

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

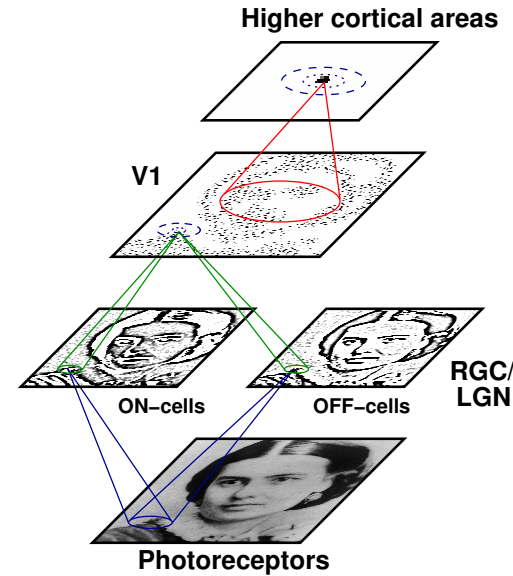
GCAL

In turn, GCAL (Bednar 2012; Law et al. 2011) was designed to remove some of the artificial limitations and biologically unrealistic features of LISSOM:

- Automatic homeostatic plasticity, instead of hand-adjusted thresholds
- No lateral connection radius shrinking or arbitrary changes to learning rates or settling steps over time
- Gain control for realistic behavior with contrast (similar to the afferent normalization of CMVC section 8.2.3)

GCAL is otherwise like LISSOM. The CMVC book and older work all focus on LISSOM, but current work uses GCAL; for this course the distinction is not important.

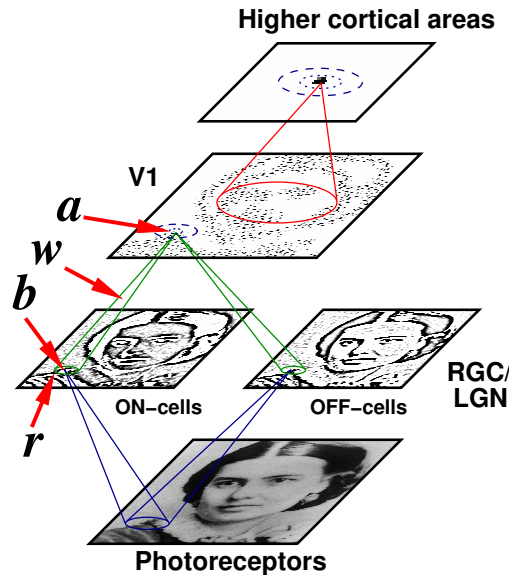
HLISSOM Architecture



Bednar & Miikkulainen, 1995–2004

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

HLISSOM Architecture

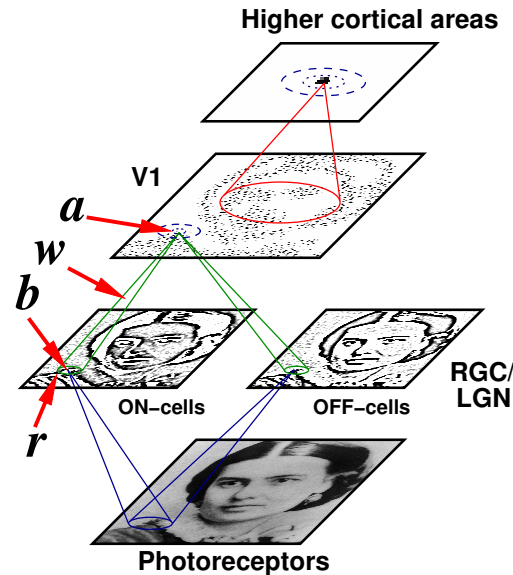


Activity: thresholded weighted sum of all receptive fields

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

- Response high when input matches weights

HLISSOM Architecture

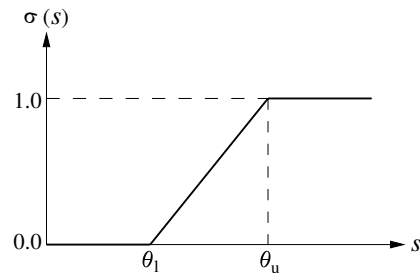


Learning:
normalized Hebbian

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

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

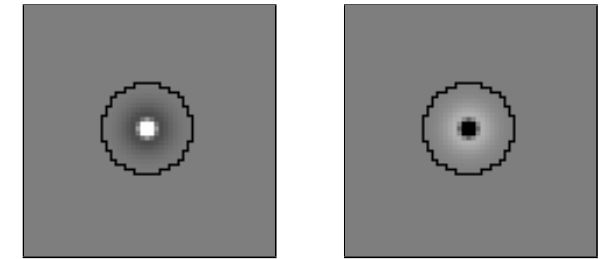
Neuron activation function $\sigma(s)$



CMVC figure 4.5

- 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 RGC/LGN RFs



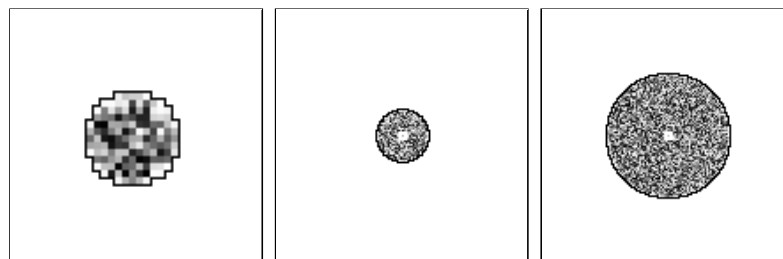
ON neuron

OFF neuron

CMVC figure 4.2

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

Initial V1 weights



Afferent (ON and OFF)

