Detailed Model of the Glomerular Networks in the Olfactory Bulb

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General Introduction

This detailed neural model was created to simulate the various computational properties of the glomerular level of the olfactory bulb, such as the Non-Topographic Contrast Enhancement as well as spike synchronization effects.

The Dimensionality Problem

The olfactory sensory modality is a special one amongst the senses due to its complexity. The odorspace is spanned by a virtually unlimited number of ligands. To perform any sort of meaningful computation in it, the olfactory bulb must have a basis for it. The most basic computation a sensory system can do is distinguishing between two stimuli, a task aided by contrast enhancement.

Given the size of the odorspace, it is impossible to store all of its basis vectors in the genome.

Picking out a small subspace to store creates a problem when the animal encounters odors not inside this reduced space, making the olfactory bulb function poorly.

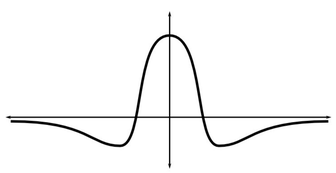
Training the network on an environment is the same as storing a smaller subspace: the organism will run into trouble sensing odors in other odor environments.

Solution via NTCE

The olfactory bulb receives its input from the Olfactory Sensory Neurons (OSNs) that express chemical receptors on their membranes. This has the effect of automatically picking out the perceivable subspace from the odorspace - exactly what we were trying to do before: the odorspace was always there, encoded by the ligand-receptor interactions.

A given family of OSNs expresses a single type of receptor protein and converges onto a single glomerulus, where via synaptic connections the incoming signal is transformed by a Mexican Hat function. This is the first part of Non-Topographic Contrast Enhancement, as it depends solely on the activity of a single glomerulus.

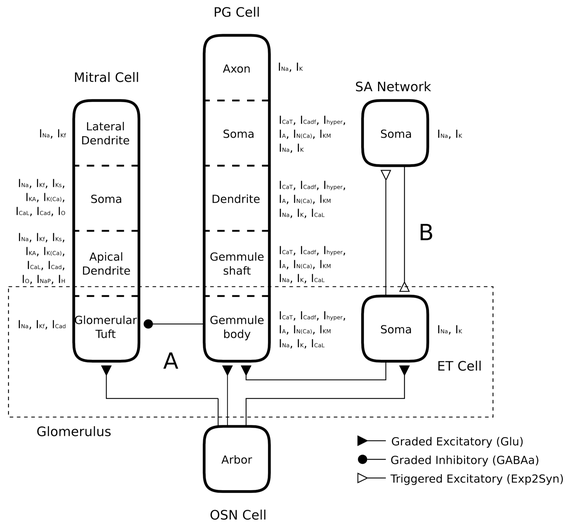
The second part of NTCE provides gain control for the system by measuring the average activity level of each glomerulus, and delivering uniform inhibition to all of them. The two parts combine produce a odor concentration independent contrast enhancement.



The Mexican Hat function

Methods

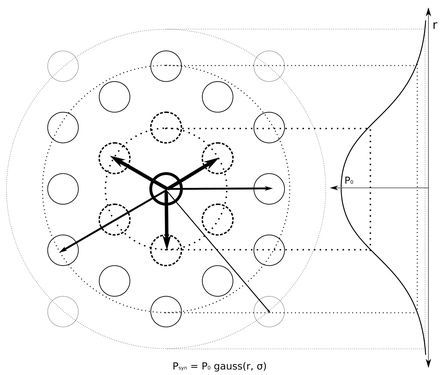
The model consists of physiologically detailed, multi-compartmental models of neurons, connected by simulated synapses. The network is implemented in the NEURON modeling environment.



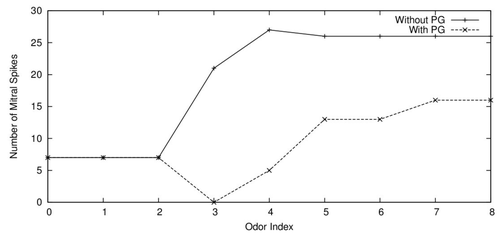
The layout of the model. Graded synapses are dendro-dendritic, and triggered synapses are axo-dendritic. Ionic currents present in each compartment are listed to the side.

A. The OSN population (modeled by a single compartment cell) excites both the Mitral and the Periglomerular (PG) 1 cell. PG1 in turn inhibits the Mitral cell. Mitral cell is the the principal output cell of the olfactory bulb. This so called paradoxical synapse mediates NTCE part I effect, as the dendritic spines of the PG cell have high input resistance, and thus inhibit the Mitral cell at intermediate odor affinities.

