

# 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 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).

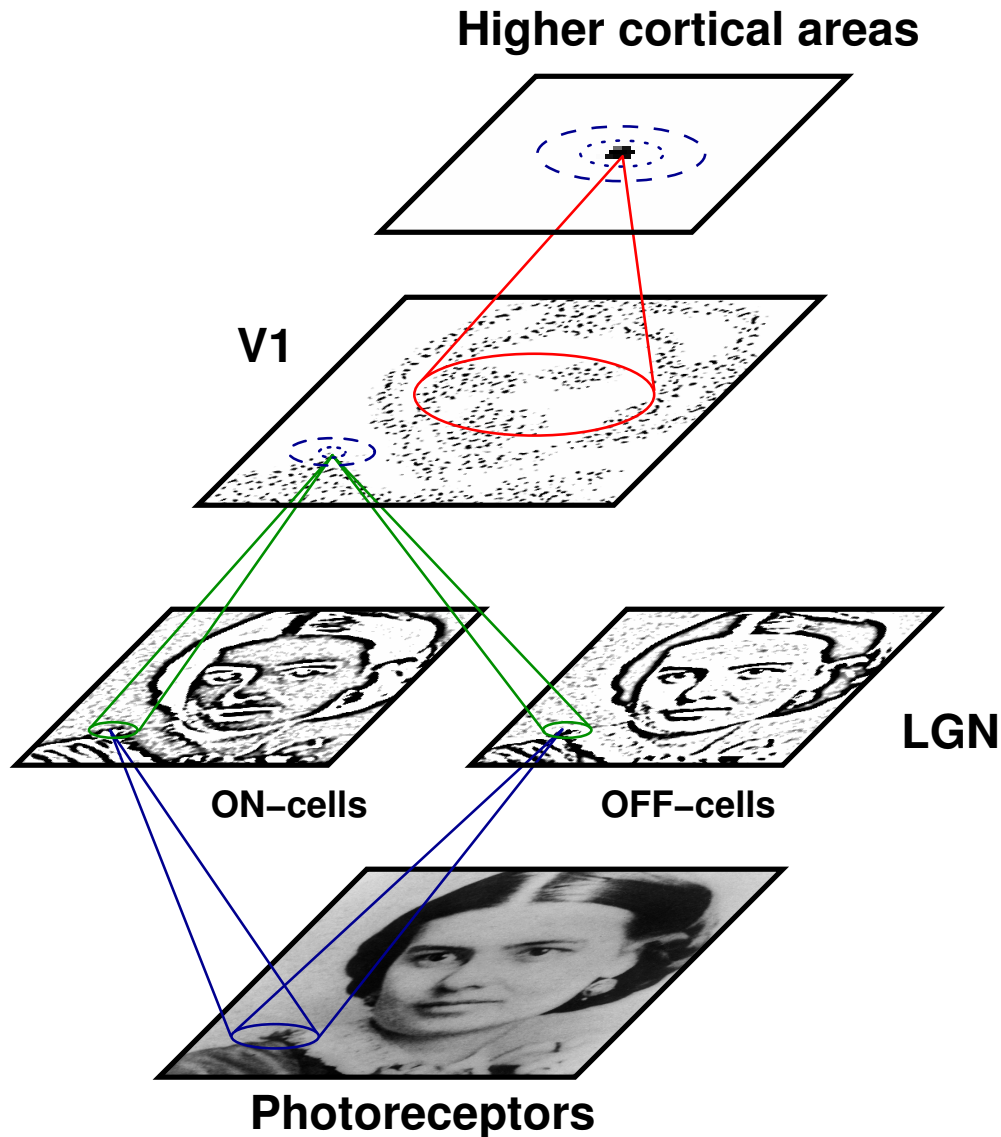
In any case, activity appears to be required for map refinement, and it's interesting that in principle an unfolding process like in the SOM simulation could work.

