Unsupervised Learning of Precise Spike Times with Membrane Potential Dependent Synaptic Plasticity
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Abstract
Precise spatio-temporal patterns of neuronal action potentials underly e.g. sensory representations and control of muscle activities. It is, however, not known how the synaptic efficacies in the neuronal networks of the brain adapt such that they can reliably generate spikes at specific points in time. Known activity dependent synaptic plasticity rules are agnostic to the goal of learning spike times, while the existing formal and supervised methods are barely biologically plausible. Here, we propose a simple unsupervised synaptic plasticity mechanism that depends on the postsynaptic membrane potential and overcomes shortcomings of previous rules. It is derived from the basic requirement of membrane potential balance and is supported by experiments. The voltage is a global signal that makes graded and precise information about the state of the neuron available locally at the synapse. This allows plasticity to terminate when a desired state is achieved. This feature of the proposed synaptic mechanism extends the theoretical principles underlying the classical Perceptron Learning Rule to realistic spiking feed-forward networks. In particular, the sensitivity of the proposed plasticity mechanism to the membrane potential allows to introduce an adjustable margin, which makes the networks’ output robust against noise. Furthermore, the stereotypic dynamics of the membrane potential close to action potentials causes a sensitivity of synaptic change to pre- and postsynaptic spike times which can reproduce anti-Hebbian forms of spike timing dependent plasticity. For spatio-temporal input spike patterns our conceptually elementary plasticity rule achieves a surprisingly high storage capacity for spike associations, with robust memory retrieval even in the presence of input activity corrupted by noise.

Author Summary
A central hypothesis in neuroscience is that learning is ultimately based on synaptic changes. However, there still is a wide gap between the plethora of experimental findings on synaptic plasticity and theoretical models attempting to explain how neuronal systems can realistically absorb information. All learning rules which have been proposed for the learning of exact spike times rely on a hypothetical supervisor for which there is no known neural correlate. We bridge this gap by introducing a plasticity mechanism that relies on the neuronal membrane potential as a global signal controlling local synaptic plasticity. Despite its simplicity the proposed mechanism is sufficient to unify the Perceptron and the Chronotron, two fundamental theoretical concepts of learning. The mechanism works in a purely associative manner, and can achieve close to maximal memory capacity for Chronotrons, without requiring supervisory mechanisms that monitor the progress of learning in more artificial rules.

Introduction
Experiments show that exact spike times down to the millisecond scale are important for some computations in the brain (see \cite{1,2} for reviews). In the salamander retina, for example, information about a stimulus is reliably encoded in the relative latency of spike times across different neurons \cite{3}. In simultaneous recordings, absolute latency fluctuations covaried between neurons. This implies that information is contained in precise spatio-temporal firing patterns, which also explains how information can be coded in relative latencies without having knowledge about stimulus onsets \cite{1,4}. Another study showed that the
somatosensory cortex in rats can learn to discriminate between simultaneous and time-ordered electrical stimulation in two separate neurons if the difference in stimulation onset is as low as one millisecond [5]. These experimental results show that there are brain areas which perform computations with spike timing codes, and can adapt to new computations. However, there is a lack of biophysically plausible synaptic plasticity rules which might achieve the needed adjustments.

The simplest conceivable model of a system which learns to reproduce exact spike times is a spiking one-layered feed-forward network, sometimes called “Chronotron” [6]. A postsynaptic neuron is trained to reproduce desired spike trains with sub-millisecond precision whenever one particular stimulus out of a fixed set of stimuli is presented. There are a number of learning rules for this task [6–9]. All of them depend on a hypothetical supervisor who compares the networks’ output to a target and changes weights accordingly. However, there is no known neural correlate for this supervisor. It would have to generate feedback signals with high temporal precision, which conflicts with retrospective evaluation of activity usually done by these supervisors. On the other hand, Spike-Timing-Dependent Plasticity (STDP, [10–13]) is an activity dependent plasticity rule which could provide the necessary temporal precision. But it is unable to fill in missing spikes and reinforces all existing spikes whether they are correct or not [14].

It is well known that the postsynaptic membrane potential plays a role in synaptic plasticity [15–17]. NMDA receptors at the postsynaptic side act as coincidence detectors which allow calcium influx when transmitters bind during postsynaptic depolarization. STDP rules have been shown to depend on the voltage around the time of the postsynaptic spike [15]. It was also shown that pairing presynaptic spikes only with postsynaptic depolarization (but without postsynaptic spiking!) leads to timing-dependent plasticity [18, 19].

Here, we derive a Membrane Potential Dependent Plasticity rule (MPDP) from a balance requirement. Synapses change in order to keep the voltage bounded, which results in Anti-Hebbian plasticity: When the postsynaptic neuron is hyperpolarized, concurrently active synapses potentiate, and vice versa. Although not as ubiquitous, Anti-Hebbian STDP in excitatory synapses has been reported [20]. Note that Hebbian STDP of inhibitory synapses is in effect equal to Anti-Hebbian STDP in excitatory synapses [21]. Curiously, one study reports synaptic potentiation for synapses that are active during postsynaptic hyperpolarization depending on calcium-permeable AMPA receptors [22]. The sensitivity of MPDP to the exact time course of the voltage around a spike allows the synapse to assess the progress of learning, which we use to emulate supervised learning rules in two fundamental learning tasks. We map MPDP to the Perceptron Learning Rule in an almost perfect match. Furthermore, we evaluate the capability of MPDP in the Chronotron task by comparing the attainable memory capacity of MPDP to a range of supervised learning rules. We find that the capacity achieved by MPDP is half of the known maximum value. Also, MPDP comes with an adjustable robustness against noise, which guarantees recall of activity patterns even in the presence of noise in the input.

Materials and Methods

Neuron and network model

We investigate plasticity processes in a simple single-layered feed-forward network with N (presynaptic) input neurons and one (postsynaptic) output neuron (see Fig. 1). The input neurons fire either in purely spatial (Perceptron case) or in spatio-temporal spike patterns (Chronotron). The patterns are generated stochastically and kept fixed for a single network (frozen noise), except in noise conditions for which spike times are jittered. We denote the time of the k-th spike of presynaptic neuron with index i as $t_{k}^{i}$.

