Hi Riccardo,
Thanks for all the links above and I have noted and printed the relevant.

Just a short summary of what we discussed yesteray - minutes of the meeting:

Physiological observation: At a certain base level luminance L0, a change in contrast results in amplitude modulation in the LGN cells (relay cells?).
An increase in luminance (L1), shows a similar amplitude modulation, but now there is also a frequency modulation, i.e. the frequency of oscillation increases with increased contrast.
With further increase in luminance (L2), there is a frequency modulation with varying contrast. However, there is no further amplitude modulation.

We would like to mimic the above phenomena with the simple LGN model with Izhikevich neurons.

In the current model, we have 140 neurons. We make multiple instances of this, where each model is a node. Nodes are connected in a circular ring formation. Desirable to have 100 - 200 nodes - so we will have a total of 14000 - 28000 neurons.

The DC bias to each Izhikevich neuron (parameter i_offset) will correspond to the luminance.
There can be two manners of implementation to the the ring network mentioned above:
Loom: where the luminance is first spot applied to only one node of the ring, and then gradually increased to cover all the nodes in the ring.
Bar: This is equivalent to a passing light bar, and therefore the DC bias will be moved from one node in the ring to the other, applied to a certain node over a fixed length of time.
The overall progress of the luminance for the above-mentioned three cases will be kept as:
alpha * i_offset,
where alpha=0.1 (L0), 1.0 (L1) and 10 (L2).

Inter-node connectivity in the ring will be between the nearest neighbours only. Also, connections will be between the TCR and TRN, reciprocal, of neighbouring nodes, as well as between the TRN s.

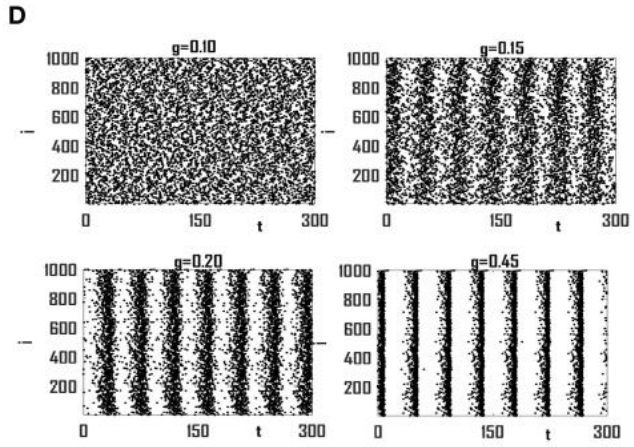
The variation of contrast will be simulated by external current inputs - which may be a base Poisson input. So we increase or decrease the Poisson frequency to vary contrast.

While changing the probability of connections (i.e. the number of estimated projections between neurons) will need to be set with trial and error - Riccardo came up with a novel thought:
A desirable asynchrony (randomness) in the output may be brought about by varying the initial membrane potential of the neurons in a population.
To elucidate - say our Relay cell population has 80 neurons. And let the Izhikevich neuron firing thresholds are set at -45 mv. Then, we could set the initial voltage for each of the 80 neurons in the population to be generated randomly from between -45 to -65 mV so that each neuron will therefore bound to start firing at different instances at the beginning. We will then have to see how they synchronise with increased luminance.

Alternative: to keep the neurons desynchronised, at least when their connectivity weights (w) are small we can set different Idc values for each neuron e.g. by sampling from a Poisson distribution. This seems to work nicely in

https://www.frontiersin.org/articles/10.3389/fncom.2018.00059/full

see figure 4D (g here are the weights of connections – note however this is simply a network of excitatory neurons connected to each other) :



Desirable goal of the work will be to:

Use the model to identify regimes in which the contrast evoked oscillations in TCR neurons are

- [AM] Amplitude modulated – different contrasts result in different amplitude of gamma oscillations with no change in frequency of these oscillations
- [AM & FM] Frequency & Amplitude modulated - different contrasts result in different amplitude and frequencies of gamma oscillations
- [FM] Frequency modulated - different contrasts result in different frequency of gamma oscillations with little or no change in frequency of these oscillations

Hypothesis:

The different regimes could be obtained by changing the average amplitude of of the Idc current where the overall progress of the luminance for the above-mentioned three cases will be kept as: alpha * i_offset, where alpha=0.1 (L0), 1.0 (L1) and 10 (L2).
This current will be driving both IN and TCR neurons.

In order to obtain the different regimes it is possible that the relative excitation of TCR and TRN will go in different directions.

[AM] This regime can be driven by TRN oscillations. Since TRN do not directly receive visual input (they only get it from TCR) then when TCR are not very active – in dim light – then TRN oscillations will be driven by a constant, contrast independent DC. This is the equivalent of an ING gamma model (Whittington MA (2000) J Psychophysiol **38**:315–336, doi:10.1016/S0167-8760(00)00173-2, pmid:11102670). The TCR activity will then be turned on and off by TRN oscillations always at the same frequency irrespective of the contrast of the stimuli.
Across multiple nodes we expect to see a global oscillations in TRN neurons.

[AM & FM] This regime can be driven by interplay between TRN-TRN oscillations and TCR-TRN oscillations. Increase in TCR will speed up TRN response that in turn will shut down TCR faster.
We do not expect to see global oscillators across TRN or TCR nodes.

[FM] This regime is harder to understand. It is possible that it arises from the interplay between the baseline oscillatory frequency from nodes that do not receive contrast stimulus with nodes that receive it. This interplay plays out at the level of TRN receiving both contrast related TCR activity and baseline TCR activity.
We expect to see global oscillators across TRN - TCR nodes.

I hope I remember all the salient points and doables. And also have understood correctly. Please do let me know if I have left out any. Or indeed any other.
cheers
Basab