1 The Spiking Model

1.1 Temporally Asymmetric Hebbian Learning on Asynchronous Populations

An important component of the model presented in this paper is a temporally asymmetric Hebbian plasticity rule which changes the connectivity among populations depending on the timing of changes in their mean firing rate. Such a rule could be justified if given a pair of small neuronal populations \( A \) and \( B \), interconnected with synapses following an antisymmetric STDP rule, we could have that an increase in the firing rate of \( A \) followed by an increase in the firing rate of \( B \) would lead to an increase in the mean strength of connections from neurons in \( A \) towards neurons in \( B \), and a decrease in the mean strength of connections from neurons in \( B \) towards neurons in \( A \). Existing studies on populations of neurons with STDP connections indicate that the distribution of synaptic weights in a spiking network with additive STDP tends to become bimodal [23, 55, 58]. We prefer to use a weight-dependent STDP rule to avoid the hard nonlinearity at saturation and to better adjust to experimental data [46]. Weight-dependent STDP rules tend to produce unimodal distributions unless the right correlations exist between presynaptic and postsynaptic activity [23, 55]. A basic characteristic of these studies was the use of initial homogeneous random connections. For the spiking model in this section we instead assume the existence of densely connected neuronal populations; neurons in different populations communicate using STDP connections. This breaks the homogeneity in the network, and helps to avoid problems such as synfire explosions and the difficulties in creating excitation pathways with synchronous inputs and STDP plasticity ( [45]; Kunkel et al. 2010, submitted). In order to create connections between populations we stimulated them with Poisson spike trains, and found that by using the right timing in our inputs we could strengthen the projections from the first stimulated population to the second stimulated population, and weaken the opposite projections (as will be described below). It should be remarked that the model with spiking neurons only intended to show the feasibility of temporally asymmetric Hebbian plasticity at the population level; it is not concerned with sequential activity and is not meant to have the same dynamics as the firing rate model in this paper.

1.2 Methods

All neurons used integrate-and-fire dynamics with conductance-based synapses. Postsynaptic potentials were modeled by alpha functions. Populations 1, 2, and 3 consisted of 100 neurons each, with homogeneous parameters. The inhibitory population consisted of 80 neurons. Each neuron had connections to all the neurons in its corresponding population, and to half of the neurons in the other two populations; the connections between populations were randomly chosen. A more detailed description of the model is provided in tables 1-6. All simulations were implemented in NEST [21], and commented source code is available.

We used a simple model network of conductance-based integrate and fire neurons (figure 1A) to test our ideas. The network consisted of four populations, three excitatory (100 neurons each), and one inhibitory (80 neurons). The neurons in each population randomly sent projections towards half of the neurons in each of the other populations. The excitatory-to-excitatory connections used STDP synapses with a power law exponent \( \mu = 0.1 \), as described in [23]. Each neuron received 20000 independent Poisson spike trains, 16000 excitatory with a mean firing rate of 4 Hz, and 4000 inhibitory with a mean firing rate of 5 Hz. The neurons in each population had static all-to-all connectivity within themselves; inhibitory populations had no connections between themselves. Since the excitatory neurons had no constant inputs and were driven by independent Poisson trains, increasing the connection strength within a population could increase its coherence. The strength of the connections within each population was

![Fig. 1 Architecture of the spiking network. The spiking model consists of four excitatory populations (circles labeled E\(_i\)) and one inhibitory population. Arrowheads denote excitatory connections, and dots inhibitory connections.](image-url)
Table 1 Spiking Model Summary.

<table>
<thead>
<tr>
<th>Populations</th>
<th>Four: three excitatory, one inhibitory; external input</th>
</tr>
</thead>
<tbody>
<tr>
<td>Topology</td>
<td>All-to-all within populations, random divergent across populations</td>
</tr>
<tr>
<td>Connectivity</td>
<td>All-to-all within populations, random divergent across populations</td>
</tr>
<tr>
<td>Neuron Model</td>
<td>Conductance-based leaky integrate-and-fire</td>
</tr>
<tr>
<td>Channel Models</td>
<td>—</td>
</tr>
<tr>
<td>Synapse Model</td>
<td>Alpha functions</td>
</tr>
<tr>
<td>Plasticity</td>
<td>STDP on excitatory connections between different populations</td>
</tr>
<tr>
<td>Input</td>
<td>Independent fixed-rate Poisson spike trains to all neurons</td>
</tr>
<tr>
<td>Measurements</td>
<td>Spike times for all neurons, voltage trace for one neuron</td>
</tr>
</tbody>
</table>

Table 2 Populations in the spiking model.