The postsynaptic neuron is modeled as a leaky integrate-and-fire (LIF) neuron. We use the formulation of the spike response model (SRM0) [23], since this simplifies the formulation of the plasticity rule. The membrane potential at time t is given by the weighted sum of postsynaptic potentials (PSPs), the sum
of refractory kernels due to postsynaptic spikes, and the filtered input current $I_{ext}(t)$:

$$V(t) = \sum_i w_i \sum_k \varepsilon(t - t^*_k) + \sum_{t_{post}} R(t - t_{post}) + \int_0^\infty \kappa(t - s) I_{ext}(s) ds$$  \hspace{1cm} (1)

with passive response kernel $\kappa = \exp\left(-\frac{(t - s)}{\tau_m}\right)$. The equation implies that in the absence of any input the neuron settles into an equilibrium potential of $V_{eq} = 0$. $\varepsilon(s)$ is the PSP kernel: Any spike in a presynaptic neuron leads to a stereotyped time course of the deflection of the postsynaptic membrane potential with amplitude proportional to the synaptic weight. Throughout this study we use

$$\varepsilon(s) = \Theta(s) \frac{1}{\tau_m - \tau_s} \left[ \exp\left(-\frac{s}{\tau_m}\right) - \exp\left(-\frac{s}{\tau_s}\right) \right],$$  \hspace{1cm} (2)

where $\Theta(s)$ is the Heaviside step function, and $\tau_m$ and $\tau_s$ are the time constants of the membrane potential and synaptic currents, respectively. The factor normalizes the PSP to unit area. Whenever the membrane potential reaches the spiking threshold $V_{thr}$ at time $t_{post}$, a postsynaptic spike is registered, and a reset kernel $R(t_{post})$ is added to the membrane potential. The reset kernel makes sure that the membrane is set to reset potential $V_{reset}$, from where it relaxes back to equilibrium. We use two different kernels. The first one is a kernel where the membrane potential gets reset to $V_{eq}$, and the neuron undergoes hyperpolarization with a time course equivalent to a PSP:

$$R_p(s) = -\Theta(s) \left[ V_{thr} \exp\left(-\frac{s}{\tau_m}\right) + \lambda \varepsilon(s) \right].$$  \hspace{1cm} (3)

$\lambda > 0$ is a parameter which controls the magnitude of the hyperpolarization. The second one is a kernel where the neuron gets reset to $V_{reset} < 0$, from where it passively relaxes back to $V_{eq}$:

$$R_c(s) = \Theta(s) (V_{reset} - V_{thr}) \exp\left(-\frac{s}{\tau_m}\right).$$  \hspace{1cm} (4)

We use $R_p(s)$ in perceptron training and $R_c(s)$ in chronotron training with MPDP.

![Figure 1](image-url) **Figure 1. Network and neuron model.** The network has a simple feed-forward structure. All presynaptic inputs converge onto a single postsynaptic leaky integrate-and-fire neuron. Shown is the membrane potential in response to the shown presynaptic input pattern and the weights. Line widths of inputs are roughly proportional to synaptic weight.
Derivation of plasticity rule

For the formal derivation of Membrane Potential Dependent Plasticity (MPDP), we introduce an objective for the postsynaptic neuron: Its membrane potential should be kept between two bounds at all times.

We formalize this by defining an error function:

\[ E(t) = \gamma (|V(t) - \vartheta_D|_+)^\nu + (|\vartheta_P - V(t)|_+)^\nu, \tag{5} \]

where we introduce two thresholds \( \vartheta_D \) and \( \vartheta_P \) with \( V_{\text{thr}} > \vartheta_D > \vartheta_P \). \( \gamma \) is a factor which scales depression and potentiation relative to each other, and \( \nu \in \{1, 2\} \) determines the impact of large deviations compared to small ones. \([x]_+\) is the positive rectifying bracket, i.e. \([x]_+ = x \) if \( x > 0 \) and zero otherwise. The error function is greater than zero each time the membrane potential is greater than \( \vartheta_D \) or lower than \( \vartheta_P \).

Throughout this study, we set \( \vartheta_P \) either to be zero or negative with very small magnitude.

From this error function, we compute a gradient descent rule by taking the partial derivative with respect to weight \( w_i \):

\[ \frac{dw_i}{dt} = \frac{\eta \partial E(t)}{\nu \partial w_i} = \eta \sum_k \varepsilon(t - t_k^i) \cdot \begin{cases} (\Theta(\vartheta_P - V(t)) - \gamma \Theta(V(t) - \vartheta_D)) & \text{if } \nu = 1 \\ (|\vartheta_P - V(t)|_+ - \gamma |V(t) - \vartheta_D|_+) & \text{if } \nu = 2 \end{cases}, \tag{6} \]

where \( \eta \) is the learning rate. The effect of this rule on the membrane potential is demonstrated in Fig. 2.

For simplicity, in this study synapses can change their sign due to weight change; we do not differentiate between inhibitory and excitatory synapse types.

Figure 2. Illustration of Anti-Hebbian Membrane Potential Dependent Plasticity (MPDP). A LIF neuron is presented twice with the same presynaptic input pattern. Excitation never gets strong enough to cause output spikes (\( V_{\text{thr}} \): Firing threshold). Nevertheless, through the action of MPDP synapses get potentiated, reducing hyperpolarization (green trace; arrow up) and depolarization (arrow down) on the second presentation. For times where \( V(t) \) stays within the bounds, synapses remain unchanged, leaving the membrane potential as it was before.
Perceptron setup

A Perceptron is a feed-forward neural network and serves as a simple model of memory. Commonly, input and output neurons are simple binary rate neurons \[23\]. This is easy to translate to spiking neurons: In a presentation of an input pattern, the state of binary neuron \( i \) sets the occurrence of a spike in the respective spiking neuron \( i \) at time \( t = 0 \). Therefore, to implement a perceptron with spiking neurons, we generate \( P = \alpha N \) different binary patterns \( \{x_i^\mu\} = \vec{x}^\mu \), where \( \alpha \) is the load parameter. If \( x_i^\mu = 1 \), neuron \( i \) will spike in pattern \( \mu \in \{1, 2, \ldots, P\} \). Else if \( x_i^\mu = 0 \), neuron \( i \) stays silent in this pattern. We set the probability that neuron \( i \) spikes in pattern \( \mu \) independent of other neurons and other patterns, and the probability of spiking is \( p(x_i^\mu = 1) = 1/2 \). With each pattern \( \vec{x}^\mu \) we associate a desired output \( y^\mu \in \{0, 1\} \) of the postsynaptic neuron, which is again chosen randomly with probability 1/2. The neuronal dynamics are given by Eqn. 1 with \( R(s) = R_p(s) \). The plasticity rule is Eqn. 6 with \( \nu = 1 \).

