Complex Spiking Models: A Role for Diffuse Thalamic Projections in Complex Cortical Activity

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Abstract. Cortical activity exhibits complex, persistent self-sustained dynamics, which is hypothesised to support the brain's sophisticated processing capabilities. Prior studies have shown how complex activity can be sustained for some time in spiking neural network models, but network activity in these models resembled high firing rate seizure which would eventually fail, leading to indefinite quiescence. We present a spiking network model of cortex innervated by diffuse thalamic projections, called the Complex Spiking Model (CSM). The model exhibits persistent, self-sustained, non-periodic, complex dynamics at low firing rates. Multiple network configurations were tested, systematically varying diffuse excitation from the thalamus, strength of the local cortical inhibition and excitation, neighbourhood diameters, synaptic efficacies and synaptic current time constants. Complex activity in all the network configurations depended strongly upon the strength of the diffuse excitation from the thalamus. We propose that diffuse thalamic projections to cortex facilitate complex cortical dynamics and are likely to be an important factor in the support of cognitive functions.

Keywords: Spiking neural networks; complex cortical activity; diffuse thalamic projections; thalamo-cortical model; Izhikevich neurons.

1 Introduction

Cortical activity exhibits persistent ongoing complex dynamics [1, 2], with oscillatory activity which approximates a power law [3]. As activity shifts continuously throughout the brain, brain regions constantly couple (integrate) and de-couple (segregate) across multiple spatial and temporal scales [4-6]. This complex ongoing dynamics is hypothesised to support the brain's flexibility and sophisticated processing capabilities [1, 7, 8], including memory retrieval, planning and problem solving [9, 10]. During times when the brain is not actively processing sensory stimuli or task-related events, and as such is in a state known as the 'resting' or 'default-mode' state, brain activity is concentrated in a well-defined sub-network including regions of frontal and association cortices [11]. These are regions usually associated with higher-level cortical processing.

The thalamus has strong reciprocal connections with the cortex. A set of connections known as the *specific* thalamo-cortical connections originate in distinct thalamic nuclei and terminate in distinct cortical regions; one of the primary roles of these connections is to convey perceptual stimuli to the cortex. The *diffuse* connections, on the other hand, project non-specifically from the thalamus to many cortical regions, notably frontal and association cortices; brain regions that are known to be important for cognitive functions such as memory retrieval, planning and problem solving. There is a significant degree of overlap between these regions and those identified as belonging to the default-mode network. Activation of any of the regions of the thalamus from which these diffuse connections arise results in the recruitment and co-activation of many or all of the other regions, and consequent propagation of activity to all connected regions of the cortex [12]. Thus the diffuse thalamo-cortical projection system is implicated in general cortical arousal and control of gross cortical activity.

Previous studies have shown how complex activity can be sustained for some time in spiking neural network models with connection delays [4, 13]. These models utilised small-world connectivity as the structural facilitator of dynamical integration and segregation. The sparse long-range connections encouraged propagation of activity in self-perpetuating loops, provided that the connection densities and propagation delays were appropriately matched and within well-defined ranges. However, activity in these models was characterised by repetitive population- or cluster-wide oscillations; the complexity manifested only as unpredictability or 'jitter' in individual neuron spike times while population activity remained semi-synchronous and seizure-like. Such activity is not observed in healthy, functioning cortex. Additionally, the activity in these models was not indefinitely self-sustained; it would always eventually fail and return the networks to the quiescent state.

Recent work has shown how complex, non-periodic, low firing-rate, and indefinitely self-sustained activity can be supported in a cortical network model [14]. The cortical network combined local neighbourhood connectivity with a number of long-range connections and all connections were subject to activity-dependent facilitation and depression. The network activity did not enter seizure or quiescence, and activity patterns did not repeat periodically despite network dynamics being deterministic and network input being constant. However, the network incorporated a single artificial globally-connected inhibitory neuron and no local inhibition. In the current study, we show that complex, self-sustained activity can be achieved with a cortical model incorporating realistic thalamic connectivity and local inhibition, and that the thalamic connectivity, analogous to the global inhibition of the previous model, is in fact required to support complex activity in the cortical neurons.

2 Complex Spiking Model (CSM)

We present a spiking network model of cortex innervated by diffuse thalamic projections, called the Complex Spiking Model (CSM) (see Fig. 1). The connectivity from the diffuse thalamic nucleus (*Diffuse*), to the cortex, to the reticular nucleus (*RN*) with inhibition back to the diffuse nucleus, is assumed to have a role in cortical activation control in brains, since if cortical activity rises, inhibition to the diffuse

nucleus increases (through the reticular nucleus), which decreases the excitation being sent to the cortex.

