

The LISSOM Cortical Model

Dr. James A. Bednar

jbednar@inf.ed.ac.uk

<http://homepages.inf.ed.ac.uk/jbednar>

Problems with SOMs

A Kohonen SOM is very limited as a model of cortical function:

- Picking one winner is valid only for a very small patch with very strong lateral inhibition.
- Full connectivity is possible only for very small cortical networks.
- Lateral interactions are forced to be isotropic, contrary to biological evidence.
- Euclidean distance metric is not clearly relatable to neural firing or synaptic plasticity.

Problems with SOM retinotopy

The particular model of SOM retinotopy we've been looking at also has other problems:

- There is no known state when the connections from the eye are evenly distributed across a target region; even the initial connections are retinotopic.
- The overall retinotopy is established by axons following gradients of signaling molecules such as Ephrins, though activity may have some role in this process (reviewed in Flanagan 2006; Huberman et al. 2008).

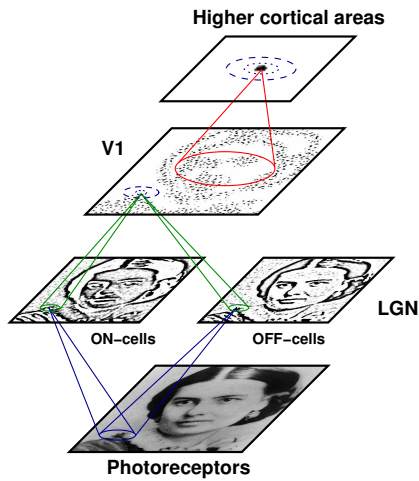
In any case, activity appears to be required for map refinement, and it's interesting that in principle an unfolding process like in the SOM simulation could work.

LISSOM

The LISSOM model (Sirosh & Miikkulainen 1994) was designed to remove some of the artificial limitations and biologically unrealistic features of a SOM:

- Recurrent lateral interactions, instead of global winner
- Specific lateral connections, instead of isotropic neighborhood
- Spatially localized RFs, instead of full connectivity
- Activation by sigmoided dot product, rather than Euclidean distance
- Learning by Hebbian rule

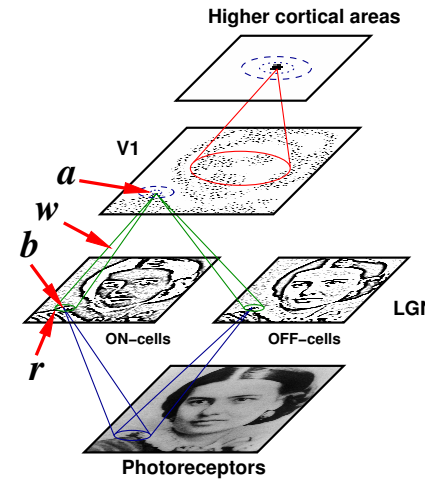
HLISSOM Architecture



Bednar & Miikkulainen, 1995–2004

Preference maps, receptive fields, patchy lateral connections, multiple areas, natural images

HLISSOM Architecture

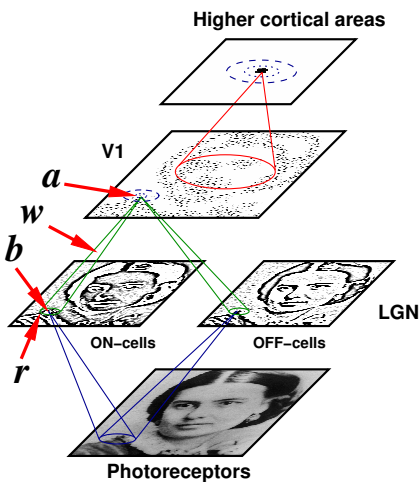


Activity: thresholded weighted sum of all receptive fields

$$\eta_a = \sigma \left(\sum_r \gamma_r \sum_b X_{rb} w_{a,rb} \right)$$

- Response high when input matches weights

HLISSOM Architecture



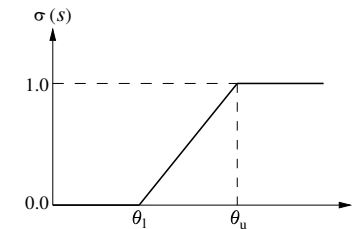
Learning:

normalized Hebbian

$$w_{a,rb}(t+1) = \frac{w_{a,rb}(t) + \alpha_r \eta_a X_{rb}}{\sum_c [w_{a,rc}(t) + \alpha_r \eta_a X_{rc}]}$$

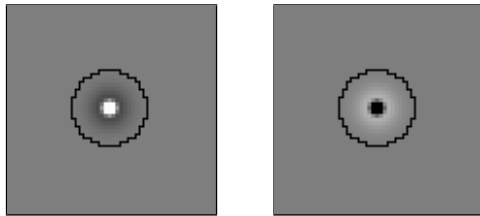
- Coactivation → strong connection
- **Normalization:** distributes strength

