A biologically-plausible computational architecture for sensor-based machine olfaction

R. Gutierrez-Osuna[1] and P. Sun[2]
[1]Department of Computer Science, Texas A&M University, College Station, TX 77843
[2]Department of Computer Science and Engineering, Wright State University, Dayton, OH 45435

This article presents a computational architecture for sensor-based machine olfaction based on biologically plausible models of the early stages in the olfactory pathway. We derive a concentration-response model that maps conventional sensor-array patterns into activation patterns for a population of olfactory receptor neurons (ORNs). A chemotopic convergence model is employed to generate spatial activation patterns at the glomerular layer that are consistent with neurobiology. These glomerular images serve as inputs to an implementation of Freeman's KIII neurodynamics model.

1 Experimental

An array of four temperature-modulated metal-oxide chemoresistors (TGS 2610, 2611, 2620 and 2600) was employed to collect a database of sensor-array patterns on serial dilutions of three organic solvents. The sensors were placed in a 30-ml vial containing 6-ml of the analytes, and excited with two 150-sec periods of a 1-7V sinusoidal heater voltage. The second cycle was sampled down to 5 measurements to generate a 20-dimensional feature vector. For visualization purposes, the feature vector was projected down to a two-dimensional space by means of Principal Components Analysis (PCA). Figure 1(left) illustrates the resulting patterns for three organic solvents (acetone (A), isopropyl alcohol (I) and ammonia (M)) at four water dilution levels (1: strongest; 4: weakest). To make the pattern-classification problem non-trivial, these dilution levels were chosen close to the isothermal detection threshold of the sensor array.

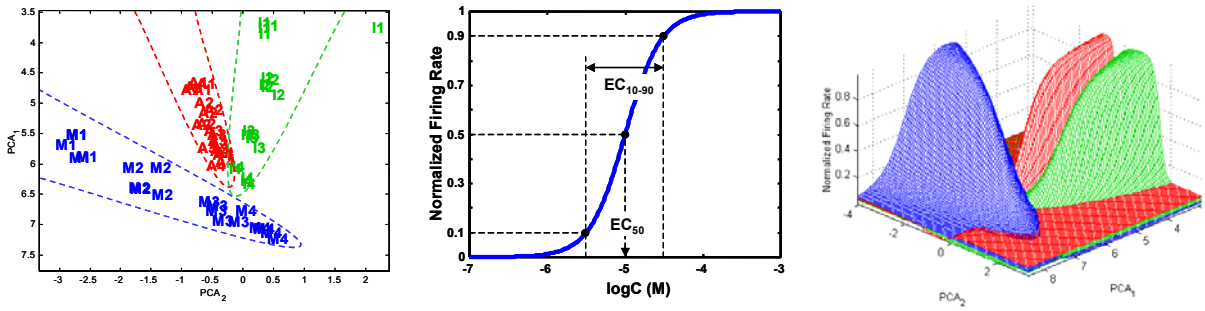

Figure 1. Principal components scatter plot of serial dilutions from three organic solvents (left), typical ORN dose-response curve (middle) and the final PCA-ORN mapping (right)

2 Olfactory Receptor Neuron model

The dose-response curve of individual ORNs was modeled with a sigmoidal activation function $R_i^L = 1/(1+(K_i^L[L])^{-m})$, where R_i^L is the average instantaneous firing rate of ORN_i when exposed to analyte L at concentration $[L]$, K_i^L is the binding affinity between ORN_i and L, and m is the molecular Hill equivalent [1]. Therefore, the affinity can be interpreted as the inverse of the effective concentration EC_{50} at which the ORN shows half-saturation, whereas m determines its intensity-tuning range or $EC_{10\text{-}90}$, as shown in Figure 1(middle). Thus, to simulate the activation function of ORNs, a regression mapping needs to be formed from feature space onto firing rates. This is accomplished by assigning a monotonically decreasing firing rate to the different analyte dilutions and building a non-linear regression model. Figure 1(right) shows a final ORN model obtained using a multilayer perceptron (MLP) with logistic activation functions at the hidden nodes (two in this case) and output nodes. It must be emphasized that biological plausibility stems from the monotonic dose-response curves, not from the MLP regression model.

3 Chemotopic convergence

A population of ORNs with different selectivities is simulated by assigning a particular odor dilution to the EC_{50} firing rate, and working toward higher and lower dilutions in a monotonic fashion. The distribution of EC_{50} responses for a particular analyte across a population of ORNs is modeled with the Receptor Affinity Distribution [2], a universal model for ligand-receptor interactions with strong implications for olfactory coding. As a result, each ORN can be expressed by a vector of affinities, and its selectivity is defined by the direction of this vector. In [3] we have proposed a self-organizing model of chemotopic convergence capable of generating an olfactory code of glomerular maps in close agreement with neurobiology. Figure 2(a) shows the spatial activation patterns across a 20x20 glomerular lattice for four concentrations of three analytes. Each analyte elicits a unique glomerular image, with higher activity in those GLs that receive projections from ORNs with high affinity to that analyte. As the concentration of the analyte increases, additional ORNs with lower affinity are recruited, resulting in an increased activation level and a larger spread of the analyte-specific loci. Thus,

these glomerular maps are capable of decoupling odor quality from odor intensity, quality being encoded by the spatial pattern across glomeruli and intensity being captured by the amplitude and spread of this pattern.

