

Nonlinear Dendritic Coincidence Detection for Supervised Learning

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1 Introduction

In recent years, a growing body of research has addressed the functional implications of the distinct physiology and anatomy of cortical pyramidal neurons. In particular, on the theoretical side, we saw a paradigm shift from treating neurons as point-like electrical structures towards embracing the entire dendritic structure. This was mostly due to the fact that experimental work uncovered dynamical properties of these cells that simply could not be accounted for by point models.

An important finding was that the apical dendritic tree of cortical pyramidal neurons can act as a separate non-linear synaptic integration zone. Under certain conditions, a dendritic Ca^{2+} spike can be elicited that propagates towards the soma, causing rapid, bursting spiking activity. One of the cases in which dendritic spiking can occur was termed 'backpropagation-activated Ca^{2+} spike firing' ('BAC firing'): A single somatic spike can back-propagate towards the apical spike initiation zone, in turn significantly facilitating the initiation of a dendritic spike. This reciprocal coupling is believed to act as a form of coincidence detection: If apical and basal synaptic input co-occurs, the neuron can respond with a rapid burst of spiking activity. The firing rate of these temporal bursts exceeds the firing rate that is maximally achievable under basal synaptic input alone, therefore representing a form of temporal coincidence detection between apical and basal input.

Naturally, these mechanisms also affect plasticity and thus learning within the cortex. While the interplay between basal and apical stimulation and its effect on synaptic efficacies is subject to ongoing research, there is some evidence that BAC-firing tends to shift plasticity towards long-term potentiation (LTP). Thus, coincidence between basal and apical input appears to also gate synaptic plasticity.

In a supervised learning scheme, where the top down input arriving at the apical compartment acts as the teaching signal, the most straight-forward learning rule for the basal synaptic weights would be derived from an appropriate loss function, such as a mean square error, based on the difference between basal and apical input, i.e. $I_p - I_d$. Theoretical work has investigated possible learning mechanisms that could utilize an intracellular error signal (Urbanczik and Senn, 2014; Schiess et al., 2016; Guerguiev et al., 2017). However, a clear experimental evidence

for a physical quantity encoding such an error is—to our knowledge—yet to be found. On the other hand, Hebbian-type plasticity is extensively documented in experiments. Therefore, our work was based on the question whether the non-linear interactions between basal and apical synaptic input could, when combined with a Hebbian plasticity rule, allow a neuron to learn to reproduce an apical teaching signal in its proximal input.

In our work, we combined a phenomenological model predicting the output firing rate as a function of two streams of synaptic input (subsuming basal and apical input) with a BCM-like plasticity rule on basal synapses. LTP was induced in our model if the postsynaptic activity surpassed a threshold level that was only achievable if both basal and apical inputs were present. Therefore, we hypothesized that this combination of neural activation and plasticity rules would lead to an increased correlation between basal and apical input.

Furthermore, this temporal alignment could potentially facilitate apical inputs to act as top-down teaching signals, without the need for an explicit error-driven learning rule. Thus, we also tested our model in a simple linear supervised classification task and compared it to the performance of simple point neuron combined with a BCM-plasticity rule on the basal inputs.

2 Model

2.1 Neuron Model

The neuron model used throughout this study is a discrete-time rate encoding model that uses two separate input variables, subsuming the total synaptic input current injected arriving at the basal (proximal) and apical (distal) dendritic structure of a pyramidal neuron, respectively. The model is a slightly simplified version of a phenomenological model proposed by Shai et al. (2015). Denoting the input currents by I_p (proximal) and I_d (distal), the model is written as

$$y(t) = \sigma(I_p(t) - \theta_{p0}) [1 - \sigma(I_d(t) - \theta_d)] + \alpha \sigma(I_d(t) - \theta_d) \sigma(I_p(t) - \theta_{p1}) \quad (1)$$

$$\sigma(x) \equiv \frac{1}{1 + \exp(-4x)} \quad (2)$$

Here, θ_{p0} , θ_{p1} and θ_d are threshold variables with respect to proximal and distal input. Overall, this equation describes two distinct regions of neural activation in the