<table>
<thead>
<tr>
<th>Name</th>
<th>Elements</th>
<th>Size</th>
</tr>
</thead>
<tbody>
<tr>
<td>E_{1,2,3}</td>
<td>IAF neurons</td>
<td>100</td>
</tr>
<tr>
<td>I</td>
<td>IAF neurons</td>
<td>80</td>
</tr>
<tr>
<td>P</td>
<td>Independent Poisson generators</td>
<td>380</td>
</tr>
<tr>
<td>O_{1}</td>
<td>Independent Poisson generators</td>
<td>500</td>
</tr>
<tr>
<td>O_{2}</td>
<td>Independent Poisson generators</td>
<td>500</td>
</tr>
</tbody>
</table>

Table 3 Connectivity of the spiking model.

<table>
<thead>
<tr>
<th>Name</th>
<th>Source</th>
<th>Target</th>
<th>Pattern</th>
</tr>
</thead>
<tbody>
<tr>
<td>E_{E_{int}}</td>
<td>E_{x}</td>
<td>E_{y}</td>
<td>All-to-all for x=1,2,3. Weight $w_{e_{epsc}}$, delay $w_{d}$. No autapses or multapses</td>
</tr>
<tr>
<td>E_{E_{ext}}</td>
<td>E_{x}</td>
<td>E_{y}</td>
<td>50 targets randomly selected for each source neuron, no multapses. Weight $w_{e_{epsc}}$, delay $w_{ed}$</td>
</tr>
<tr>
<td>E_{I}</td>
<td>E_{x}</td>
<td>I</td>
<td>40 targets randomly selected from I population for each source neuron, no multapses. Weight $w_{e_{epsc}}$, delay $w_{ed}$</td>
</tr>
<tr>
<td>I_{E}</td>
<td>I</td>
<td>E_{x}</td>
<td>50 targets randomly selected for each source neuron, no multapses. Weight $w_{e_{epsc}}$, delay $w_{ed}$</td>
</tr>
<tr>
<td>P_{E_{1}}</td>
<td>P</td>
<td>E_{x}, I</td>
<td>16000 excitatory Poisson inputs for each target neuron, each one with static rate $r_{ex}$</td>
</tr>
<tr>
<td>P_{I_{1}}</td>
<td>P</td>
<td>E_{x}, I</td>
<td>4000 inhibitory Poisson inputs for each target neuron, each one with static rate $r_{in}$</td>
</tr>
<tr>
<td>O_{1}</td>
<td>O_{1}</td>
<td>E_{1}</td>
<td>Each target neuron receives input from all generators in O_{1}, each one with static rate $r_{o1}$</td>
</tr>
<tr>
<td>O_{2}</td>
<td>O_{2}</td>
<td>E_{2}</td>
<td>Each target neuron receives input from all generators in O_{2}, each one with static rate $r_{o2}$</td>
</tr>
</tbody>
</table>

not enough to cause synchronization given the normal Poisson background input, but stronger asynchronous input could increase the coherence in the population to the point where most neurons would fire together (see figure 2A-D). This increase in synchrony was beneficial when creating connections between populations, as it would allow a presynaptic population to emit spike volleys which could drive many postsynaptic neurons beyond threshold, in a manner similar to a synfire chain model. The intermediate inhibitory population was included in order to maintain the architecture of the firing rate model to be presented below, but it also had a beneficial function in maintaining the stability of the asynchronous firing, and reducing the positive correlation between the excitatory populations.

