Contrast dependent response latency in a spiking neural network

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Abstract

The onset latency of single neuron responses in the visual system depends strongly on stimulus contrast. While in V1 latency increases are tens of ms, in higher visual areas (IT) the latency can increase 200ms at the lowest contrast (Oram at al 2002). We present a layered neural network model of noisy integrate-and-fire neurons. Crucially, the model has strong recurrent connectivity and synapses with short-term synaptic depression. With these realistic ingredients, the model reproduces the contrast dependent latencies. The model furthermore predicts a strong dependence of the spiking statistics on the contrast and time after stimulus onset. We analyse the response statistics predicted by the model and compare them to the data. The study shows that recurrence and short-term synaptic depression are important to explain dynamics and statistics of visually evoked responses in higher visual areas.

Introduction

Neurons in the superior temporal sulcus of the macaque exhibit strong dependence of response latency on stimulus contrast (see figure 1), with the lowest stimulus contrast leading to response latencies of up to 200 ms (Oram at al 2002). Figure 2 displays some of the latency data from different levels in the visual system hierarchy.

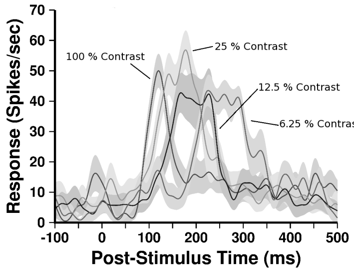

Figure 1: Single Neuron in STS responses to different contrasts.

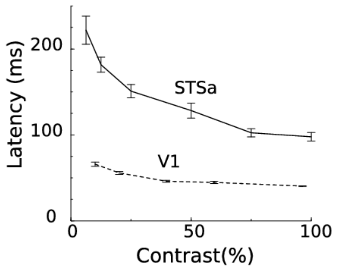

Figure 2: Average response latency from 19 V1 neurons and 18 STS neurons against stimulus contrast (Wiener et al, 2001, and Oram et al, 1999)

Here, we extend an existing rate-based model of this phenomenon. The basic premise is that the network uses recurrent, depressing synaptic connections. When the input contrast is high, the recurrent connections depress quickly, allowing fast synaptic transmission. When the contrast is low, the recurrent connections allow the integration of the input signal over time, reducing it's susceptibility to noise.

Model Architecture

Figure 3 shows the architectures of the models involved. They are colour coded to allow for cross referencing. The neurons were modelled as integrate and fire neurons (200 per layer), with noisy bias currents injected into them to provoke a background firing rate, and prevent synchronisation. The synapses were all conductance based, with the activation profiles of the NMDA synapses modelled as a difference of two exponentials, with a rise time of 1.5ms, and a fall time of 150ms, and the excitatory synapses modelled by a single exponential decay with time-constants of 5ms. Each population-to-population connection indicates 10% connectivity between two populations, with recurrent synapses twice as strong as the feed-forward ones. The gain was tuned so that an input of 50 Hz leads to an output of 50 Hz.

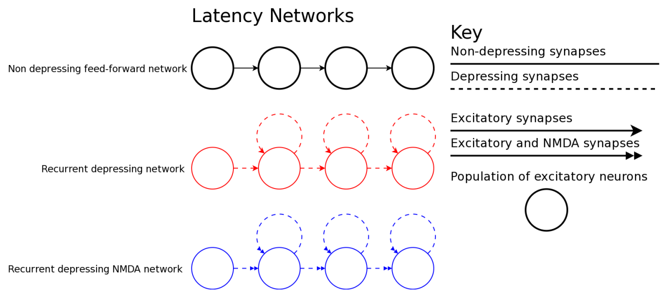

Figure 3: Architecture of different latency networks

Model Response

In figure 4, the average response (1ms binned PSTH, smoothed by 5ms Gaussian, averaged over neurons) of the third layer of each network is shown for different input strengths (shaded intervals are standard error). The recurrent networks both show strong non-linearities, while the feed-forward network transmits the signal more linearly.

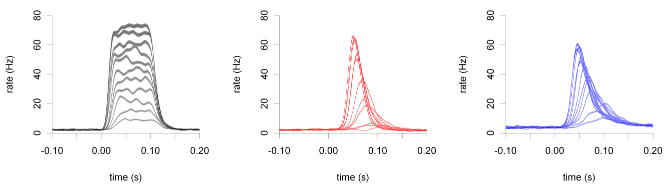

Figure 4: Average PSTH in third layer of the three latency networks, with decreasing peak matching decreasing stimulus strength.