Training of the perceptron is done in batch mode. In a learning block, each of the \( P \) patterns is presented exactly once, with the order of presentation randomized in every learning block. The weight changes are applied after each presentation. A presentation of a pattern \( \mu \) consists of a volley of synchronous spikes in neurons \( i \) with \( x_i^\mu = 1 \). If \( y^\mu = 1 \), an external delta-shaped suprathreshold current is injected into the postsynaptic neuron such that it spikes exactly at the same time as the presynaptic volley. If \( y^\mu = 0 \), the postsynaptic neuron is not excited externally. After each learning block, we test the recall of the trained associations. Each presynaptic pattern is presented again, but this time without any external stimulation. If learning was successful, the postsynaptic neuron will spike in response to patterns with \( y^\mu = 1 \) and stay silent otherwise without teacher intervention.

To test the capacity of the network and its scaling behavior, we generate networks of different sizes \( N \), and for each network size we generate different numbers of patterns \( P = \alpha N \). For each combination of \( N \) and \( \alpha \), we generate 30 different pseudorandom network realizations and train them for a fixed number of learning blocks. Recall is tested after each learning block by presenting each pattern and comparing the neurons’ output (spike or not) with the desired output. Visual inspection of recall success showed that learning has converged in any combination of parameters.

Chronotron setup

The Chronotron \[6\] is the basic model of a network which can be trained to fire precisely timed spikes. The postsynaptic neuron “sees” a limited set of input patterns and during learning it is externally forced to fire at an exact time relative to the onset of each pattern specific to it. For comparability, the setup we use is very similar to the one used by Florian \[6\]. Each pattern \( \mu \) consists of exactly one spike in each presynaptic neuron, and the spike times are randomly chosen from the interval \( t_i^\mu \in [0, T] \), independent of other patterns and other neurons. To each pattern we assign a desired output spike time \( t_d^\mu \in [\Delta_{edge}, (T - \Delta_{edge})] \). We set the length of the presentation to \( T = 200ms \) and \( \Delta_{edge} = 2\tau_m = 20ms \).

Because we start each trial with a neuron at rest, a postsynaptic spike can be only generated at a minimum time into the trial, therefore we restrict the possible output spike times to a smaller sub-interval within the presentation interval. We chose network sizes \( N \in \{200, 500, 1000, 2000\} \) with loads \( P/N = \alpha \in [0.01, 0.25] \). For each combination \( \{N, \alpha\} \) we generate 50 realizations and train the network for 10000 learning blocks. Recall is tested after each learning block by presenting each pattern again without the external input. Recall is counted as a success if the postsynaptic neuron produces exactly one output spike not further away than \( \vartheta_{CC} = 2ms \) from the desired time. We record the fraction of correctly recalled spikes and their average distance from the teacher as a function of learning blocks (time).

We define two measures of the capacity in terms of the load \( \alpha \). The critical load for perfect recall \( \alpha_{pr} \) is the load below which all output spikes have been learned correctly. Else, there is a fraction of patterns which do not generate the desired output spike. This definition is imperfect for two reasons. First, we sample the load in discrete steps, so the value of \( \alpha_{pr} \) provides a lower bound for the actual critical load.
Second, there are some conditions for which recall is never perfect, especially when input noise is involved. Therefore, we define a critical load with a less strict requirement. We plot the fraction of correct output spikes, i.e. spikes that lie in the window of size $2\theta_{CC}$ centered at $t^*_i$ as a function of the load; see Fig. 7 (A) for an example. The critical load $\alpha_{90}$ is the intersection of a line at 90 % correctly recalled spikes with the plotted line. The obtained values are linear approximations of the true value and because of the sigmoidal course of the capacity curves they are lower bounds, but closer to the respective critical loads than $\alpha_{pr}$.

Testing noise tolerance

In MPDP, the threshold for depression $\theta_D$ lies below the firing threshold. When learning has converged, the membrane potential stays below $\theta_D$ for all times except just before the desired output spike. This allows the trained network to tolerate noise-induced fluctuations of the membrane potential. We investigate the ability of networks to resist noise in the input under two conditions. In the first condition we test the network after training with noise-free input by applying jitter to the patterns during recall. The jittered spike times $\hat{t}_i^\mu$ are generated by adding gaussian noise with zero mean and standard deviation $\sigma$:

$$\hat{t}_i^\mu = t_i^\mu + \mathcal{N}(0, \sigma). \quad (7)$$

In the second condition we train the network with jittered input patterns and test recall with noise-free patterns. This condition evaluates the ability of the plasticity rule to extract common features of a distribution of inputs, i.e. generalization.

In jittered simulations we pad the simulated interval by 20 ms to prevent excessive loss of spikes. Spikes which due to jitter fall outside the interval $[-20, T+20]$ ms are dropped. We test a range of jitter variances, $\sigma \in \{0, 0.1, 0.2, 0.5, 1, 2, 5, 10\}$ ms.

We plot the fraction of correctly recalled spikes as a function of load $\alpha$ for a constant network size and different jitter levels $\sigma$. We then estimate the intersection of the graph with 90 % correct recall, which results in $\alpha_{90}$ as a function of jitter variance (see Figs. 8 and 9). For a given network size, we plot this function to track the deterioration of recall with noise.

Comparison to other learning rules

We compare the quantitative results of Chronotron learning with MPDP to the same task done with other learning rules. We chose ReSuMe [7], E-Learning [4] and FP-Learning [9]. ReSuMe is the prototypical learning rule for exact spike time learning. In each learning trial, for each output spike at time $t_{out}$ weights of presynaptic neurons $\{i\}$ that fired before it at times $t_i$ are depressed proportional to $\exp(- (t_{out} - t_i)/\tau_{plas})$. On the other hand, weights of all neurons which fired before the desired output spike times $t_d$ are potentiated proportional to $\exp(-(t_d - t_i)/\tau_{plas})$, with the same factor of proportionality as for depression. If every $t_{out} = t_d$, all weight changes exactly cancel and learning has converged. ReSuMe has convergence problems, though. Spike generation is strongly non-linear, as small changes in synaptic weights can make a spike appear or disappear. Even worse, a spurious spike distorts the membrane potential for later times, which hampers learning [9]. E-Learning was proposed to remedy these problems. It is based on ReSuMe and uses the PSP kernel (Eqn. 2) to calculate weight changes instead of an exponential decay kernel. The difference to ReSuMe is the handling of output spikes close to the desired time. For those, the learning rate decreases linearly with the time difference. This considerably smoothens convergence and improves capacity [6]. We found that ReSuMe with the PSP kernel yields worse capacity than with the exponential kernel (data not shown).

