The LISSOM and GCAL Cortical Models

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Problems with SOMs

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

- Picking one winner requires a global supervisor, valid only for a tiny patch with very strong lateral inhibition.
- Full connectivity is possible only for tiny cortical networks.
- Lateral interactions are forced to be isotropic, contrary to biological evidence.
- There is no evidence for lateral radius shrinking; in fact the opposite appears to be true: initially diffuse, becoming patchier and longer-range
- The Euclidean distance metric is not clearly relatable to neural firing or synaptic plasticity.

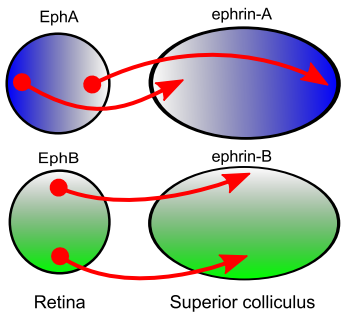
Problems with SOM retinotopy

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 roughly retinotopic.
- Activity has relatively little role in this initial establishment of retinotopy, as reviewed on the following slide

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.

Mapping via chemoaffinity



- Gradients of chemicals called Ephs and Ephrins provide a 2D coordinate system for the retina and its target area
- Axons find their way to their approximate locations by following the gradients within the target area
- Activity's role appears to be mainly local, affecting the axonal arbor size and shape
- Reviewed in Flanagan 2006; Huberman et al. 2008

LISSOM

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

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

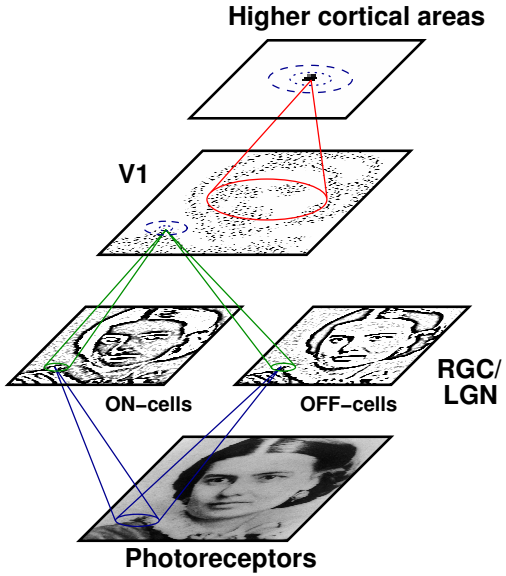
GCAL

In turn, GCAL (Bednar 2012; Stevens et al. 2013) 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.

