A SIMPLE SPIKING NEURON MODEL BASED ON STOCHASTIC STDP



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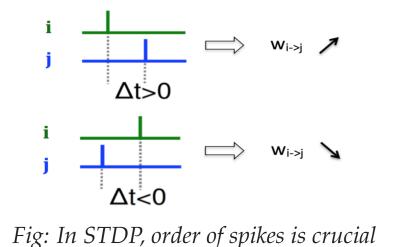
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1.FRAMEWORK

STDP: Thought to be responsible for memory, synaptic plasticity is the change of strength of neuron's links. Popular plasticity models are based on Spike Timing Dependent Plasticity (STDP):

Hebb's law (1949):

"When an axon of cell A[...] repeatedly or persistently takes part in firing (a cell B), [...]A's efficiency, as one of the cells firing B, is increased" [1]



Problem: Current models use deterministic plasticity rules whereas the biological mechanisms involved are mainly stochastic ones. Moreover, there exists few mathematical studies [2] taking into account the precise spikes timings. Finally, there is a need to understand how to bridge the time scale gap at the synapse level and how weights dynamics interplay with the network one.

Novelty: Stochastic STDP rule with discrete synaptic weights which allows a mathematical analysis of their dynamics.

2.MODEL CONSTRAINTS

- Rich enough to reproduce biological phenomena
- Simple enough to be analyzed mathematically and simulated
- Observe global properties of the network due to neurons firing

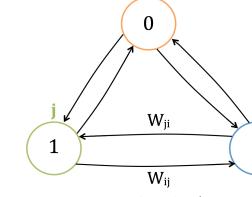
3. NEURONAL NETWORK MODEL

Individual neuron: Simple model for the membrane potential [3].

At time t, the neuron i is
$$\begin{cases} & \text{at rest if } V_t^i = 0 \\ & \text{excited if } V_t^i = 1 \end{cases}$$

Dynamic of V_t^i :

$$\frac{\alpha_i(W_t, V_t)}{\beta} \quad 1$$



- β =constant
- Fig: 3 neurons, state in circle, weights on links
- $\alpha_i(W_t, V_t) = f\left(\sum_{j=1}^N W_t^{ji} V_t^j\right) + \alpha_m \text{ with } f(x) = \frac{\alpha_M}{1 + e^{-\sigma(x \theta)}}$

Remark: α_i depends on current neurons states and weights

Dynamic of synaptic weights W_t :

Weights have probability to change only when a neuron jumps from 0 to 1:

In that particular case, $\lim_{w\to\infty} r_{+/-}(w) = R_{+/-}$ exist and we

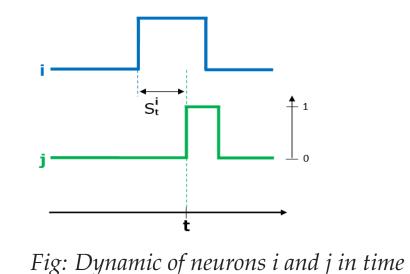
can prove the process converges to its unique invariant mea-

sure if $R_+ - R_- < 0$. We computed, thanks to (1), the difference

 $r_+(w) - r_-(w)$. It depends on (w_{12}, w_{21}) . We found parame-

ters, $A_{+}=0.8$, $A_{-}=0.7$, $w_{21}<70$, for which w_{12} diverges when

Weight explosion for w21 = 10



 $\mathbb{P}\left(W_t^{ij}=W_{t_-}^{ij}+\Delta w\right)=p^+(S_t^i)$ $\mathbb{P}\left(W_t^{ij}=W_{t_-}^{ij}
ight) \quad = \quad 1-p^+(S_t^i)$ $\mathbb{P}\left(W_t^{ji}=W_{t_-}^{ji}-\Delta w
ight)=p^-(S_t^i)$ $\mathbb{P}\left(W_t^{ji}=W_{t_-}^{ji}
ight) = 1-p^-(S_t^i)$

Remark: Need to have access to S_t in a Markovian manner

the integral of the learning window is negative:

4.MATHEMATICAL RESULTS

Markov process $(\mathbf{V_t}, \mathbf{S_t}, \mathbf{W_t})_{\mathbf{t} \geq \mathbf{0}} \in E$ from (v_0, s_0, w_0) :

- $W_t \in E_1 = \mathbb{R}^{N^2}$: matrix of synaptic weights
- $S_t \in \mathbb{R}^N_+$: last spike of neuron i occurred at time $t S_t^i$
- $V_t \in I = \{0, 1\}^N$: neuron system state.

Hypothesis: plasticity is slow compared to the network dynamics.