The last rule we discuss is Finite Precision (FP-) Learning. It was proposed recently as a heuristic rule. In each learning trial, this rule checks if for every desired output spike time $t_d$ there exists exactly one output spike within a window of finite size around $t_d$. If not, it finds the earliest mismatch of the trial. If the first mismatch is an unpaired desired output spike, weights of neurons which fired before
Here, $t_d$ are potentiated proportional to $\varepsilon(t_d - t_i)$ (PSP kernel). If the first mismatch is an output spike not within a desired window or an additional spike within a window, weights are depressed proportional to $\varepsilon(t_{out} - t_i)$. This treatment avoids the problems associated with the nonlinear spike generation, and it was shown that FP-Learning provides an upper bound of the capacity of the Chronotron which gets tighter as the window size is decreased [9]. In this study, we chose the window to be of 4 ms length to match $\vartheta_{CC}$.

We subject all plasticity rules to the same tests as MPDP, i.e. capacity, testing against noise during recall and noise during learning with noiseless recall.

### Numerical procedures

All simulations are done with custom made programs written in Matlab (Mathworks, Natick, MA). To speed up computations in the case of Chronotron networks with ReSuMe, E-Learning and FP-Learning, we use an event-based exact integration scheme [25]. This is possible because the learning rules only rely on spike times. For MPDP we implemented an Euler integration scheme with step size 0.1 ms.

Weights are always initialized randomly. The mean is chosen such that without spiking the average membrane potential would be 30 mV, well above the firing threshold. This tests the ability of a plasticity rule to extinguish spurious spikes.

The parameters like learning rates and thresholds we use are set by hand for all plasticity rules. Before doing the final simulations, we did a search in parameter space by hand to find combinations which yield high performance in the Chronotron task. We use the following parameters: The neurons’ parameters are $\tau_s = 3\, \text{ms}$, $\tau_m = 10\, \text{ms}$, and $V_{thr} = 20\, \text{mV}$. In the Chronotron, the reset kernel is $R_c(s)$ with $V_{reset} = -5\, \text{mV}$ in MPDP and $V_{reset} = 0\, \text{mV}$ for the other learning rules. The reset kernel in Perceptron learning is $R_p(s)$. $\lambda$ is determined by the desired margin against noise, see below. For MPDP we use $\vartheta_P = 18\, \text{mV}$, $\vartheta_D = 0\, \text{mV}$, $\gamma = 14$, and $\eta = 5 \cdot 10^{-4}$. With ReSuMe, we find $\tau_{plas} = 10\, \text{ms}$, and $\eta = \{10, 4, 2, 1\} \cdot 10^{-10}$ for 200, 500, 1000 and 2000 neurons as good parameters. For E-Learning we use $\eta = 5 \cdot 10^{-7}$, $\eta_r = 0.01$, $\tau_q = 5\, \text{ms}$. FP-Learning has only a single free parameter, the learning rate $\eta = 10^{-9}$.

### Results

In this study, we investigate the learning capabilities of a plasticity rule which relies only on postsynaptic membrane potential and presynaptic spikes as signals. For this we use a simple setup of a one-layered feed-forward network of $N$ input neurons and one LIF output neuron, see Fig. 1. The formulation of the MPDP rule used here is based on a balance requirement. Synaptic plasticity acts to keep the membrane potential bounded. We introduce two thresholds $\vartheta_P$ and $\vartheta_D$ expressed in millivolts with $\vartheta_P < \vartheta_D < V_{thr}$. The objective of the neuron is to keep its membrane potential between $\vartheta_P$ and $\vartheta_D$. From this objective we derive an activity-dependent plasticity rule: Whenever the membrane potential is below $\vartheta_P$, synapses of neurons which were active shortly before are potentiated, and depressed if they are above $\vartheta_D$. The weight change of a synapse is proportional to the overlap of the unit PSP with the duration the voltage is either below $\vartheta_P$ or above $\vartheta_D$. See Fig. 2 for the effects this plasticity rule exerts on the membrane potential.

In the following we will show that despite its Anti-Hebbian characteristic this plasticity rule is well equipped to associatively learn spike times, while providing robustness against input noise.

**Anti-Hebbian MPDP implements associative learning**

At first glance, it might seem unlikely that Anti-Hebbian plasticity can achieve associative learning. However, the neuronal dynamics shows stereotypic behavior before, during and after each spike. To
induce a spike, the neuron needs to be depolarized until active feed-back processes kick in. These processes cause a very short and strong depolarization and a subsequent undershoot of the membrane potential (hyperpolarization), from where it relaxes back to equilibrium.

We consider an idealized scenario to demonstrate the capability for associative learning. A population of presynaptic neurons fire one spike in each neuron at equidistant times. They have projections onto a single postsynaptic LIF neuron, and all weights are zero initially. In each training trial an external delta-shaped suprathreshold current is induced at the postsynaptic neuron at a fixed time relative to the onset of the input pattern (teacher spike). The postsynaptic neuron reaches its firing threshold instantaneously, spikes and undergoes reset into a hyperpolarized state (blue trace on the left in Fig. 3). This is mathematically equivalent to adding a reset kernel at the time of the external current \[ \theta_P \]. Because we set \[ \theta_P = V_{eq} = 0 \], potentiation is induced in all synapses which have overlap of their PSP-kernel with the hyperpolarization. Probing the neuron a second time without the external spike shows a small bump in the membrane potential around the time of the teacher spike. We continue to present the same input pattern, alternating between teaching trials (with teacher spike) and recall trials (without teacher, and synaptic plasticity switched off). Plasticity is Hebbian until the weights are strong enough such that there is considerable depolarization before the teacher spike, which induces synaptic depression for neurons firing before. Also, spike after-hyperpolarization is partially compensated by excitation, which reduces the window for potentiation. Continuation of learning after the spike association has been achieved (second to right plot) shrinks the windows for depression and potentiation, until they are very narrow and very close to each other in time. Because synaptic plasticity is determined by the overlap of the normalized PSP with periods of depolarization and hyperpolarization, depression and potentiation ultimately cancel for most synapses, therefore learning converges.

