# Biologically Plausible Plasticity

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**Abstract**

We construct the minimal set of rules for robust biologically plausible learning based on dopamine-modulated spike-timing-dependent plasticity. Furthermore, we apply the developed method to the existing realistic model of a column in mouse V1 and observe stable learning.

## 1 Introduction

The remarkable success of deep learning was originally inspired by biological neural networks in the brain [5, 6]. However, modern deep learning techniques, such as stochastic gradient descent (SGD), bear little resemblance to known biological learning mechanisms [8]. In computational neuroscience, numerous methods have been shown to demonstrate learning [3, 10, 11], yet, they too rely on machine learning approaches like SGD or computing a matrix inverse.

In this project, we investigate the components of biologically plausible plasticity and show that consistent learning can be achieved by augmenting spike-timing-dependent plasticity (STDP) with other biological mechanisms.

## 2 Method

Simulating neurons in sufficient detail remains computationally expensive, so throughout this project, the networks are tasked with learning the correct labels for 8 binary strings[1]. Importantly, the networks are trained in a reinforcement learning (RL) manner, rather than supervised: the correct label is not provided, but a reward is given when a prediction is made. Instead of using the raw reward, which is either 1 or -1, we supply the network with the success signal, defined as "reward minus expected reward" [4].

### 2.1 Spike-timing-dependent plasticity

STDP is a key mechanism for synaptic learning in neurons [2], so we select it as the primary plasticity rule of the system. However, pure STDP is not suitable for RL, as it lacks the reward mechanism. We, therefore, build upon the dopamine-modulated STDP model (DM-STDP) proposed by Izhikevich [7], and tie the success signal to the dopamine level in all synapses.

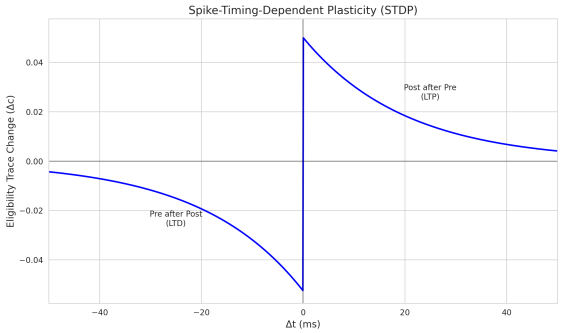


Figure 1: Change in eligibility trace for $\Delta t$ between a spike of the postsynaptic neuron and the presynaptic neuron. The eligibility trace decays to zero over time.

[1]The full dataset is presented in Appendix A

The primary equation for DM-STDP describes the change of synaptic strength as a function of eligibility trace and dopamine:

$$\frac{d(\text{synaptic\_strength})}{dt} = \frac{\text{eligibility\_trace} \cdot \text{dopamine}}{\tau_s} \tag{1}$$

As a result, the synapse that had a presynaptic spike shortly before the postsynaptic spike strengthens if the following prediction was correct and weakens otherwise. The inverse is true for a synapse that experienced a postsynaptic spike before the presynaptic one.

### 2.2 Minimal standalone model

DM-STDP by itself is not sufficient for consistent learning for two main reasons. First, without strong background noise, some neurons will inevitably initialize with weak incoming synapses that will never elicit a spike, and without the spike, there will be no eligibility trace signal to increase the synaptic strength. Such neurons will thus remain silent for the entire duration. Second, generally, the network will continue firing after a prediction is made, and the produced spikes will change the eligibility traces, such that they do not represent the spikes that led to the prediction.

To address these issues, we experiment with additional rules and mechanisms to derive the minimal model capable of robust learning.

**Homeostatic adaptation** of excitatory synapses mitigates the problem with silent neurons. Homeostatic adaptation, described in Eq. 2, adds a term to the synaptic strength that decays at rest and becomes negligible if the postsynaptic neuron fires at least half as frequently as the presynaptic neuron.

$$\begin{aligned} s_h &\mathrel{+}= \Delta h && \text{(on presynaptic spike)} \\ s_h &\mathrel{-}= 2 \cdot \Delta h && \text{(on postsynaptic spike)} \\ \frac{ds_h}{dt} &= -\frac{s_h}{\tau_h} \\ s_h &= \text{clip}(s_h, 0, s_{\max}) \\ s_{\text{total}} &= s + s_h && \text{(on presynaptic spike)} \end{aligned} \tag{2}$$

**Post-prediction inhibition** aims to reduce or entirely suppress the activity of the network after a prediction is made. This helps to preserve the eligibility traces and sets the network to a more consistent (silent) state before a new input arrives. The strength of inhibition is similar to that of typical inhibitory synapses in the network, but the inhibition is applied to the entire excitatory population.