Mathematically, this hypothesis enables us to consider the probability that the weight changes are really small. This probability is $\sum_{\widetilde{w}} \phi_{\epsilon}(s, w, \widetilde{w}) = O(\epsilon)$. Our **process dynamic** is then given by:

$$(v, s, w) \rightarrow (v - e_i, s, w) : \delta_1(v^i)\beta$$

$$(v, s, w) \rightarrow (v + e_i, s - s_i e_i, w) : \phi_{\epsilon}(s, w, w)\delta_0(v^i)\alpha(w, v)$$

$$(v, s, w) \rightarrow (v + e_i, s - s_i e_i, \widetilde{w}) : \phi_{\epsilon}(s, w, \widetilde{w})\delta_0(v^i)\alpha(w, v)$$

- $(e_i)_i$ is the canonical basis of \mathbb{R}^N
- $\phi_{\epsilon}(s, w, \widetilde{w})$ gives the probability to jump in \widetilde{w} knowing s

Results:

We derive an equation for the slow weight dynamic alone, in which neurons dynamics are replaced by their stationary distributions. We work on the time scale $\tau_{\epsilon} = \frac{\mathbf{t}}{\epsilon}$ when $\epsilon \to 0$.

1.Invariant measure:

When $W_t = w$ is fixed, there exists a unique invariant measure π_w for the process $(V_t, S_t)_{t>0}$:

- Existence : Lyapunov function as in [4]
- Uniqueness: characterization of Laplace transform of π_w

We didn't find explicitly $(v,s) \mapsto \pi_w(v,s)$ but we studied its behavior near the diagonal $s_i=s_j$. We prove that it is not continuous in most cases at the diagonal:

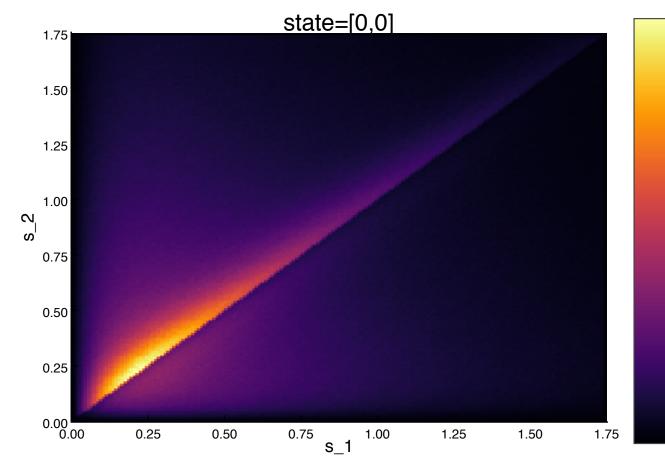


Fig: Invariant measure in neuron state (0,0), 2 neurons.

2.Slow fast analysis:

Let $(V_t, S_t, W_t)_{t>0}$ such that $(V_t, S_t) \sim \pi_{W_t}$ and (W_t) is the solution of the martingale problem associated to the operator C:

$$Cf(w) = \int_{E_2} Af(v, s, w) \pi_w(ds, dv)$$
 (1)

Using [5], we prove that $(V_{\tau_{\epsilon}}, S_{\tau_{\epsilon}}, W_{\tau_{\epsilon}})_{t \geq 0}$ converges in law to $(V_t, S_t, W_t)_{t>0}$ when $\epsilon \to 0$.

Discussion:

This time scale separation gives the infinitesimal generator of the weight dynamic on the slow time scale. However, we don't know explicitly π_w but its Laplace transform. Under some simple assumptions, we can get explicitly the dynamic of the weights which is a Markov chain on $\{(w_{ij})_{i,j}, w_{ij} = w_0^{ij} + k\Delta w \geq 0, k \in \mathbb{Z}\}$ with inhomogeneous jump rates depending on the Laplace transform of π_w . Thus, we don't need to simulate the all network any more, only the limit model. Moreover, we can analyze the weights dynamics. An example is given in simulations.

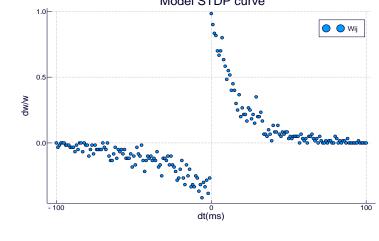
5.SIMULATIONS

Biologically coherent parameters:

Even if simple, our model depends on many parameters. First, let's detail the probability to jump:

$$p^{+}(s) = A_{+}e^{-\frac{s}{\tau_{+}}}$$
 and $p^{-}(s) = A_{-}e^{-\frac{s}{\tau_{-}}}$

Such functions enable to be close to **biological experiments**:



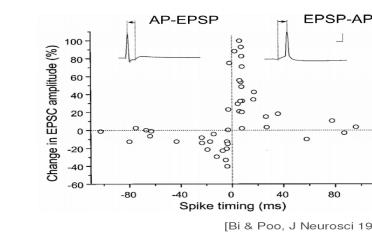
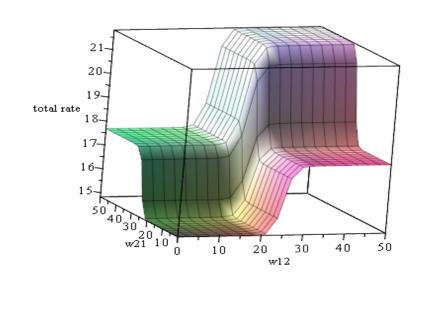


Fig: Bi-Poo experiment on our model compare to the real one

Parameters for the figure are: $A_{+}=1, A_{-}=0.4, \tau_{-}=2\tau_{+}=34ms$ as in [6]. These parameters have to be added to the first ones: β , α_m , α_M . Time of influence of a spike 1ms so $\beta \sim 1$. Firing rates of neurons are bounded by $\alpha_m \sim 0.01$ and $\alpha_M \leq \beta$. STDP parameters are in the following range: $\tau_{+/-} \in [5, 40], A_{+/-} \in [0, 1].$ Finally, $\epsilon \in [0.1, 0.01]$.