**Figure 3.** Hebbian learning with Anti-Hebbian MPDP. A postsynaptic neuron is presented the same input pattern multiple times, alternating between teaching trials with teacher spike (blue trace) and recall trials (green trace) to test the output. Initially, all weights are zero (left). Learning is Hebbian initially, until strong depolarization occurs. When the spike first appears, it is still not at the exact location of the teacher spike (second to right). Continued learning moves it closer to the desired location. Also, the time windows of the voltage being above \[ \theta_D \] and below \[ \theta_P \] shrink and move closer in time, therefore depression and potentiation ultimately cancel.
Perceptron learning with MPDP

Perceptrons were conceived as strongly abstracted basic models for perception and memory. In the simplest form, a perceptron is a one-layered feed-forward neuronal network consisting of \( N \) binary rate-based input neurons and one such output neuron. It transforms the input activity into an output, which can be viewed as an association between both. By changing the weight vector it is possible to teach the perceptron input-output associations, and it was shown that it can hold up to \( 2^N \) arbitrary associations within a single weight vector [26]. The Perceptron Learning Rule (PLR) is able to find the weights for any learnable set of associations. It has several desirable traits: It achieves optimal memory capacity, learning terminates in a finite number of steps, it has a stop condition to prevent overlearning, and it allows to add a margin to make the output robust against noise [24]. During training with the PLR, an input pattern \( \vec{x}^\mu \) is fed into the network and the output is compared to the target \( y^\mu \). If the networks’ output is wrong, the weights are iteratively changed towards the correct solution:

\[
\Delta w_i = \begin{cases} 
\eta x_i^\mu & \text{if } y^\mu = 1 \text{ and } \sum_i w_ix_i^\mu < \theta + \epsilon \\ 
-\eta x_i^\mu & \text{if } y^\mu = 0 \text{ and } \sum_i w_ix_i^\mu > \theta - \epsilon \\ 0 & \text{else} 
\end{cases}
\]  

(8)

\( \theta \) is the activity threshold of the postsynaptic neuron, \( \eta \) is the learning rate and \( \epsilon > 0 \) is the margin against noise. In general, the higher the margin, the more the input pattern can be corrupted and still lead to the correct output.

MPDP can be mapped to the PLR. An input pattern consists of a volley of synchronous spikes in neurons \( i \) with \( x_i^\mu = 1 \) at time \( t = 0 \), the others stay silent. If \( y^\mu = 1 \), a postsynaptic spike is induced externally at \( t = 0 \), concurrent with the presynaptic volley. This spike causes a reset to the resting potential and a subsequent hyperpolarization which is in shape equal to a PSP, see Eqn. 3. During training, each input-output association \( \{\vec{x}^\mu, y^\mu\} \) is presented to the postsynaptic neuron with sufficient time between presentations for the membrane potential to settle back into equilibrium and for weight changes to take effect. By assuming that the synaptic input is not strong enough to cause additional spikes, we can write the membrane potential for \( t > 0 \) as

\[
V(t) = \left( \sum_i w_i x_i^\mu - \lambda y^\mu \right) \varepsilon(t) .
\]

The resulting amplitude of \( V(t) \) is

\[
A_V^\mu = \left( \sum_i w_i x_i^\mu - \lambda y^\mu \right) \varepsilon_{\text{max}} .
\]

(10)

with \( \varepsilon_{\text{max}} = \max_s \{ \varepsilon(s) \} \). The plasticity rule is Eqn. 8 with \( \nu = 1 \). We set \( \vartheta_D = V_{\text{thr}} - \epsilon, \lambda \varepsilon_{\text{max}} = V_{\text{thr}} + \epsilon, \vartheta_p < 0 \) and \( \vartheta_p \approx 0 \). From Eqn. 10 we derive two cases where synaptic plasticity is induced:

- For \( y^\mu = 1 \), if \( \sum_i w_i x_i^\mu < \lambda \), the neuron will by hyperpolarized for \( t > 0 \), and therefore synapses that were active in the pattern undergo potentiation.
- For \( y^\mu = 0 \), if \( \sum_i w_i x_i^\mu > \vartheta_D / \varepsilon_{\text{max}} \) (and \( \sum_i w_i x_i^\mu < V_{\text{thr}} / \varepsilon_{\text{max}} \)), the synapses of active neurons will be depressed.

Comparison shows that the conditions and direction of weight changes in both cases are equivalent to the respective cases in the PLR (Eqn. 8). In the first case, learning stops if \( \sum_i w_i x_i^\mu = \lambda \). When tested without the teacher spike, the amplitude of the compound EPSP is \( A = \lambda \varepsilon_{\text{max}} = V_{\text{thr}} + \epsilon \) which is above the spiking threshold. The second case is simpler, because both during learning after convergence and during recall the membrane potential stays below the firing threshold. Fig. 4B illustrates the mapping.
The first difference to the PLR is that the weight changes are not constant, but a function of the amplitude \( A_V^\mu \). Let \( t_1, t_2 \) with \( t_1 < t_2 \) be the intersection times of the membrane potential with either \( \vartheta_D \) or \( \vartheta_P \). Weight changes are proportional to \( \int_{t_1}^{t_2} \varepsilon(t)dt \), the area of the PSP kernel function \( \varepsilon(t) \) between \( t_1 \) and \( t_2 \). If \( A_V^\mu \) is close to one of the thresholds, \( \varepsilon(t) \) can be approximated by a parabola around the maximum to calculate the intersections. The difference \( \Delta t = t_2 - t_1 \) goes towards zero with the square root of \( A_V^\mu - \vartheta_D \) or \( \vartheta_P - A_V^\mu \), which for all practical purposes results in a finite \( \Delta t \). Therefore, the learning rate stays finite and decreases systematically as a pattern gets closer to meeting the stop condition. This poses no problem for perceptron learning: With a finite learning rate a pattern will be still learned in a finite number of steps.

The second difference is that the two cases laid out above are not the only ones which induce synaptic plasticity. During learning of multiple associations, some will meet the stop condition earlier than others. Weight changes induced by ongoing learning will distort the membrane potential of an already learned memory. For example, in Fig. 4B plasticity came to a halt for this specific pattern. Weight changes from other patterns can drive upwards the membrane potential for this pattern without any consequence for correct output. However, if during training the voltage amplitude increases above \( \vartheta_D \), synaptic depression is induced. From the viewpoint of the specific pattern, this depression is superfluous, it adds or subtracts nothing to the already correct recall. But it is an additional constraint on the weights, which reduces capacity compared to a binary Perceptron.