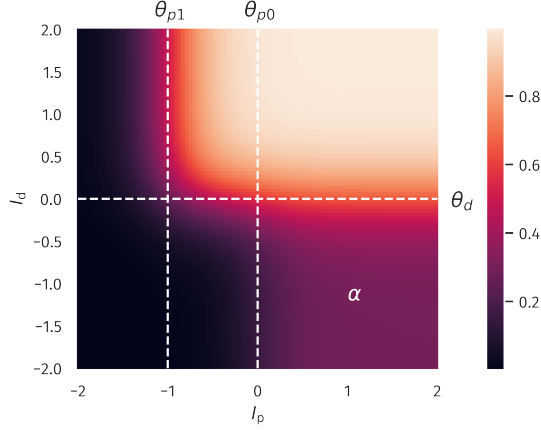


Figure 1: **Two-compartment rate model.** Firing rate as a function of proximal and distal input I_p and I_d , see (1). The thresholds θ_{p0} , θ_{p1} and θ_d define two regions of neural activity with a maximal firing rate of 1 and α .

(I_p, I_d) -space which differ in their maximal firing rates, which are set to 1 and α , where $0 < \alpha < 1$. A plot of (1) is shown in Fig. 1.

In all our numerical experiments, we compared this model with a simple point neuron model that was given by

$$y(t) = \sigma(I_p(t) + I_d(t) - \theta). \quad (3)$$

In all our numerical experiments, the apical input I_d was generated “as is”, meaning, it was not dynamically calculated as a superposition of multiple presynaptic inputs, but given by

$$I_d(t) = n_d(t)x_d(t) - b_d(t), \quad (4)$$

where $n_d(t)$ is a scaling factor, $x_d(t)$ a pre-generated discrete time sequence and $b_d(t)$ a bias. Note that n_d and b_d are time dependent since they were subject to adaptation processes that are described in the next section.

Similarly, $I_p(t)$ was given by

$$I_p(t) = n_p(t) \sum_{i=1}^N x_{p,i}(t)w_i(t) - b_p(t), \quad (5)$$

where N is the number of presynaptic afferents, $x_{p,i}(t)$ the corresponding sequences and $w_i(t)$ the synaptic efficacies. As for $I_d(t)$, $n_p(t)$ and $b_p(t)$ was used as a time dependent scaling and bias.

2.2 Plasticity

We implemented a BCM-like plasticity rule for the basal synaptic weights given by the following update equation:

$$w_i(t+1) = w_i(t) + \mu_w (x_{p,i}(t) - \tilde{x}_{p,i}(t)) y(t) [y(t) - \theta_y] \quad (6)$$

$$\tilde{x}_{p,i}(t+1) = (1 - \mu_{av})\tilde{x}_{p,i}(t) + \mu_{av}x_{p,i}(t+1) \quad (7)$$

θ_{p0}	0	V_d^t	0.25
θ_{p1}	-1	μ_b	10^{-3}
θ_d	0	μ_n	10^{-4}
α	0.3	μ_{av}	$5 \cdot 10^{-3}$
μ_w	$5 \cdot 10^{-5}$	I_p^t	0
V_p^t	0.25	I_d^t	0

Table 1: Model parameters

While the classical BCM-rule dynamically modifies the threshold θ_y to stabilize weights, we used a synaptic normalization constraint

$$w_i(t) \rightarrow \frac{w_i(t)}{\|\mathbf{w}(t)\|} \quad (8)$$

in each time step, where $\|\mathbf{w}(t)\|$ denotes the Euclidean norm of the synaptic weight vector. The threshold θ_y was chosen such that LTP is present if neural activity is in the high-activity regime (yellow area in Fig. 1), while LTD is present in the low-activity regime (given by α , see Fig. 1). Hence, we set $\theta_y = (1 + \alpha)/2$.