Analytic versus Numeric:

First, we wanted to visually show our limit model is licit. In simulations, an easy value to get is the sum of jump rates of weights:



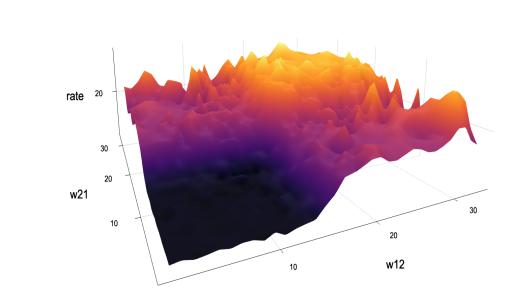


Fig: Analytic (left) and numerical (right) total jump rate of weights, 2 neurons.

We get similar results in the case of 2 neurons. In higher dimension it is hard to get equivalent analytic and numerical precision.

Weight divergence:

A big problem in plasticity models is the divergence of weights. We could have put hard bounds or soft bounds but we wanted to see in which limits weights diverged without them. We tested some criteria of non divergence of weights in our model. In particular in the article [7], such a criteria is to have the integral of the learning window negative. Nevertheless, it is not the case for our model. In a really simple case, our limit model enabled us to find parameters for which this criteria leads to a weight divergence.

One weight free and 2 neurons:

We get a birth and death process with w_{21} fixed, $w=(w_{12}, w_{21})$:

$$w_{12} \to w_{12} + \Delta w : r_{+}(w) > 0$$

 $w_{12} \to w_{12} - \Delta w : r_{-}(w) > 0$

10 neurons:

When depression is really higher than potentiation, weights seem to converge to a stationary distribution and have such trajectories:

Fig: Evolution of the weight.

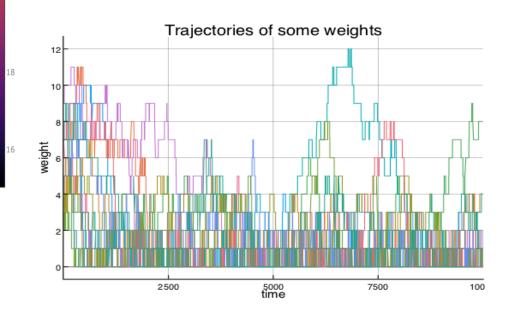


Fig: Plot of $r_{\perp}(w) - r_{\perp}(w)$.

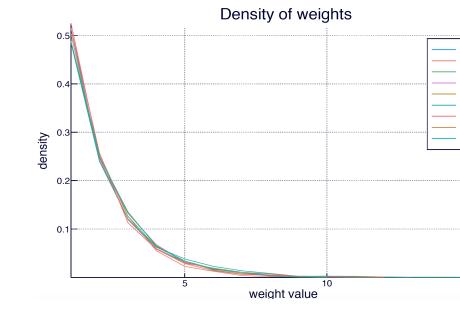
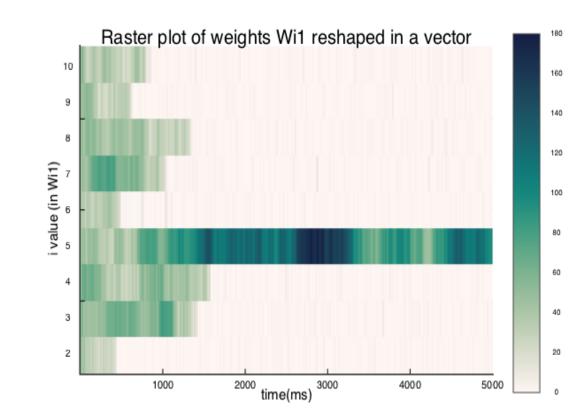


Fig: Plot of p^+ , p^- on the same graph.

However, initial weights play an important role. With parameters $A_{+}=0.8, \ A_{-}=0.9, \ \beta=1, \alpha_{m}=0.01, \alpha_{M}=0.5 \text{ and } \epsilon=0.1, \text{ we}$ have no divergence in short time with low initial weights and selection of one weight from big initial ones, $\mathbf{W_0^{i1}} = \mathbf{50}$:



The selected weight is different from one trajectory to another.

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6. CONCLUSION

We showed divergence of weights even when integral of the learning window is negative. Additive terms, depending on weights, seem necessary to avoid divergence in the context of biological parameters. However, our first mathematical results are encouraging for deeper study and our model showed more interesting behavior than those already presented: bidirectional as unidirectional connections can be strong.

7.PERSPECTIVES

Maths:

- Weights dynamics
- Mean field approximations

Modeling:

- Simulations to test other plasticity rules
- Neurons states from discrete to continuous