To further bridge the gap between our simulation and the brain, we separate neurons into excitatory and inhibitory populations, and apply the **exponential inhibitory homeostatic plasticity** rule, rather than training the inhibitory synapses with STDP. This ensures that the network is in e-i balance. As can be seen in Eq. 3, the rule increase the strength by a smaller amount when the current value is larger, and vice versa. This leads to a nice property that e-i balance can be achieved even when excitatory and inhibitory populations fire at different rates, and introduces a soft threshold on the connection strength.

$$\begin{aligned} s &\mathrel{+}= exp(-(s - \text{baseline\_s})/\text{amplitude}) \cdot \text{lr} && \text{(on presynaptic spike)} \\ s &\mathrel{-}= exp(+(s - \text{baseline\_s})/\text{amplitude}) \cdot \text{lr} && \text{(on postsynaptic spike)} \\ s &= \text{clip(s}, -inf, 0) \end{aligned} \tag{3}$$

We also explored **spike frequency adaptation** and **background noise**, but these did not lead to significant improvements when combined with the other components.

The performance of the combined model, as well as the impact of the individual components is discussed in sections 4 and 4.2.

## 3 Learning in the cortical column

With the insights gathered from the minimal model, we implement the mechanisms from section 2.2 into the anatomical model of a column from the mouse primary visual cortex, developed by Moreni et al. [9]. This model is of interest because it replicates the structure of a real brain, including four cell types, appropriate connection probabilities between the populations, and realistic parameters of neuronal equations.

We find that the synaptic strengths are quite low, and high background noise is required to produce excitations. For example, in the absence of other inputs, forcing all excitatory neurons of layer 2/3 to fire simultaneously 4 times in a row does not produce a spike in either of the 3 inhibitory populations of the same layer. High noise levels are problematic for the STDP learning rule, and the synapses should have a contribution in creating the postsynaptic spike. Thus, we choose to modify the model by increasing the conductance of all synapses by a factor of 50 and removing the noise. Crucially, we keep all other parameters the same[2], so the model remains realistic in many ways.

Inspecting the synaptic groups (where a group of synapses is all synapses connecting any two populations), we find that the average strength of synapses within some groups is two orders of magnitude larger than others. Thus, while in the minimal model it was not problematic to have a single set of learning-related hyperparameters (e.g. strength bounds for excitatory, and baseline value for inhibitory), for the cortical column we define global coefficients and scale them with the initial strength of the particular synapse group. In addition, we mitigate a small limitation of the model: all synaptic strengths in a group were initialized to the same value, which is an implausible state for a real system. Instead, we initialize with a Gaussian around the value.

Apart from these minor challenges, we find that all identified learning components translate smoothly to the cortical column model.

# 4 Results

First, we present a training run for the minimal model with all discussed components and moderate background noise. Figure 2 shows the dopamine level graph at the top and the spike raster plot at the bottom. Each spike of an output neuron (depicted in red) triggers a sharp change in dopamine level. A bump up means the prediction was correct, and a bump down indicates a wrong prediction. As the learning is based on the success signal rather than reward, the magnitude of the bump is determined by the value that the network expected to receive. The expected value is updated with an exponential moving average. A sample from the dataset is presented every 100 ms by spiking the corresponding combination of input neurons twice in short succession.

From the figure, it is clear that the network was initialized into a state where most predictions are incorrect, and after training for 100 seconds it predicts the correct value most of the time.

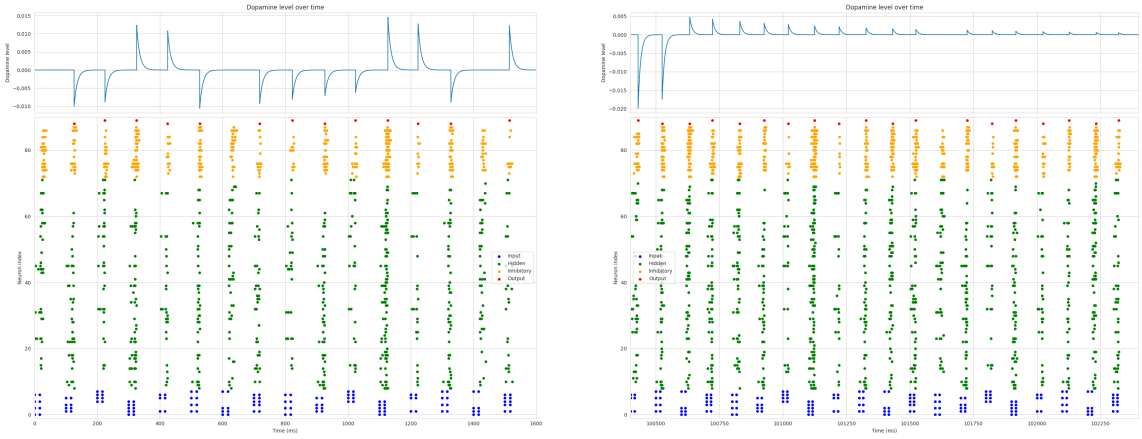


Figure 2: Training the minimal model. Left: first 2 epochs. Right: after 100 simulation seconds of training.