Fig. 5A shows the capacity of the spiking perceptron with MPDP and a margin of \( 2mV \) for different network sizes. For a load up to 1.3 recall is almost perfect for all network sizes. The networks with \( N = 100, 200, 500 \) reach a capacity closer to 1.4. As expected, the small network with \( N = 50 \) is worse due to finite size effects. Unexpectedly, the network with \( N = 1000 \) also has a worse recall than the others, with a lower fraction of correct recall for all loads than all other sizes. Increasing the margin to \( 10mV \) strongly reduces the capacity. Perfect recall is achieved for loads below 1 except for \( N = 50 \).

**Chronotron learning with MPDP**

MPDP can be used to train a feed-forward network to fire at desired times. To assess the memory capacity quantitatively, we adapted the Chronotron framework [6]. We monitor the success of recall over time. The network of size \( N = 2000 \) generates the desired output spikes quickly after 300 learning blocks (Fig. 6A). Training has not converged at this point, and continuation of training reduces the difference of actual and desired output spike time (see Fig. 6B). For \( \alpha \leq 0.1 \) the generated output spike is on average less than 0.5 ms away from the desired time.

To evaluate the recall after convergence, for each \( \{ N, \alpha \} \) we average the fraction of recalled spikes and distance to teacher over the last 100 learning blocks and all 50 realizations. The final fraction of recalled spikes and average distance are shown in Fig. 7. The smallest network (\( N = 200 \)) never reaches perfect recall, but has a capacity of \( \alpha_{90} = 0.095 \). All other networks achieve perfect recall of \( \alpha_{pr} = 0.1 \) and a capacity of \( \alpha_{90} \approx 0.135 \). The average distance of spikes from teacher grows with the load, but stays below 0.5 ms.

Having fixed input spike times is a highly idealized situation. Spikes in the brain have finite precision [1]. Jitter in the input leads to jitter of the membrane potential. Because of the nonlinearity of spike generation, when the membrane potential is close to the firing threshold even small deflections can generate spurious spikes. Training a network with MPDP provides a natural safeguard against noise, because after convergence the voltage stays at a distance of \( V_{thr} - \vartheta_P \) away from the firing threshold at all non-spike times. We test the tolerance of the trained network against noise by presenting the input spike patterns again, but we add gaussian noise of different variances to each input spike (Eqn. 7). In Fig. 8 we plot the fraction of recalled spikes as a function of the load for \( N = 1000 \) (A) and 2000 (B) for different \( \sigma \). Recall is unaffected for jitter levels \( \sigma \leq 0.2ms \). For \( \sigma = 0.5ms \), the capacity \( \alpha_{90} \) is slightly lower, and for higher jitter variances the recall stays below 90 %. However, Fig. 8A and B shows a prominent “dip” in the
Figure 4. perceptron learning with MPDP. A: The amplitude of the voltage depends on the input pattern. The red and blue spikes belong to two different patterns, which are overlaid to highlight the difference in the resulting amplitude of the PSP. All spikes occur synchronously. B: Learning of a pattern with $y^n = 1$. Initially, the combined input (dashed green line) is not enough to compensate the hyperpolarization (dashed red line). As a result, all synapses of neurons which fired in the pattern will get potentiated. Plasticity comes to a halt when input and hyperpolarization exactly cancel. Removing the teacher spike during recall unveils the compound EPSP, which by construction is guaranteed to cross the firing threshold $V_{thr}$.

recall. Intermediate loads around $\alpha = 0.05$ lead to worse recall than low and high loads. This is a curious and unexpected feature, for which we do not yet have a satisfactory explanation. From the perspective of a single pattern, trying to learn other patterns at the same time is equivalent to noise on the weights. Because the tolerance against input noise during recall is better at a load of 0.1, we trained Chronotrons of size $N = 1000$ and $N = 2000$, but applied input jitter of variance $\sigma = 0.2ms$ during training. After learning, we tested noise robustness (Fig. 8 C and D). The capacity without noise decreases slightly from around 0.135 to 0.13, while robustness against input is improved, as the network now tolerates an input jitter of $\sigma = 0.5ms$ with hardly any decline of recall from noise-free input. Moreover, the dip is reduced.

We next test the ability of a Chronotron trained with MPDP to generalize from noisy examples. During training, the input patterns are induced with jitter on the spike times (Eqn. 7). After training
Figure 5. Capacity of perceptrons trained with MPDP: Fraction of correctly classified patterns as a function of the total load $\alpha = P/N$, for different network sizes. A: Recall with a small margin $\epsilon = 2mV$. Capacity is lowest for the largest network. B: Same as A with margin $\epsilon = 10mV$. The higher margin leads to more constraints on the weights, which decreases the attainable capacity of the Perceptron.

we test recall with noise free input patterns. The resulting recall in networks with $N = 1000$ is shown in Fig. [5]. The network achieves perfect recall, although during training it has only seen noisy examples. The capacity deteriorates with increasing jitter. The critical load $\alpha_{90}$ as a function of jitter is shown in Fig. [11]B (blue line).

Lastly, we do the same experiments with a range of different learning rules for the Chronotron. We estimate the critical load, the quality of recall under noisy input and ability to generalize with ReSuMe [7], E-Learning [6] and FP-Learning [9]. It was shown that the Chronotron can maximally hold a load between 0.1 and 0.3 in memory [9]. FP-Learning is used to train the Chronotron with a finite precision, which makes the capacity attained with FP-Learning an upper bound on the true value. Comparing the quantitative results from other rules provides an evaluation of the relative capabilities of MPDP.
Figure 6. Convergence of learning. **A**: Average fraction of spikes within a distance of $\vartheta_{CC} = 2\text{ms}$ from the desired time $t_d$. After 300 learning blocks all spikes have been learned except with $\alpha = 0.125$. **B**: Distance of output spikes to $t_d$. Although all desired output spikes are present early, continuation of training reduces the average temporal error. The output gets closer to the desired goal.

