1. Comparing several models: Why we observe increase in resting membrane potential for one of the models (Godlman 2001), but not for others?

2. How can one switch from bursting to spiking? Target parameters:

    - intracellular calcium concentration (effectively setting V_ca if model is Ohmic)
    - max conductance of T-Type channels
    - max conductance of L-type channels
    - max conductance for persistent sodium channel

3. Helicon-R5 synchronization: why did we observe 100ms delay in the model after helicon activation?

    - Shifted state was observed, with helicon cells leading oscillation. Hoever, this does not align with the assummption, that R5 causes SWA in helicon.
    - Possible mechanism: 'early activation' via gap junctions (as described in section 1)
    - For now, correlation was used to observe synchronization. However, correlation does not mean causality.
    - It might be interesting to see the time lag correlation between dFSB-helicon and dFSB-R5 in the SD condition. Or even better - granger causality. (Reassessing hierarchical correspondences between brain and deep networks through direct interface `https://www.science.org/doi/10.1126/sciadv.abm2219`)
    - Citations from Lauras thesis:
        - "In the Down state, Helicon is entrained to R5's compound rhythmicity via excitatory coupling. This leads to a relatively short offset (7 ms) between the two signals"
        - "For Helicon in the Down state, we find a much larger and negative offset of -77 ms (fig. 2.34a). We assume this is because Helicon now also receives inhibitory inputs from R5 neurons which prevent Helicon from firing and therefore lead to a small anti-phase correlation between the two signals."
    - Citation From Manuscript:
        - (Simulations). "This is also in line with our experimental data, which show that the balance controls the degree of synchronization between excitatory and inhibitory drive and determines whether the networks are in the shifted or synchronized configuration"

- Remarks
  - In the Lauras thesis, in the second note it should be written ”Up State” instead of ”Down State”. However, this state corresponds to daytime rather than night. Thus this will not explain the experimental observations (shifted state at night)
  - In manuscript, 1) there is no inhibition from R5 to helicon at night (in the model). Thus, the temporal shift might be due to the synaptic time constant between helicon and R5, rather than interplay between excitation and inhibition between R5 and Helicon. Synaptic time constant was set to be 100 ms (similar to resulted time delay between helicon and R5). Thus, when additional input was provided to helicon, here helicon might drive R5 and R5 might burst due to intrinsic properties.

4. Raccuglia: frequency of R5 activation is important. Actitation was done by optogenetics. If bursting is mediated by hyperpolarization activated current, then it can be that optogeneetically one directly activates fast system. Thus, you will need specific frequency of activation to induce similar effect (intrinsic bursting 1Hz).

5. What are the ”connection paths” from R5 to dFSB cells based on connectome (directed graph)? E.g. R5 - Helicon - dFSB. Should be easy to look into