Neuron activation function $\sigma(s)$



- Piecewise-linear approximation to a sigmoid
- Easy to compute
- Speeds up computation, since most neurons are truly off
- Strongly sensitive to threshold θ_l

DoG LGN RFs



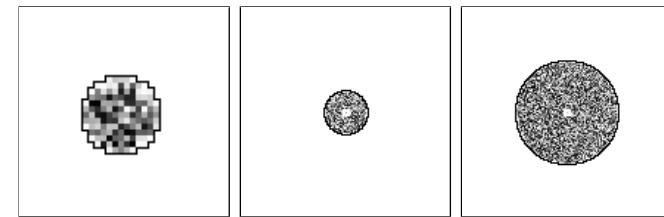
CMVC figure 4.2

ON neuron

OFF neuron

- Fixed Difference of Gaussians
- Center/surround size ratio based on experimental data
- Precisely balanced c/s strength ratio (not quite realistic)

Initial V1 weights



CMVC figure 4.3

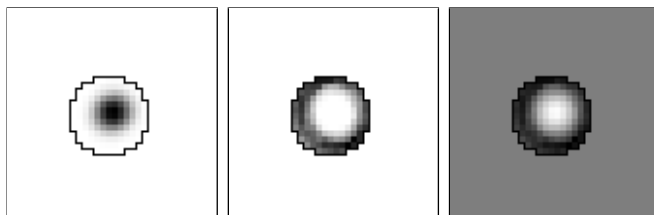
Afferent (ON and OFF)

Lateral excitatory

Lateral inhibitory

- Initial rough topographic organization
- Explicit lateral connections

Self-organized V1 afferent weights



CMVC figure 4.6

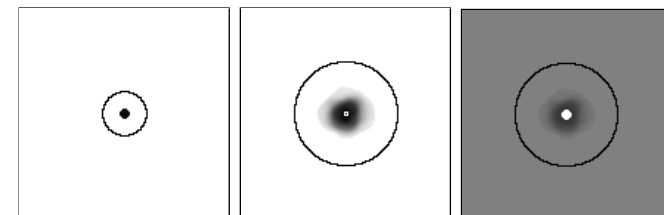
ON

OFF

Combined
(ON-OFF)

Given isotropic Gaussians, learns isotropic Gaussians

Self-organized V1 lateral weights



CMVC figure 4.9

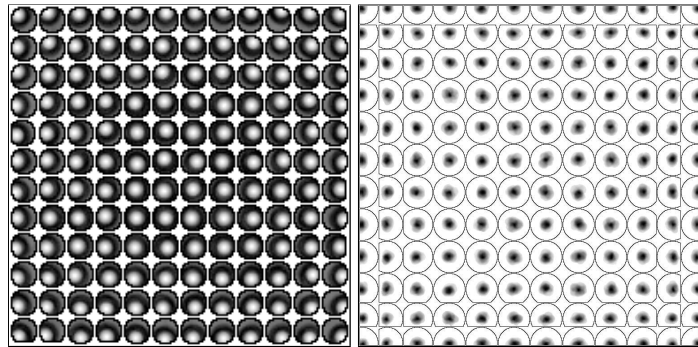
Lateral excitatory

Lateral inhibitory

Combined
(exc.-inh.)

- Learns isotropic (Mexican-hat) lateral interactions
- Reflects the flatness of learned map (no folding)

Self-organized afferent and lateral weights across V1

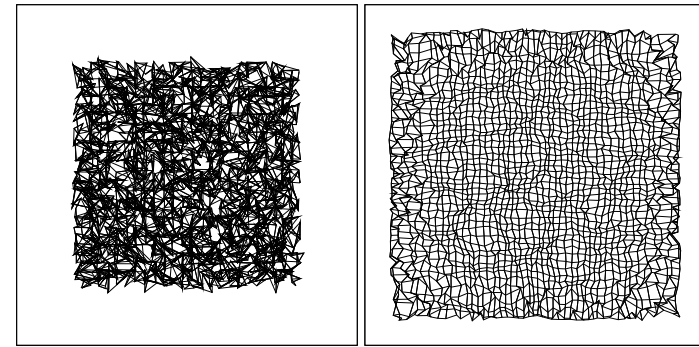


Afferent (ON—OFF)

Lateral inhibitory

CMVC figure 4.7

Self-organization of the retinotopic map

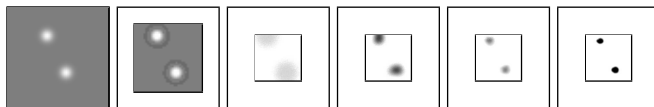


Initial disordered map

Final retinotopic map

CMVC figure 4.8

Retinotopy input and response



Retinal activation LGN response Iteration 0: Initial V1 response Iteration 0: Settled V1 response 10,000: Initial V1 response 10,000: Settled V1 response

CMVC figure 4.4

- Settling process: Sharpens activity around strongly activated patches
- Multiple winners occur for multiple features on input

Summary

LISSOM: same basic process as a SOM, but:

- More plausible
- More powerful:
 - Multiple winners
 - Specific lateral connections
- More sensitive to parameters
- More computation and memory intensive

References

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