Fig. 10 shows the capacity of all plasticity rules. The upper bound established by FP-Learning is $\alpha_{90} \approx 0.26$. Therefore, MPDP is capable of storing half of the maximal associations in a network. Next, we evaluate the capacity under noise during recall for all learning rules (Fig. 11 A). With MPDP, the recall is almost constant up to $\sigma = 0.5$. FP-Learning and E-Learning show a steep decrease of recall with jitter, although in the largest network the decrease is not as strong. MPDP is the only rule which has a guaranteed distance of the voltage to the firing threshold, therefore we expect that training the Chronotron with MPDP leads to noise resistance. Last, we compare the ability for generalization of all learning rules (Fig. 11 B). For MPDP, FP-Learning and E-Learning the capacity decreases monotonically with jitter. FP-Learning, E-Learning and ReSuMe do not achieve 90% recall with jitter greater than 2 ms. Interestingly, the capacity with ReSuMe shows a maximum at $\sigma = 0.5\text{ms}$. It is likely that without noise ReSuMe gets stuck in local minima which prevents it from reaching better solutions.

Discussion

We introduced a synaptic plasticity mechanism that is based on the requirement to balance the membrane potential and therefore utilizes the postsynaptic membrane potential rather than postsynaptic spike times as the relevant signal for synaptic changes. We have shown that this rule not only can be mapped to the Perceptron Learning Rule for synchronous activity patterns, but also allows the temporal association of output spikes with arbitrary input spike patterns (Chronotron). Before, both network types could only be trained with supervised learning rules. The sensitivity of MPDP to subthreshold membrane potential induces robustness against noise.
Figure 7. Recall of a chronotron trained with MPDP. A: Average fraction of recalled spikes after convergence. For the larger networks, recall is perfect up to $\alpha = 0.1$. B: Average distance of recalled spikes from teacher. After convergence, the output spikes are on average very close to the teacher. This temporal error grows with load, but stays below 0.5 ms.

Biological plausibility of MPDP

Spike-Timing-Dependent Plasticity (STDP) is experimentally well established and simple to formalize, which made it a widely used plasticity mechanism in modelling. It is therefore important to note that MPDP is compatible with STDP. Spikes come with a stereotypic trace in the membrane potential. The voltage rises to the threshold, the spike itself is a short and strong depolarization, and afterwards the neuron undergoes reset, all of which are signals for MPDP. Pairing a postsynaptic spike with presynaptic spikes at different timings gives rise to a plasticity window which shares its main features with the STDP window: The magnitude of weight change drops with the temporal distance between both spikes and the sign switches close to concurrent spiking.

It is known that the somatic membrane potential plays a role in synaptic plasticity. Many studies investigated the effect of prolonged voltage deflections by clamping the voltage for an extended time while repeatedly exciting presynaptic neurons (e.g. see [27]). However, MPDP predicts that synaptic plasticity is sensitive to the exact time course of the membrane potential, as well as the timing of presynaptic spikes. This necessitates that dendrites and spines reproduce the time course of somatic voltage without substantial attenuation. Morphologically the dendritic spines form a compartment separated from the dendrite, which keeps calcium, for example, localized in the spine. It has been a topic under investigation whether the spine neck dampens invading currents. Despite experimental difficulties in measuring spine voltage, recent studies found that backpropagating action potentials indeed invade spines almost unhindered [28]. Furthermore, independently of spine morphology and proximity to soma, the time course of a somatic hyperpolarizing current step is well reproduced in spines [29]. This shows that at least in principle the somatic voltage trace is available at the synapse. In turn, voltage-dependent calcium channels can transform subthreshold voltage deflections into an influx of calcium, the major messenger for synaptic plasticity. A few studies found that short depolarization events act as signals for synaptic plasticity [18, 19], with a dependence of sign and magnitude of weight change on the timing of presynaptic spikes.

Another important point is the sign of synaptic change. “Membrane Potential Dependent Plasticity”
Figure 8. Recall in the case of input noise. A and B: Recall of networks with input noise after noise-free training (A: $N = 1000$, B: $N = 2000$). For small jitter $\leq 0.2 ms$, recall is unaffected. For higher jitter variance, recall shows a slump for intermediate loads. C and D: The same after training with noisy examples; $\sigma = 0.2 ms$ during training. This alleviates the slump and improves noise tolerance.
Figure 9. Recall success after training with noisy examples. Fraction of recalled spikes in networks with 1000 neurons for different levels of input jitter. The capacity $\alpha_{90}$ decays with noise.

Figure 10. Capacity of Chronotrons trained with different rules. Estimated critical load $\alpha_{90}$ for all four learning rules. FP-Learning provides an upper bound, E-Learning a lower bound for Chronotron capacity. Therefore, MPDP reaches half of the maximal capacity. Capacity in all three rules scales with network size. The Chronotron trained with ReSuMe leads to the lowest capacity.
Figure 11. Capacity of Chronotrons under different learning rules. A: Recall with noisy input. Critical load $\alpha_{90}$ for MPDP, E-Learning and FP-Learning with $N = 1000$ (dashed line) and $N = 2000$ (solid line). Discontinued lines are due to correct recall being lower than 90% for $\sigma < 1$ ms. In MPDP, recall is mostly unaffected of $N$ and $\sigma$ up to $\sigma = 0.5$. Please note that we ignored the dip in recall here (see Fig. 8). B: Recall after training with noisy examples in different learning rules. Similar to A, here we show the critical load $\alpha_{90}$ for MPDP, E-Learning and FP-Learning with $N = 500$ (dashed line) and $N = 1000$ (solid line) as a function of jitter variance $\sigma$. Only MPDP reaches 90% recall with a jitter of 5 ms, therefore the plots for the other rules break off at $\sigma = 2$ ms. Up to this point, in MPDP, FP-Learning and E-Learning capacity decays gracefully. An interesting exception is ReSuMe, where the capacity reaches a peak at $\sigma = 0.5$ ms.
per se is a very general term which potentially could include many different rules\cite{30,31}. Here, MPDP serves as a mechanism that tries to keep the membrane potential within bounds. For inhibitory synapses this requirement results in a Hebbian plasticity rule, which has been reported\cite{21}. Inhibitory neurons in cortex have been implied to balance excitatory inputs in a very specific way\cite{32}. MPDP on excitatory synapses is necessarily “Anti-Hebbian”. Lamasa et al.\cite{22} found that pairing presynaptic spikes with postsynaptic hyperpolarization can lead to synaptic potentiation. This was caused by calcium permeable AMPA receptors (CP-AMPARs) present in these synapses. However, Anti-Hebbian plasticity does not rely on CP-AMPARs alone. Verhoog et al.\cite{20}, for example, found Anti-Hebbian STDP in human cortex, which depends on dendritic voltage-dependent calcium channels. Taken together, these findings demonstrate the existence of cellular machinery which could implement Anti-Hebbian MPDP.