As a first foray into the dynamics of our spiking model, we tested whether stimulation of population I produced by independent Poisson trains could strengthen its projections towards the other two excitatory populations (2 and 3). The idea was that by increasing the firing rate in I, the neurons in this population would cause neurons in 2 and 3 to fire, thus strengthening their STDP connections. The problem was that the neurons in I firing asynchronously would not provide enough excitation to drive the neurons in 2 and 3 beyond threshold. We overcame this in two ways. The first was to alter the balance between excitation and inhibition so that the distribution of voltages remained near threshold in the postsynaptic populations. Indeed, signals can propagate their firing rates when the subthreshold voltages of the underlying network have a distribution that is broad and close to threshold [54, 67]. The second solution was to have strong enough connections within the populations, so that they would fire synchronously...
The targets with mean voltages near threshold. We can  

is that the connections being formed are not specific,  

reduction of the background input. The reason for this  

changing the balance between the excitation and in-  

creating an ensemble target population (figure 2H).  

results are shown in figure 2. An interesting additional  

also depends on the balance between excitation and in-  

when an additional stimulus was applied to them. The  

coexistence of synchronous and asynchronous dynam-  

and inhibitory connections became stronger, and its  

asynchronous firing driving the activity, there is  

two populations are connected with weight-dependent  

a shift in the excitation and inhibition of the postsy-  

that its mean voltage is near threshold, and each tran-  

specify a target by changing the balance in its inputs so  

that its mean voltage is near threshold, and each tran-  

In the last paragraph we mentioned that the sta-  

Time of each spike, and identity of the neuron emitting it. Voltage trace for first indexed neuron.

<table>
<thead>
<tr>
<th>Name</th>
<th>Value</th>
<th>Name</th>
<th>Value</th>
<th>Name</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>(C_m)</td>
<td>250 pF</td>
<td>(E_L)</td>
<td>-70 mV</td>
<td>(E_E)</td>
<td>0 mV</td>
</tr>
<tr>
<td>(E_n)</td>
<td>-85 mV</td>
<td>(V_{reset})</td>
<td>-60 mV</td>
<td>(\theta)</td>
<td>-55 mV</td>
</tr>
<tr>
<td>(\tau_{synE})</td>
<td>0.2 ms</td>
<td>(\tau_{synI})</td>
<td>1.2 ms</td>
<td>(\tau)</td>
<td>20 ms</td>
</tr>
<tr>
<td>(\mu)</td>
<td>0.1</td>
<td>(\alpha)</td>
<td>1</td>
<td>(\lambda)</td>
<td>0.1</td>
</tr>
<tr>
<td>(w_{max})</td>
<td>2 nS</td>
<td>(w_{epsc})</td>
<td>1.5 nS</td>
<td>(w_{ed})</td>
<td>1 ms</td>
</tr>
<tr>
<td>(\varepsilon_{epsc})</td>
<td>0.2/1 nS</td>
<td>(\varepsilon_{ed})</td>
<td>1 ms</td>
<td>(\varepsilon_{epsc})</td>
<td>1 nS</td>
</tr>
<tr>
<td>(\kappa_{ed})</td>
<td>2 ms</td>
<td>(\kappa_{epsc})</td>
<td>-2 nS</td>
<td>(\kappa_{ed})</td>
<td>2 ms</td>
</tr>
<tr>
<td>(t_{ex})</td>
<td>4 Hz</td>
<td>(t_{in})</td>
<td>5 Hz</td>
<td>(\alpha_{1/0})</td>
<td>15 Hz</td>
</tr>
</tbody>
</table>

when an additional stimulus was applied to them. The  

as well as the balance between excitation and in-  

structured (Poisson) firing driving the activity, there is  

rule, the stability of the changes in the synaptic weights  

probability of the changes in the synaptic weights  

the stability of the changes in the synaptic weights  

that its mean voltage is near threshold, and each tran-  

that its mean voltage is near threshold, and each tran-  

In the last paragraph we mentioned that the sta-
Fig. 2 Formation of unidirectional excitation pathways and population ensembles. A-D) Spike rasters of the three excitatory populations and the inhibitory population. Independent Poisson stimuli are applied to population 1 from $t = 2000$ ms to $t = 2300$ ms, which causes synchronous activity. The activity in population 1 spreads to the other 3 populations, causing significant changes in the mean synaptic weight between populations. E) Voltage trace of the first neuron in population 1. F) Mean synaptic weight of the connections between populations 1 and 2. The formation of a unidirectional excitation pathway is observed here. G) Mean synaptic weight of the connections between populations 1 and 3. H) Mean synaptic weight of the connections between populations 2 and 3. These two populations experience a bidirectional strengthening of their connections.
scaled (in our case, one half of the maximum weight). The intuition behind this is that when a weight is below this value, potentiation is stronger than depression, and vice versa. Thus, assuming no correlations between the firing of both populations, the mean of their connections will drift towards this fixed point. Having one population excite another breaks the symmetry in the connections, but if the change is small the connections will drift back towards the fixed point, at a rate that will become faster at higher firing rates. By reducing the firing rates we can have changes in connectivity that last for longer periods. On the other hand, if the symmetry in the connections is broken by a large enough alteration, this might introduce correlations in the activity of the populations, and these correlations might reinforce the weight asymmetry. This idea will be used below.