## 4.1 Cortical column

With the goal of making the training more computationally tractable, we experiment only on layer 2/3 of the model. Nonetheless, the approach and learning rules completely generalize to the rest of the column. We use Optuna [1] to systematically address the choice of learning-related hyperparameters in the cortical column. Additionally, we note that due to the initial low strength of the incoming weights, the SST neurons fire very rarely, unless the homeostatic rules are given significantly more weight.

[2]NMDA is replaced with AMPA, as it would introduce strong correlations between neighboring inputs, unless the frequency of presenting samples is reduced.

In figure 3, we present the chance-level performance of the network upon initialization, and 100% accuracy just 23 simulation seconds later.

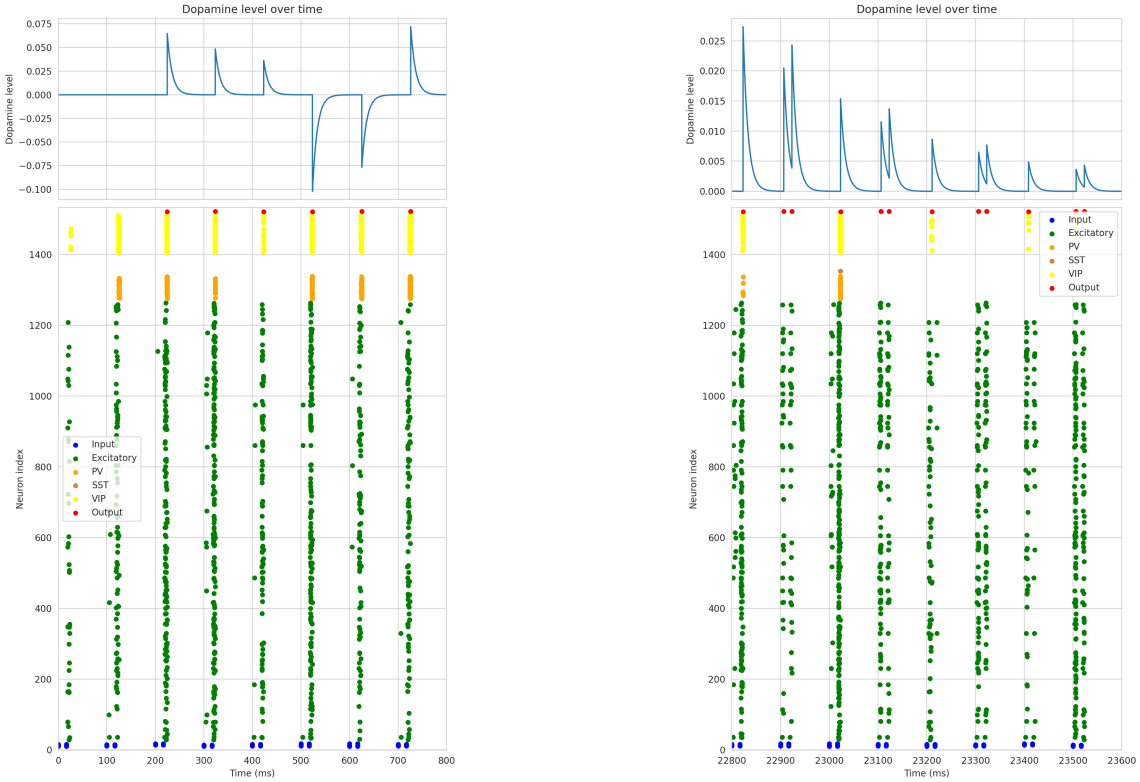


Figure 3: Training in cortical column. Left: the first epoch (4/8 correct, chance level). Right: after training for 30 epochs (8/8 correct)

## 4.2 Ablations

To investigate the importance of the individual pieces, we present training results in the minimal model when one of the components is qualitatively changed or disabled. Figure 4 shows that as we anticipated, spontaneous activity is detrimental to learning. We demonstrate that even with long training, no improvement in prediction accuracy can be observed.