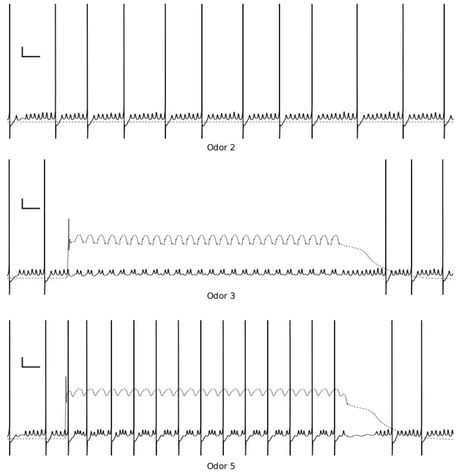
B. The OSN population also excites the External tufted (ET) cells which communicate between other ET cells via the Short Axon (SA) cells: connections that can span multiple glomeruli. This set of connections mediates the NTCE part II effect. The probability of the synapse forming between any given ET and SA cell, and SA and ET cell is governed by Gaussian probability distribution, as shown below. SA→ET connections have a large enough σ to yield a uniform excitation of the entire network, delivering uniform inhibition via the PG2 cells. r is measured in glomerular radii.

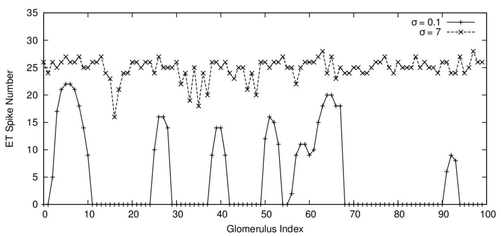


Results and Discussion

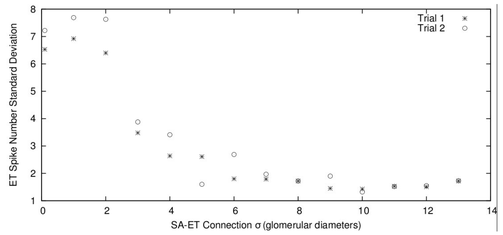


NTCE part I effect. Odors of increasing affinity were presented to the simulated glomerulus (odor 8 has the highest affinity). Dashed trace shows the Mexican Hat modulation as a result of the inhibition of the PG cell. Solid trace shows the control with the PG→Mitral synapse removed. Some representative traces of the PG (dashed) and Mitral (solid) membrane potentials are shown below. Oscillation seen in the PG trace is the result of simulated respiration (8Hz). The effect is clearly dependent on the slow T currents present in the PG1 cell (responsible for the plateau potential).

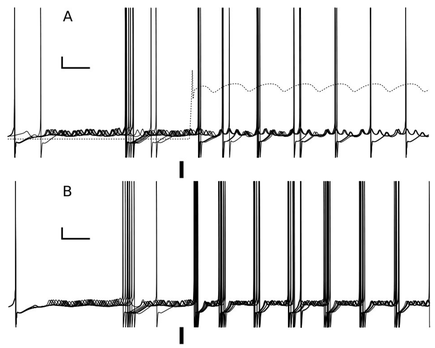




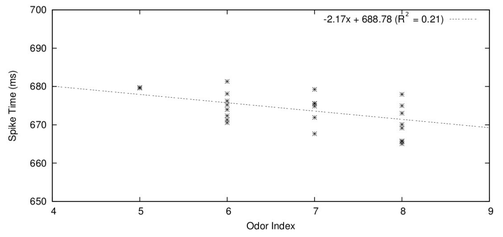
NTCE part II effect. A complex odor is presented to a 10x10 glomerular simulated olfactory bulb. Solid trace shows the activation levels (ET spike counts) of disconnected glomeruli. Dashed trace shows the activation levels with the SA inter-glomerular connections in place. Note that the σ used is far from being that of an all to all network.



Result of varying the σ of the SA→ET connection distribution on the variance of the glomerular activation levels on the 10x10 glomerular model. The network reaches almost uniform excitation levels at relatively small σ values.



PG1 induced synchronization of the Mitral cell spiking. The inhibition resets the phase of the sub-threshold oscillations of the Mitral cell, bringing it into synch with the respiration. Multiple runs with randomized initial phase of the STOs are shown. In **A** the PG1 cell is present, and in **B** it is absent.



Odor affinity dependent delay in the generation of the first Mitral spike during an odor presentation.

Acknowledgements

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