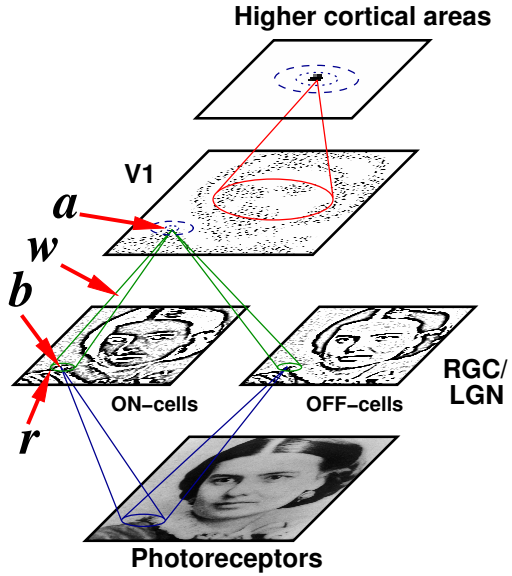
HLISSOM Architecture



Bednar & Miikkulainen, 1995–2004

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

HLISSOM Architecture

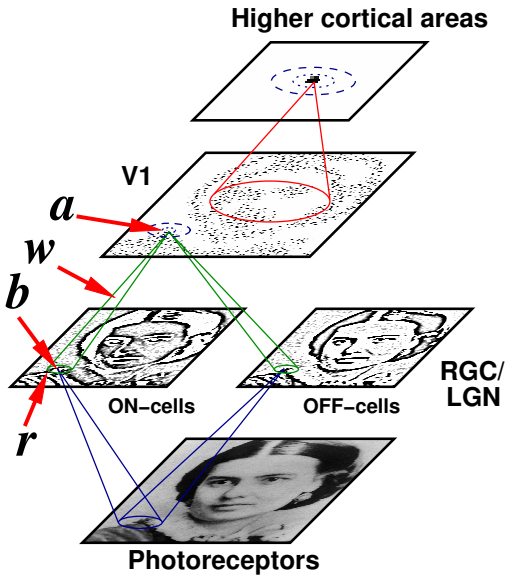


Activity: thresholded weighted sum of all receptive fields

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

- Response high when input matches weights

HLISSOM Architecture



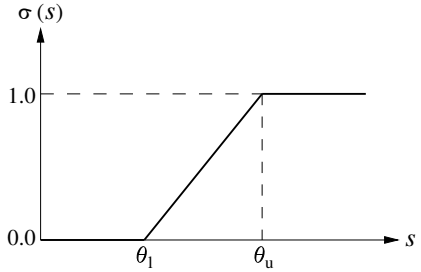
Learning:

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

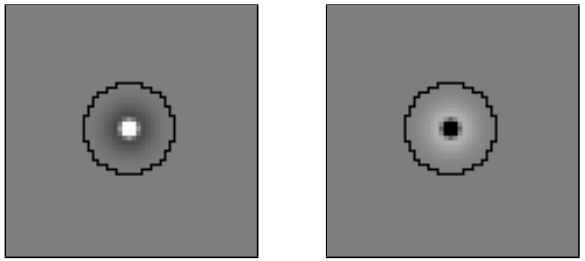
Neuron activation function $\sigma(s)$



CMVC figure 4.5

- LISSOM: Easy-to-compute piecewise-linear sigmoid
  Strongly sensitive to thresholds θ_l and θ_u
- GCAL: No θ_u (approximates s^2)
  θ_l set automatically to achieve target average firing rate

DoG RGC/LGN RFs

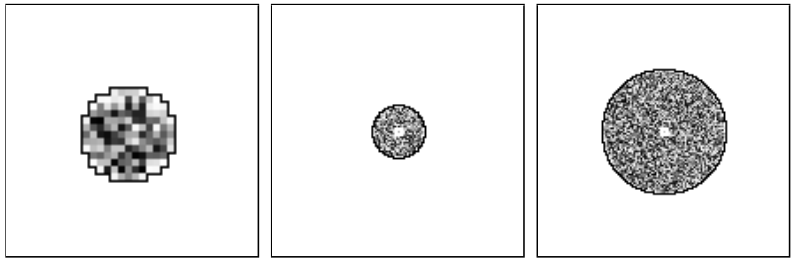


ON neuron OFF neuron

CMVC figure 4.2

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

Initial V1 weights

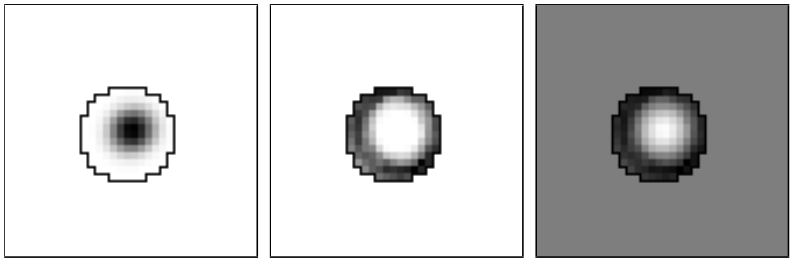


Afferent (ON and OFF) Lateral excitatory Lateral inhibitory

CMVC figure 4.3

- Initial rough topographic organization
- Explicit lateral connections
- LISSOM: Initially larger lateral excitatory radius

