The LISSOM Cortical Model

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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 gross retinotopy is not apparently dependent on neural activity, but on signaling molecules such as Ephrins (reviewed in **?**).

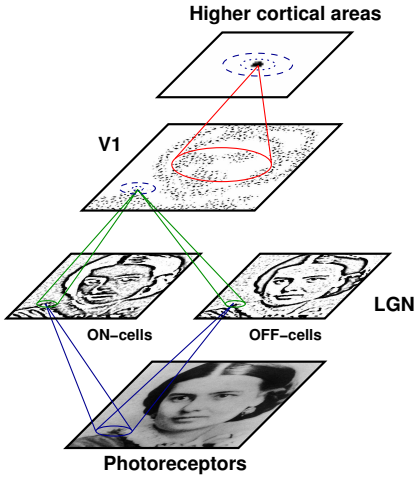
However, activity appears to be required for map refinement, and it's interesting that in principle an unfolding process like in the SOM simulation could work.

LISSOM

The LISSOM model (**?**) 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

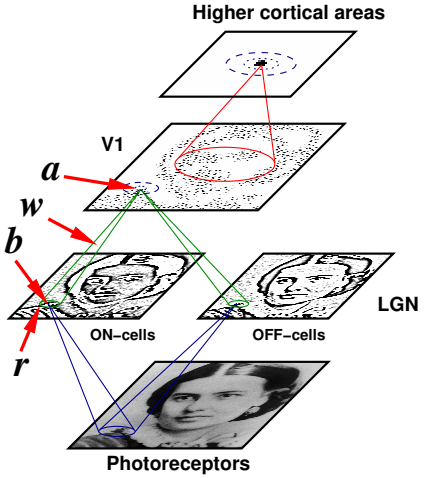
HLISSOM Architecture



Bednar & Miikkulainen, 1995–2004

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

HLISSOM Architecture

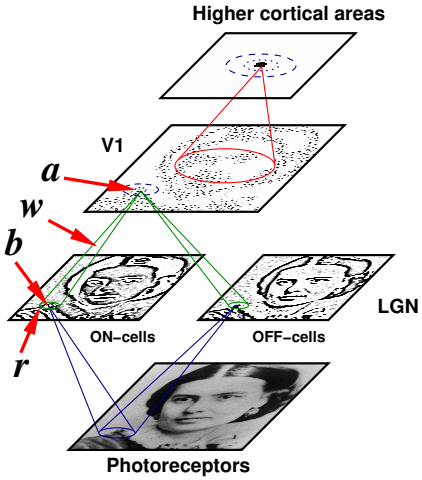


Activity: thresholded weighted sum of all receptive fields

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

- Response high when input matches weights

HLISSOM Architecture

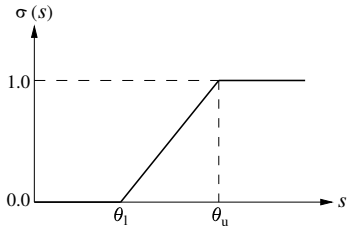


Learning: normalized Hebbian

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

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

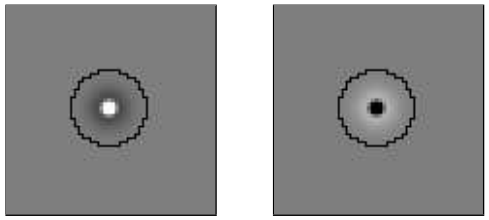
Neuron activation function $\sigma(s)$



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 LGN RFs

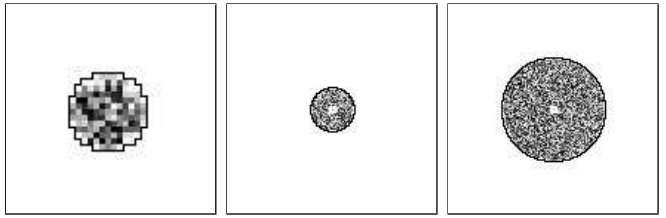


ON neuron OFF neuron

CMVC figure 4.2

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

Initial V1 weights

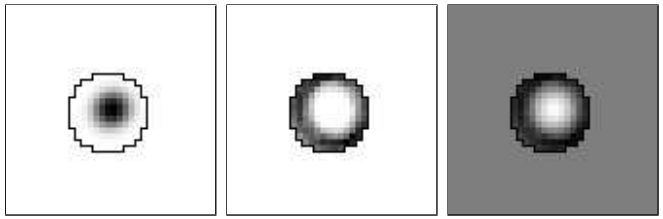


Afferent (ON and OFF) Lateral excitatory Lateral inhibitory

CMVC figure 4.3

- Initial rough topographic organization
- Explicit lateral connections

Self-organized V1 afferent weights

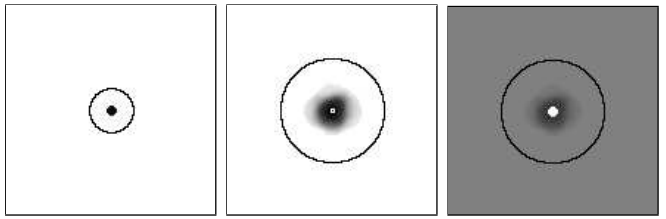


ON OFF Combined (ON—OFF)

CMVC figure 4.6

Given isotropic Gaussians, learns isotropic Gaussians

Self-organized V1 lateral weights

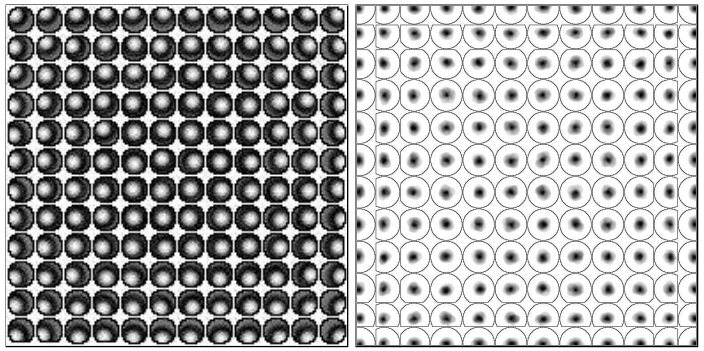


Lateral excitatory Lateral inhibitory Combined (exc.—inh.)

CMVC figure 4.9

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

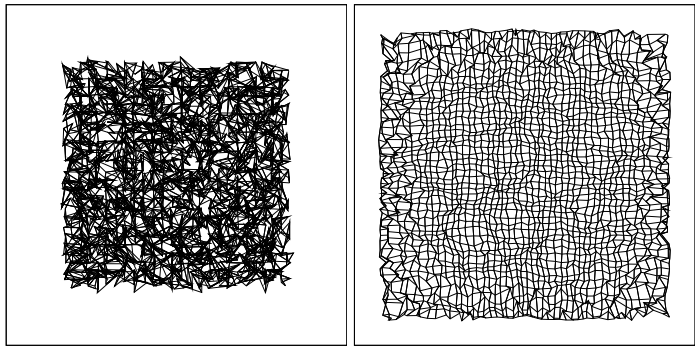
Self-organized afferent and lateral weights across V1



CMVC figure 4.7

Afferent (ON—OFF) Lateral inhibitory

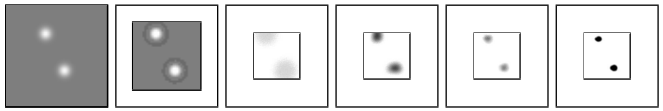
Self-organization of the retinotopic map



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 features on input

Summary

LISSOM: same basic process as a SOM, but:

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