**Properties and capabilities of Anti-Hebbian MPDP**

We derived Anti-Hebbian MPDP from a balance requirement: Synapses change in order to prevent hyperpolarization and strong depolarization for recurring input activity. This kind of balance reduces metabolic costs of a neuron and keeps it at a sensible point of operation\cite{33}. The resulting plasticity rule is Anti-Hebbian because synapses depress when the postsynaptic neuron is excited and potentiate when it is inhibited. However, spike after-hyperpolarization turns Anti-Hebbian MPDP into Hebbian plasticity. Attached to every postsynaptic spike is the reset, a period of hyperpolarisation. Therefore synapses of presynaptic neurons which fired close in time to the postsynaptic spike will potentiate. The total potentiation summed over all synapses depends on the duration (with $\nu = 2$ also on the magnitude) of hyperpolarization. Because potentiation reduces this duration if the same activity pattern occurs again, total potentiation is also reduced. The same is true for total depression, which depends on the duration where the membrane potential stays above $\vartheta_D$ and which reduces this duration in future occurrences. If the rise time of the voltage before the spike and residual spike after-hyperpolarization are both short and close in time, potentiation and depression will ultimately become balanced by cancelling around a spike. This balance is the consequence of plasticity, if a teacher spike is imposed at a fixed time relative to the onset of a pattern. In other words, MPDP with teacher spikes converges into a fixed point.

A further interesting aspect of MPDP is the emergence of robustness against noise. Most obviously, with the choice of the threshold for depression the neuron sets a minimal distance of the voltage to the firing threshold for known input patterns. This allows to have a margin as in Perceptron learning and perfect recall in the case of noisy input in the Chronotron. The second effect of the depression threshold is more subtle. Not only does it prevent spurious spikes, but through learning the slope of the membrane potential just before the desired spike becomes very steep. This is necessary to prevent spike extinction by noise. Consider an output spike with a shallow slope of the voltage. Increasing the voltage slightly around the spike time moves forward in time the intersection of the voltage with the firing threshold, by a proportionally large margin. Decreasing voltage moves it backwards in time or could even extinguish the spike; a flat slope implies a low peak of the “virtual” membrane potential. MPDP in contrast achieves a state which is robust against spike extinction as well as the generation of spurious spikes. On the downside, keeping the voltage away from the firing threshold as well as imposing steepness on the slope just before spikes puts more constraints on the weights. Robustness comes at the cost of capacity.

**Emulating supervised learning rules with MPDP**

In supervised learning rules, the “supervisor” is an external entity which compares the output activity to a goal. From the mismatch it computes synaptic changes that move the network closer to a state where it generates the desired output. The weight changes are composed of an activity dependent term and a modulation which incooperates the mismatch. For example, in the Perceptron Learning Rule weight change is proportional to the state of the input neuron times the modulatory signal. The information about the modulation is necessary for the synapse. It was shown by Legenstein et al.\cite{14} that
a supervised STDP rule with teacher spikes is in general not able to successfully learn spike times. This rule can not differentiate between desired and spurious spikes, and will not stop plasticity if training was successful, eventually unlearning the desired state. ReSuMe demonstrates what is missing: It uses explicit information about the status (either desired or spurious) of each spike, which is then transformed by the synapse into weight changes working towards the goal. However, a shortcoming of supervised learning rules so far is the lack of plausible mechanisms that generate and transmit modulatory information to the synapse, especially considering that exact spike time learning requires temporally well confined signals.

MPDP provides a solution to this problem. The neuron at the soma computes the supervisory signal from the combination of a teacher spike with concurrent neuronal input. The carrier of the information is the membrane potential which provides plenty of “bandwidth” to transmit the presence of a spike, its precise time as well as the status of each spike. For example, in the mapping of MPDP to the PLR the postsynaptic neuron compares the total synaptic input to spike after-hyperpolarization caused by the teacher and transmits the difference to its synapses, which transform the global signal by applying thresholds, and change locally after incooperating the respective presynaptic activity. In the Chronotron task, due to the immediate reset there is always residual spike after-hyperpolarization, and the information about the learning progress is contained in the duration of hyper- and depolarization.

For learning of exact spike times there are two different classes of plasticity rules. In the first class there are spike-based learning rules like ReSuMe, E-Learning and FP-Learning. They use times of desired and actual output spikes to compute synaptic changes. Recently a second class of learning rules emerged with two examples: Perceptron Based Spiking Network Learning Rule (PBSNLR, [8]) and High-Threshold Projection (HTP, [9]). It was shown that the latter rule attains maximal storage capacity in spike-time learning [9]. Both rules transform Chronotron learning into a static task by abolishing spike generation and perform a perceptron classification on the membrane potential at a given set of points in time. If the neuron is not supposed to spike at a sampling point, the membrane potential has to be below the firing threshold, which is enforced by the learning rule. At times of desired spikes, the learning rule makes sure the membrane potential crosses the firing threshold from below. HTP also adds a reset kernel at desired spike times, which avoids the problems associated with the nonlinearity of spike generation. MPDP shares some similarities with this second class of plasticity rules. In PBSNLR and to a lesser degree in HTP the sampling of non-spike times has to be relatively dense, with a frequency higher than the inverse of the membrane time constant. Membrane potential dependent depression “samples” the membrane potential continuously, without the need of a discrete sampling rate. The teacher input mimicks the addition of a reset kernel, which in MPDP provides the signal for potentiation as well as a “distorted” environment for learning. Otherwise MPDP recognizes the teacher spike as unwanted and tries to quench it. MPDP therefore justifies this second, powerful class of very artificial spike time learning rules by providing a plausible neuronal mechanism for them.

**Outlook**

The ability to learn precise spike times with MPDP is the consequence of the neurons’ objective of keeping the membrane potential in a balanced regime. This objective may also explain the strikingly detailed balance of excitation and inhibition observed in cortex [34, 30]. In particular, a recent study showed that parvalbumin expressing interneurons selectively adapt their synaptic strength in an activity dependent manner to match the excitatory inputs to target cells [32]. Besides its own merits like modest metabolic cost through sparse activity, the balance in cortex could be the mark of a powerful learning principle at work. We speculate that with its stability properties MPDP not only keeps cortex at an operating point where the network dynamics are marginally stable, but also learns to simultaneously make it highly susceptible for statistically rare and thereby unexpected inputs such that only the relevant information becomes further processed and transmitted in terms of spatio-temporal spike patterns.
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