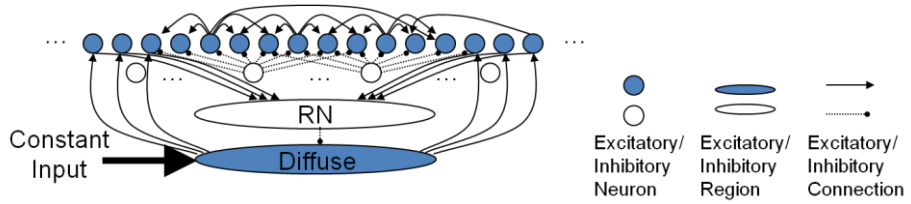

Fig. 1. The Complex Spiking Model (CSM) used for this research (not all connections shown). The model is structured as follows: Excitatory cortical neurons (small shaded circles) are innervated by diffuse thalamic projections (from shaded oval). The thalamic nucleus from which the diffuse connections arise receives continuous steady (not time-varying) input current. Cortical neurons project back to the thalamic reticular nucleus which inhibits the diffuse nucleus, completing the diffuse thalamocortical loop. Cortical neurons excite each other within a small neighbourhood radius, inhibit each other through local interneurons within a larger inhibitory radius, and project a small number of random long range excitatory synapses to other cortical neurons. All cortical excitatory synapses are subject to activity-dependent facilitation and depression. For details of the cortical neuron and synapse implementation see [14]. RN – thalamic reticular nucleus; Diffuse – diffuse (intralaminar) thalamic nucleus.

The CSM was constructed using Izhikevich model cortical neurons [15], and the reticular and diffuse thalamic nuclei were each implemented as a single analog neuron with output equal to the sum of its input currents. Synapses were modeled as postsynaptic currents, initiated by presynaptic spikes, that exponentially decayed with characteristic time constants of 35 to 50 milliseconds to emulate the long-duration synaptic currents generated by NMDA receptors [16]. To implement short-term synaptic dynamics, a combination of depression and facilitation was used [17]. For synaptic depression, all of the currently-available proportion of neurotransmitter p at each synapse was released at the occurrence of each spike; p recovered back to unity with time constant d. The rate of change of p for neuron i, $\dot{p}_i$, was given by:

$$\dot{p}_i = -\delta(t - t_i)p_i + \frac{(1 - p_i)}{d_i} \qquad \ldots(1)$$

where δ is the Dirac delta function, t_i is the time of the last spike from presynaptic neuron i. With dynamic synapses the calculation for all the synaptic currents into a postsynaptic neuron, $I_{dynamic}$, becomes:

$$I_{dynamic} = \sum_i w_i p_i \qquad \ldots(2)$$

where w_i is the synaptic weight from neuron i.

3 Methods

To measure the complexity of the spiking activity generated by the network, the interspike intervals (ISI) for each neuron were individually determined, then the standard deviations (SD) for each neuron were calculated, and finally all standard deviations were averaged to calculate the mean interspike interval standard deviation (ISI-SD) for the network. Networks exhibiting either random uncorrelated activity or seizure-like dynamics both have low ISI-SD, since the lack of any segregation in network activity reduces the spread of interspike intervals. In contrast, complex activity is characterised by a high ISI variability, since neurons fire rapidly when integrated into an active neural assembly, then can remain quiet for extended periods while segregated.

Initially the network was set up as in Fig. 1, then network activity was simulated for 10,000 seconds to check that neither seizure nor activity failure eventuated, and that complex self-sustained activity persisted for the entire simulation. Subsequently, multiple network configurations were tested with different values of network parameters. Parameters which were varied included strength of the local cortical excitation and inhibition, neighborhood diameters of excitation and inhibition, and synaptic current time constants, along with the strength of the diffuse excitation from the thalamus. For each network configuration, the mean network firing rate and ISI-SD measures were calculated. These measures were plotted on two dimensional graphs called *heat maps*, where each point represents a different network configuration and is shaded according to the rate or measure at that point. Heat maps provide a succinct visualisation of large numbers of simulations showing the range of network dynamics in different regions of parameter space. The heat maps in these studies use one network per pixel, and 100x100 networks per map.

4 Results and Discussion

The CSM exhibits persistent, self-sustained, non-periodic and complex dynamics at low firing rates (see Fig. 2). Local assemblies of neurons spontaneously coalesce and disintegrate, mediated by the neighbourhood excitation. Multiple assemblies are active simultaneously across the network, mediated by the long range connections, and firing of these assemblies correlates and decorrelates over time as their phase relationships shift. One assembly may be active several times over the course of network activity, but never with an identical set of other assemblies simultaneously active elsewhere in the network. Activity patterns never repeat and there is no periodicity in the observed network dynamics (see Fig. 2 top-left), yet there are clear oscillations in network assemblies which correlate and decorrelate over time (see Fig. 2 bottom-left). These dynamics lead to a power law-like power spectrum (see Fig. 2 right). All these results hold for simulations of activity of up to 10,000 seconds duration, despite network dynamics being deterministic and network input being constant. The network activity shown in Fig. 2 has a high ISI-SD of 0.85 sec, despite neurons firing at 40 Hz or more when part of an active assembly, since outside of this assembly activity, each neuron typically falls silent for seconds or even tens of

seconds. These results replicate those obtained in earlier work [14], but eliminates the artificial globally-connected inhibitory neuron, replacing it with a model of local inhibition and thalamic connectivity.