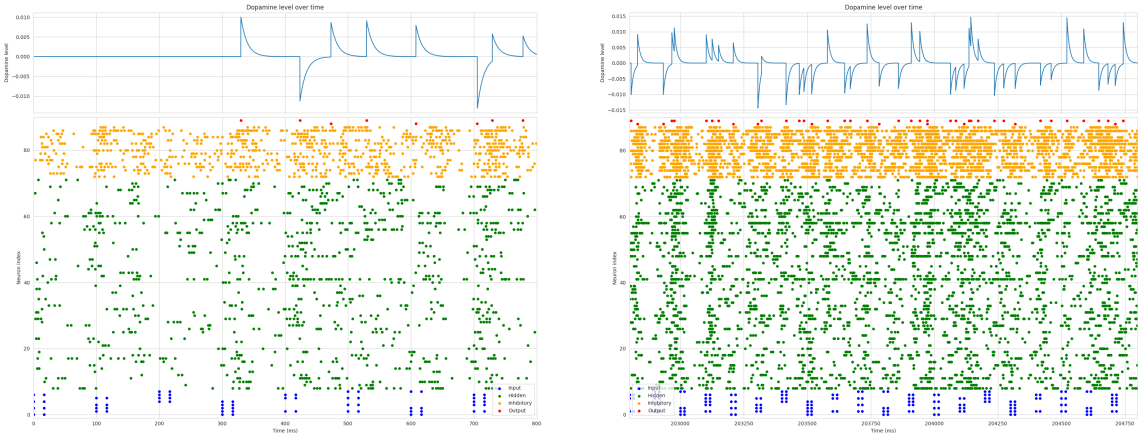


Figure 4: Training with spontaneous activity. Left: the first epoch. Right: after training for 200 seconds

Exploring the linear version of the inhibitory plasticity rule, a natural alternative to the used exponential, we find that, as presented in figure 5, both result in successful training. However, the exponential rule reaches near-convergence faster, and, unlike the alternative, stabilizes the network such that every prediction is made with a similar number of spikes.

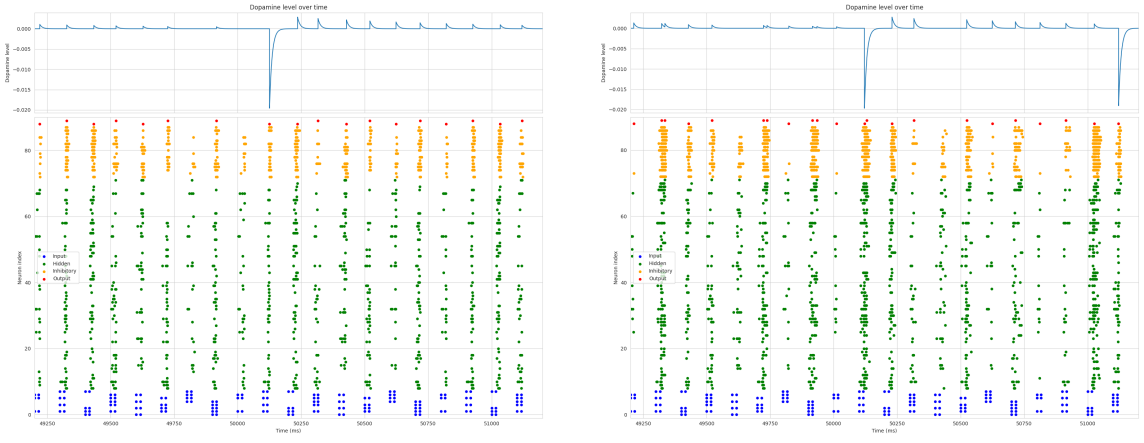


Figure 5: Comparison with linear inhibitory plasticity rule. Left: baseline, exponential rule. Right: modified, linear rule

# 5 Discussion

This report presents a method to train neural networks in a biologically plausible way. While we demonstrate successful learning in two independent models, the method is generally not competitive with machine learning systems that disregard the biological constraints. One of the fundamental issues is related to computational requirements: modern deep learning techniques allow much higher throughput of information, and benefit from large amounts of simplistic neurons.

Furthermore, we expect that generalization to unbalanced datasets will require a relaxation of some homeostatic rules. This can lead to stronger dependency on the initial network state - an undesirable property for a machine learning system. Finally, learning more than 2 classes of labels is not trivial and will likely be problematic in the current RL-based formulation.

# References


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# A Dataset

- 10101010 → 0
- 01110100 → 1
- 00001111 → 0
- 11111000 → 1
- 01000110 → 0
- 01010101 → 1
- 11100001 → 0
- 01011110 → 1

There are 8 input neurons. Input neuron $i$ spikes if and only if the sample has '1' at position $i$.