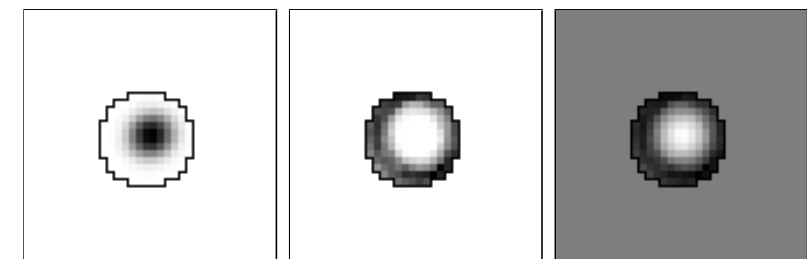
Lateral excitatory

Lateral inhibitory

CMVC figure 4.3

- Initial rough topographic organization
- Explicit lateral connections

Self-organized V1 afferent weights



ON

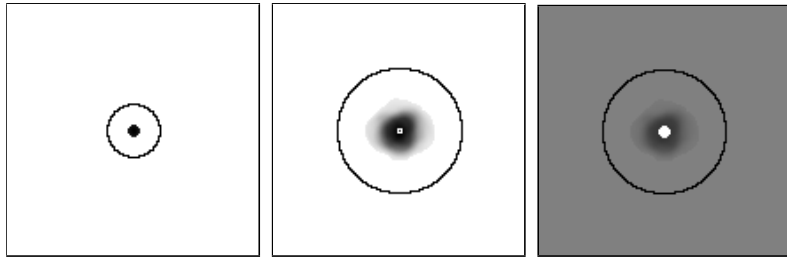
OFF

Combined (ON-OFF)

CMVC figure 4.6

Given isotropic Gaussians, learns isotropic Gaussians

Self-organized V1 lateral weights

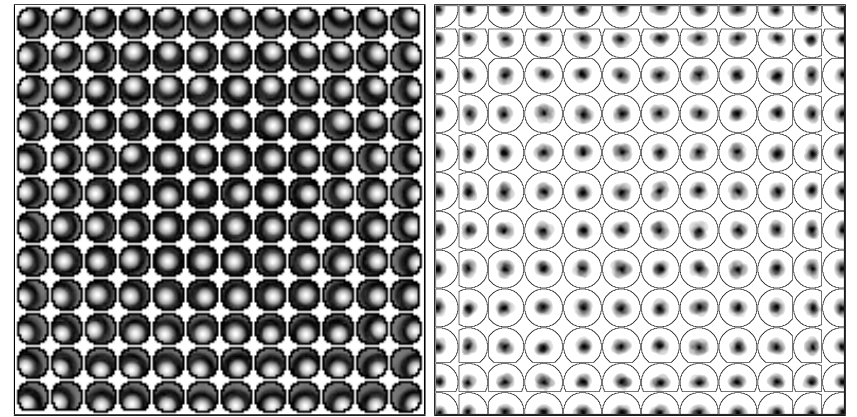


Lateral excitatory Lateral inhibitory Combined (exc. - inh.)

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

CMVC figure 4.9

Self-organized afferent and lateral weights across V1

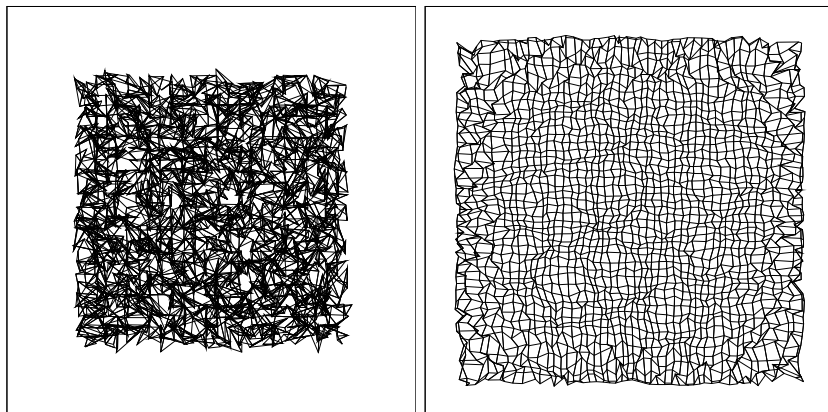


Afferent (ON-OFF)

Lateral inhibitory

CMVC figure 4.7

Self-organization of the retinotopic map

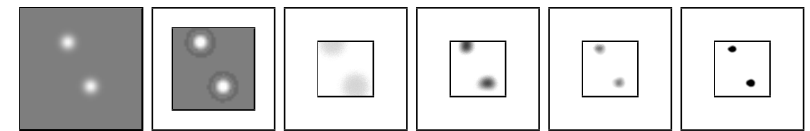


Initial disordered map

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

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

CMVC figure 4.4

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

- Bednar, J. A. (2012). Building a mechanistic model of the development and function of the primary visual cortex. *Journal of Physiology (Paris)*, 106, 194–211.
- Flanagan, J. G. (2006). Neural map specification by gradients. *Current Opinion in Neurobiology*, 16, 1–8.
- Huberman, A. D., Feller, M. B., & Chapman, B. (2008). Mechanisms underlying development of visual maps and receptive fields. *Annual Review of Neuroscience*, 31, 479–509.
- Law, J. S., Antolik, J., & Bednar, J. A. (2011). Mechanisms for stable and robust development of orientation maps and receptive fields. Tech. rep., School of Informatics, The University of Edinburgh. EDI-INF-RR-1404.

Sirosh, J., & Miikkulainen, R. (1994). Cooperative self-organization of afferent and lateral connections in cortical maps. *Biological Cybernetics*, 71, 66–78.