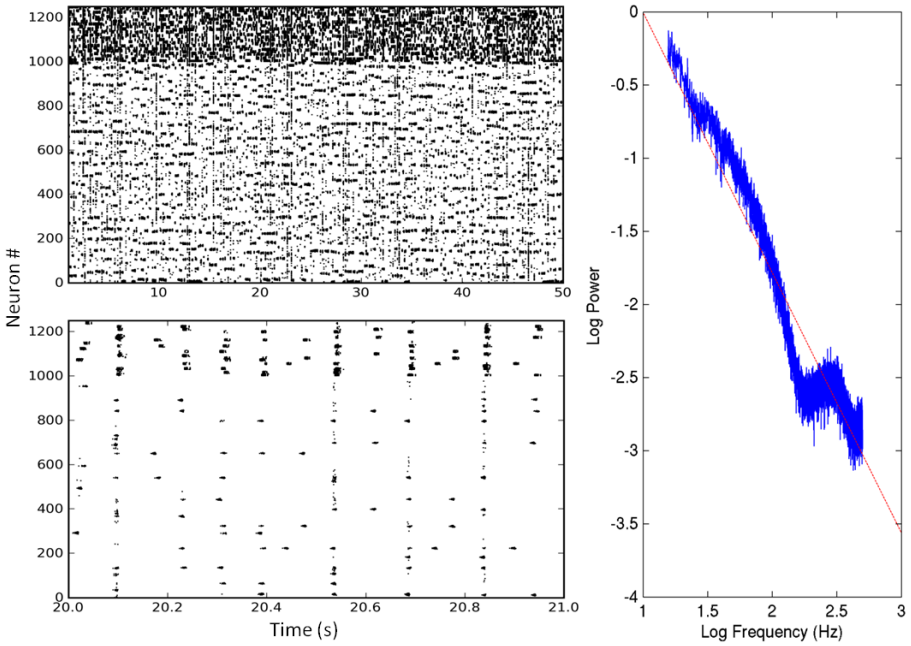

Fig. 2. Typical dynamics of the CSM. Activation of assemblies of neurons can be seen to correlate and decorrelate over time, indicating complex activity. Top-left: Raster plot showing spiking behaviour of 1000 excitatory neurons and 250 inhibitory interneurons (Y axis) over 50 seconds (X axis). Dots show spike times; many spikes appearing together indicate activation of a cell assembly and appear as horizontal lines. The network dynamics is complex, activity patterns are not seen to repeat, and the network does not enter seizure or quiescence. Bottom-left: Zooming in on one second of activity from the raster plot on the top-left shows oscillatory dynamics and sparse spiking patterns giving rise to low sustained firing rates (mean rate = 1.6 Hz). Right: As indicated by the straight line fit (dotted) on the log-log graph, the power spectrum of the activity shown on the left approximates a power law, similar to spectral activity seen in the brain [3].

To show that the thalamo-cortical loop in the CSM facilitates the generation of complex activity, we tested multiple network configurations. Two network parameters were varied – the diffuse excitation from the thalamus, and the strength of the local cortical inhibition. The mean network firing rates and ISI-SD measures were plotted on *heat maps* to facilitate visualisation of the network dynamics as parameters were varied (see Methods). The systematic mapping of the parameter spaces shows that complex activity is dependent upon the strength of the diffuse excitation from the thalamus (see Fig. 3). Provided that the diffuse excitation is in a defined range (0.015 to 0.06 synaptic conductance), then a broad range of local inhibition strength suffices for low firing rate, high complexity dynamics to ensue, and the strength of the local inhibition within this broad range has little effect on the dynamical complexity. For

diffuse excitation outside the identified range however, complex activity occurs only for a very narrow range of local inhibition strength around 0.05.

Simulation studies with an extensive range of other network parameters were conducted, varying strength of the local cortical excitation, neighbourhood diameters of excitation and inhibition, and synaptic current time constants along with the diffuse excitation from the thalamus (results not shown). All the networks simulated yielded comparable findings to that shown in Fig. 3: without the global control of network activity afforded by the thalamo-cortical loop, complex dynamics did not arise.