For comparative reasons, the point neuron model was equipped with the same plasticity rule for the proximal weights as (6), except that the threshold θ_y was dynamically adjusted as

$$\theta_y(t+1) = (1 - \mu_{av})\theta_y(t) + \mu_{av}y^2(t), \quad (9)$$

which is an implementation of a running average of the square activity, as used in the classic BCM-rule.

Additionally, the scaling and bias variables were changing dynamically according to the following homeostatic plasticity rules:

$$b_p(t+1) = b_p(t) + \mu_b [I_p(t) - I_p^t] \quad (10)$$

$$b_d(t+1) = b_d(t) + \mu_b [I_d(t) - I_d^t] \quad (11)$$

$$n_p(t+1) = n_p(t) + \mu_n \left[V_p^t - (I_p(t) - \tilde{I}_p(t))^2 \right] \quad (12)$$

$$n_d(t+1) = n_d(t) + \mu_n \left[V_d^t - (I_d(t) - \tilde{I}_d(t))^2 \right] \quad (13)$$

$$\tilde{I}_p(t+1) = (1 - \mu_{av})\tilde{I}_p(t) + \mu_{av}I_p(t+1) \quad (14)$$

$$\tilde{I}_d(t+1) = (1 - \mu_{av})\tilde{I}_d(t) + \mu_{av}I_d(t+1) \quad (15)$$

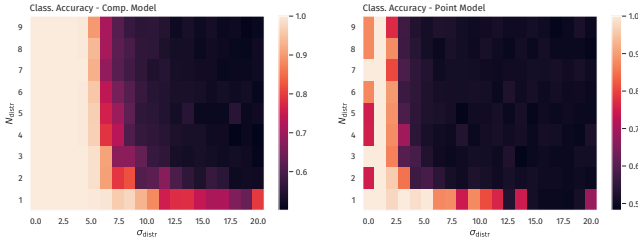
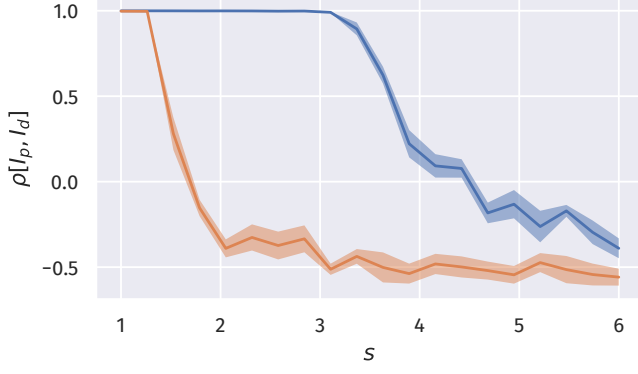
Here, I_p^t , I_d^t , V_p^t and V_d^t define targets for the temporal means and variances of I_p and I_d . The dynamic variables \tilde{I}_p and \tilde{I}_d are simply low-pass filtered running averages of I_p and I_d .

A list of all parameter values is given in Table 1.

3 Results

References

Guerguiev, J., Lillicrap, T. P., and Richards, B. A. (2017). Towards deep learning with segregated dendrites. *eLife*,



6.

- Schiess, M., Urbanczik, R., and Senn, W. (2016). Somato-dendritic Synaptic Plasticity and Error-backpropagation in Active Dendrites. *PLoS Computational Biology*, 12(2):1004638.
- Shai, A. S., Anastassiou, C. A., Larkum, M. E., and Koch, C. (2015). Physiology of Layer 5 Pyramidal Neurons in Mouse Primary Visual Cortex: Coincidence Detection through Bursting. *PLOS Computational Biology*, 11(3).
- Urbanczik, R. and Senn, W. (2014). Learning by the Dendritic Prediction of Somatic Spiking. *Neuron*, 81(3):521–528.