Self-organized V1 afferent weights

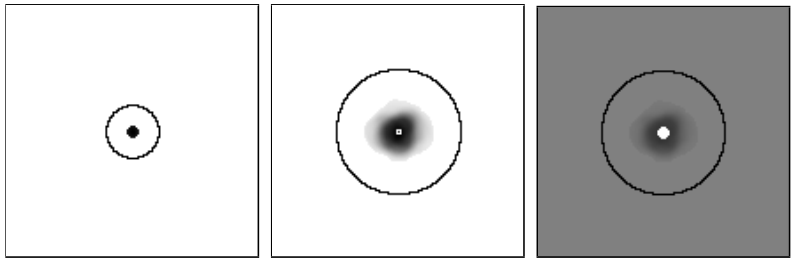


CMVC figure 4.6

ON OFF Combined (ON—OFF)

Given isotropic Gaussians, learns isotropic Gaussians

Self-organized V1 lateral weights

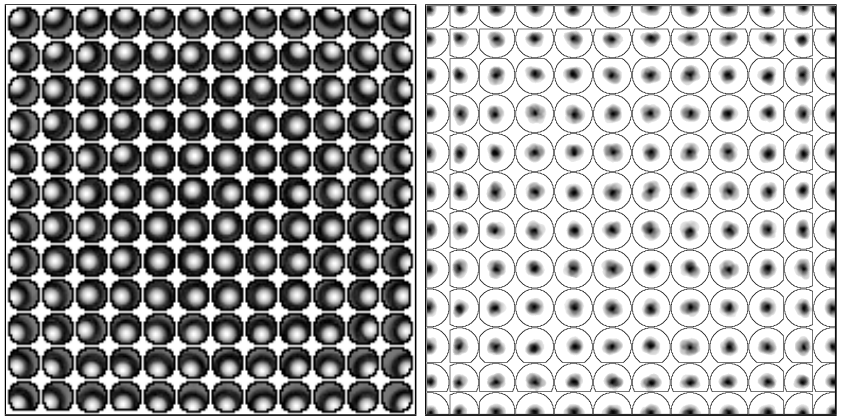


CMVC figure 4.9

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

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

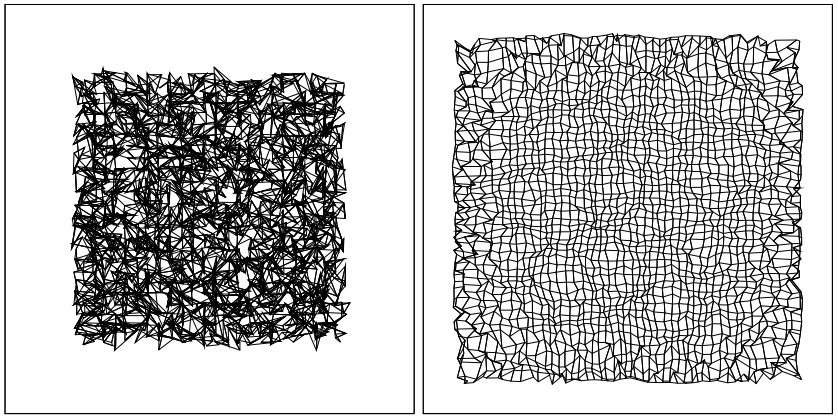
Self-organized afferent and lateral weights across V1



CMVC figure 4.7

Afferent (ON—OFF) Lateral inhibitory

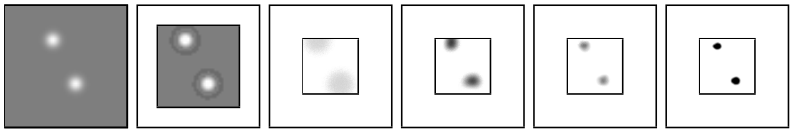
Self-organization of the retinotopic map



CMVC figure 4.8

Initial disordered map Final retinotopic map

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 or large input features

Summary

LISSOM/GCAL: same basic process as a SOM, but:

- More plausible
- More powerful:
  - Multiple winners
  - Specific lateral connections
- More computation and memory intensive
- LISSOM: Unfortunately, very sensitive to parameters
- GCAL: Still robust, now due to local gain control and homeostasis, rather than global winner picking

References

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