Model response latency

The average half-maximum latency for all the signals which successfully propagate to the third layer is shown in figure 5. There is a clear contrast dependent latency effect for the depressing, recurrent networks, compared to the feed-forward network.

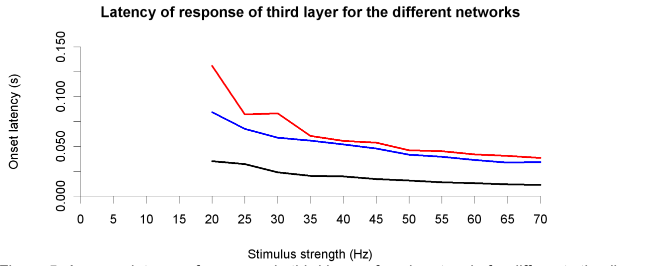

Figure 5: Average latency of response in third layer of each network, for different stimuli strengths.

The non-linearities of the recurrent networks lead to a difficulty in propagating signals to deeper layers. Figure 6 gives the average response latency of the same networks, extended to five layers The contrast-dependent part of the latency is reduced, and the signal does not propagate reliably for the two lowest strength inputs in the recurrent network. This is likely due to the layer-by-layer non-linearity progressively reducing the difference in response amplitudes, thus leading to more similar latency additions. Overall NMDA synapses reduce the contrast dependence of the latency, perhaps by further contributing to response normalisation.

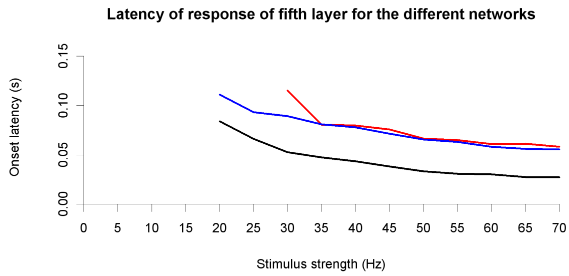

Figure 6: Average latency of response in fifth layer of each extended network, for different stimuli strengths.

Stimulus-dependent drop in response variability

In figure 7, the Fano factor of the experimentally observed spike count of a sliding 100ms window is shown. In figure 8, the corresponding measure is shown for the recurrent network. The spiking network shows a drop in Fano factor tied to the stimulus onset, in a similar fashion to the experimental data. Overall, the Fano factor remains much lower, however.

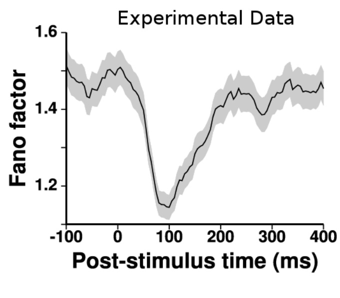

Figure 7: The observed Fano factor from the experimental data, over a 100ms window.

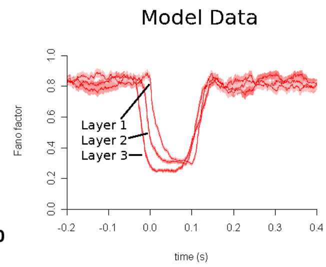

Figure 8: The Fano factor over a 100 ms sliding window for the recurrent network.

Conclusion

- Long latency responses are reproduced in a spiking neural model incorporating recurrent, depressing feedback connections.
- The nature of the recurrent depressing architecture leads to a strong dependence of response latency on stimulus strength.
- Inclusion of NMDA synapses reduces the contrast-dependence of the response latency.
- The Fano factor of the spiking model shows a drop, as it does for the experimentally observed data. The overall Fano factor from the model is much lower however.

So far, the results from the spiking model correspond well with the rate based model in terms of contrast dependent latency, however, the spiking model suffers more markedly from a problem with signal propagation. Whereas the rate-based network can propagate signals well beyond the third layer, the spiking model suffers from non-linearity problems which act to prevent this – and the non-linearities strongly reduce the contrast dependent element of the response latency. Expanding the model to include heterogeneous, parallel pathways might counteract this.

More work is needed to examine the connection between the experimentally observed statistics, and those seen in the model. This is still under active consideration.