One way of understanding the results is as follows: When global network control is absent, network activity reduces to numerous interconnected pockets of activity that can be mutually excitatory due to activity propagating through the long range connections, but cannot be mutually inhibitory (via the thalamo-cortical loop). Lack of mutual inhibition causes activity to spread rapidly through the network, resulting in simple seizure-like dynamics. By contrast, with the thalamo-cortical loop functionally active, unchecked spread of excitatory influences in the network cannot occur, since any increase in total network activity immediately exerts an influence across the entire network by reduction of diffuse thalamic input.

The CSM presented in this paper demonstrates how complex ongoing activity can result from the interplay between the varying levels of excitation arising from the diffuse thalamic nucleus, the long range cortical connectivity, and the excitability of any given neuron or neural assembly as dictated by its own history of prior activity. If a neuron or assembly has recently been firing, then its synaptic connections with its local neighbors and its long-range partners will be reduced in efficacy due to synaptic depression [17]. Therefore, even if it is receiving strong excitation from its long-range partners and strong input from the diffuse nucleus, it will be unlikely to fire. Other neurons or assemblies which might not be as strongly innervated by excitatory influences may nevertheless fire instead, explaining why the same assemblies don't repeatedly fire in unison despite being strongly connected. Because there are a large number of connections between multiple potential assemblies in the network at any one time, and each assembly has its own unique firing history and hence excitability state, the combinatorial explosion of possible active assemblies, critically mediated by excitation from the diffuse thalamic nucleus, ensures that complex activity is persistent and never gives way to either seizure (runaway activity) or quiescence (activity failure).

5 Conclusion

This study is the first to demonstrate computationally that complex, non-periodic, self-sustained cortical activity can be facilitated by diffuse thalamic connectivity. Global activation control afforded by the diffuse thalamic nucleus facilitates complex dynamics in the Complex Spiking Model (CSM) because global summation of activity can reliably cause segregation of activity across the entire network. Without global control, network activity reduces to numerous interconnected pockets of activity that can mutually integrate due to activity propagating through the long range connections, but cannot mutually segregate. As is the case in seizure or random activity states that lack segregated activity, spike train complexity is greatly reduced.

Based on the simulations presented in this study, we propose that diffuse thalamic projections to cortex facilitate complex cortical dynamics and are likely to be an important factor in the support of cognitive functions.

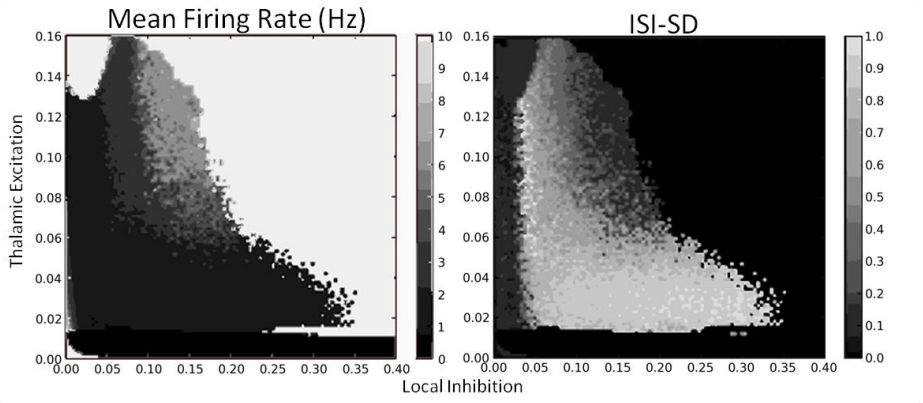

Fig. 3. Heat maps showing CSM dynamics for a range of thalamic excitation and local inhibition strengths. Networks were simulated with varying thalamic excitation (0-0.16 synaptic conductance, in 100 steps) and local inhibition (0-0.4 synaptic conductance, in 100 steps) resulting in 10,000 simulations in total (with 1 simulation per parameter combination). Low firing rate, high complexity dynamics can be seen to occur robustly for diffuse thalamic excitation within a defined range. Left: Heat map showing mean firing rate, calculated as the total number of spikes fired by the excitatory neurons in the network divided by both the number of neurons and the simulation time in seconds. Mean firing rate depends on the initial parameters set for the network (black – no firing; white – high firing rate seizure). For high values of local inhibition and thalamic excitation (top-right corner), the network enters seizure, shown by firing rates at or above 10 Hz (white). Right: Heat map showing interspike interval standard deviation (ISI-SD, see text for definition). Complex activity, indicated by high ISI-SD (light colours), occurs for a broad range of local inhibition as long as diffuse thalamic excitation is within a specific range. By comparing both heat maps, it can be seen that in the complex activity region (light colours in the heat map on the right), mean firing rate is low (see corresponding dark grey regions in the heat map on the left).

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