input-specific homosynaptic plasticity and can effectively stabilize runaway LTP.

13. [94] (•): A biophysical plasticity model is presented and fitted to experimental data. Interestingly, best fits are obtained for parameters which are suggestive of a rapid ($\sim 10$s) sliding threshold mechanism reminiscent of the BCM model.