We now present another mechanism that does not require selective modulation of the background input in order to increase connections along a specific pathway, or to increase their permanence. Synchronous activity is used to form connections, and asynchronous activity is used to consolidate them. We use the same model as before, but we reduce the initial value of the STDP connections between the excitatory populations. The effect of this is that the activity in an excitatory population will not have a large effect in the activity of the other excitatory populations. As seen in figure 3, we apply an external Poisson stimulus to population 1 for 300 ms, and another Poisson stimulus (duration 225 ms) to population 2 25 ms before the end of the first stimulus. This resulted in a fast decrease in the mean value of the synaptic weights in the 2 to 1 connections, and a gradual increase in the 1 to 2 connections. The fast changes in connectivity were due to synchronous activity in population 1 followed by synchronous activity in population 2, all due to the external inputs. The gradual changes came because once the 2 to 1 connections were decreased and the 1 to 2 connections increased, the probability of neurons in population 1 activating neurons in population 2 was larger than the probability of neurons in 2 activating neurons in 1, so that asynchronous activity increased the initial asymmetry in the connections. As can be seen in figure 3, the activity in 1 and 2 had little effect on population 3, which accounts for the small size of the variations in the connections involving this population. The overall effect of the activity in 1 and 2 was to increase the connections towards population 3, and decrease its outgoing connections, just as in the previous simulations, but on a smaller scale.

The simulation just described shows that by timing our inputs correctly we can selectively strengthen activation pathways among the populations. The possible consequences of a mechanism like this will be analyzed in the remainder of this paper using a firing rate model to describe the dynamics of a larger set of populations.

1.3 Conclusions

The model presented above indicates that temporally asymmetric Hebbian learning is a biologically plausible assumption. This model is not meant to illustrate the formation of excitation pathways in random networks with asynchronous inputs, because it assumes pre-existing populations with dense internal connectivity.

The existence of neuronal populations greatly simplifies the autonomous formation of unidirectional excitation pathways, and we presented two mechanisms which may achieve this. Both mechanisms take advantage of two traits which facilitate the formation of excitation pathways: background inputs which maintain the membrane voltages near threshold, and a connectivity within populations that allows them to operate asynchronously, and yet increase their synchrony in the presence of strong asynchronous inputs. This increase in synchrony is beneficial when using STDP to create excitation pathways, since it permits the emission of spike volleys in a manner similar to synfire chains, increasing the changes in the mean synaptic weights across populations. Depending on how strongly the synaptic weights are modified by the induced activity they may drift back towards a fixed point, or changes might consolidate through the spontaneous activity, with a speed that is dependent on the firing rate of all the involved populations. This is not an exhaustive description of the dynamics in populations that communicate with STDP synapses, but it suffices to establish the plausibility of the temporally asymmetric Hebbian plasticity used in the firing rate model of the main paper.

References

Fig. 3 Temporally asymmetric Hebbian plasticity at the population level. A-D) Spike rasters for the four populations. Independent Poisson spike trains are applied to the neurons in population 1 from $t = 1500$ ms to $t = 1800$ ms, and to the neurons in population 2 from $t = 1775$ ms to $t = 2000$ ms. The activity in the first two populations had little effect in population 3, but elicited synchronous activity in the inhibitory population. E) Voltage trace of the first neuron in population 1. F) Mean synaptic weight of the connections between populations 1 and 2. A unidirectional connection starts to form during the stimulus period, and consolidates through the spontaneous activity. G) Mean synaptic weight of the connections between populations 1 and 3. H) Mean synaptic weight of the connections between populations 2 and 3.


43. Meskenaite, V.: Calretinin-immunoreactive local circuit neurons in area 17 of the cynomolgus monkey, Macaca