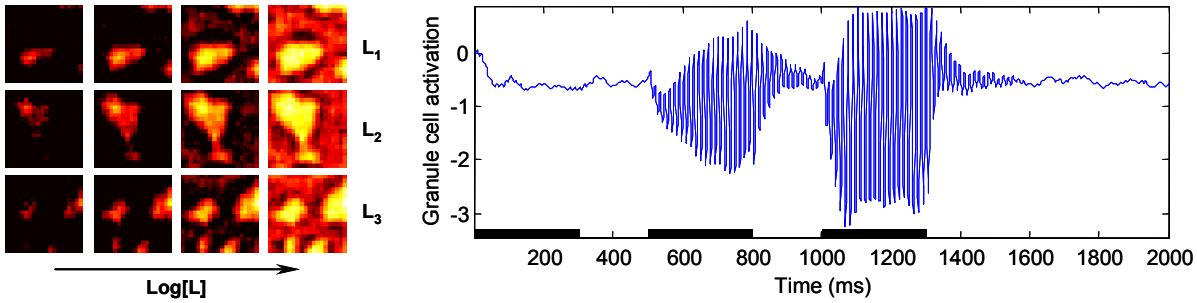

Figure 2. Glomerular activation patterns at different concentrations of three analytes (left) and granule oscillations for concentration pulses of 0.15 (0-300ms), 0.55 (500-800ms) and 0.95(1000-1300ms) (right)

4 Bulb neurodynamics

In order to study the role of dynamics on olfactory processing, we have developed a simulation based on Freeman's KIII model of the olfactory system [4]. The KIII is a realistic model that faithfully captures the population neurodynamics of the olfactory bulb as observed in electro-encephalogram (EEG) recordings. The KIII model has an ample repertoire of attractors in phase space, including fixed points, limit cycles and strange attractors. Activation at the glomerular layer, such as the one shown in Figure 2(left), serves as an input to a layer of mitral-granule coupled oscillators, moving the KIII from a basal state into an odor-specific attractor wing. Odor concentration, meanwhile, is preserved in the amplitude of these oscillations, as shown in Figure 2(right) for a granule cell stimulated with three odor pulses at increasing concentrations. After removal of the odor, the granule cell then returns to its basal state to be ready for the next stimulus.

From a pattern-recognition viewpoint, the KIII model acts as an associative memory capable of recovering an odor attractor from an incomplete input pattern. This is achieved through Hebbian learning in a layer of excitatory connections between mitral cells. Figure 3 illustrates the behavior of the KIII after learning a dataset of three binary odor patterns: A [01001001], B [10010010] and C [10100100]. In Figure 3(a) and (b), the KIII is excited with pattern B and an incomplete version B* [10000010]. As a result of the mitral connections, the system is able to recall the complete pattern by inducing a higher oscillation on the missing 4th channel. Learning in the conventional KIII model is restricted to the Hebbian connections between mitral cells. To improve the pattern-recognition capabilities of the model, we are currently investigating learning rules for the remaining layers, which otherwise have fixed connections in Freeman's formulation. In particular, we have developed a learning rule for granule-cell connections that allows the KIII to perform contrast enhancement between odor patterns [5]. As shown in Figure 3(c-d), the enhanced KIII model responds with a higher contrast between channels when exposed to the same patterns in Figure 3(a-b).

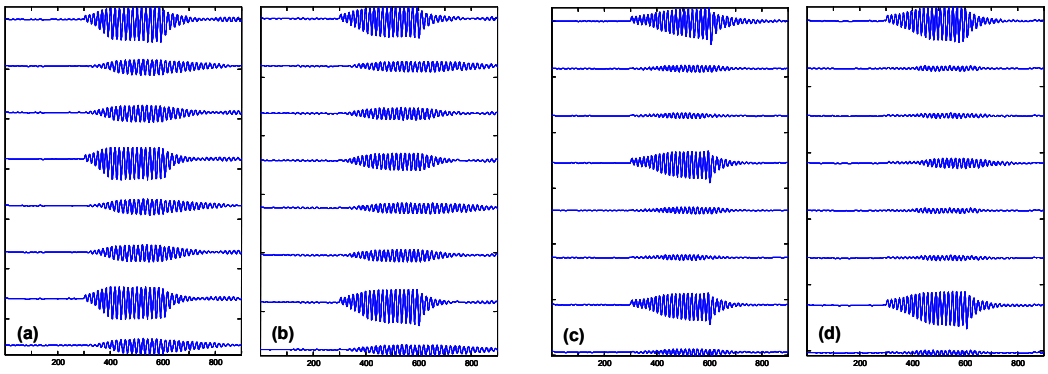

Figure 3. Response of the conventional KIII model to pattern B (a) and incomplete pattern B* (b). Contrast enhancement with modified granule connections for B (c) and B* (d).

5 Acknowledgments

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6 References

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