# 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

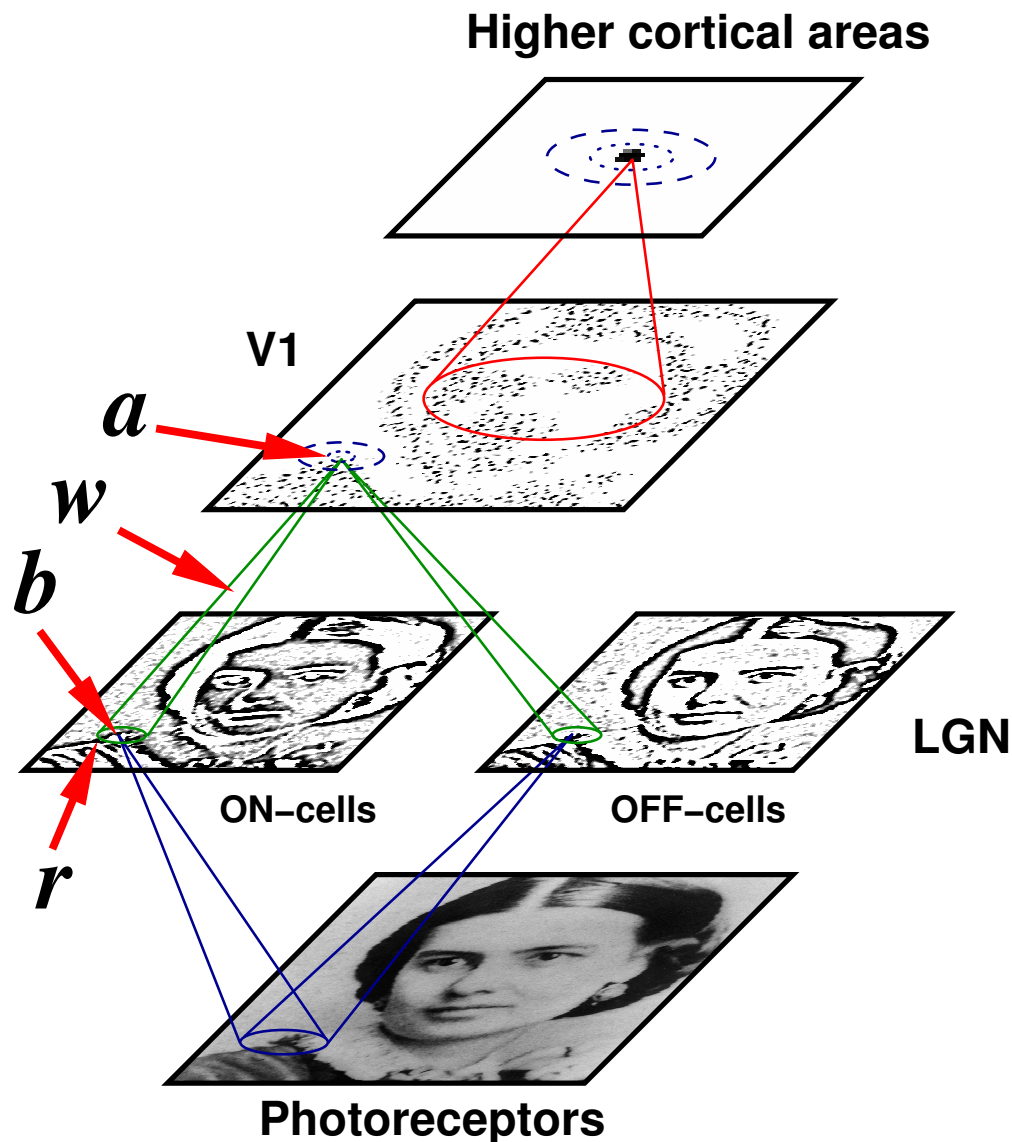
# HLISSOM Architecture



Bednar &  
Miikkulainen,  
1995–2004

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

# HLISSOM Architecture



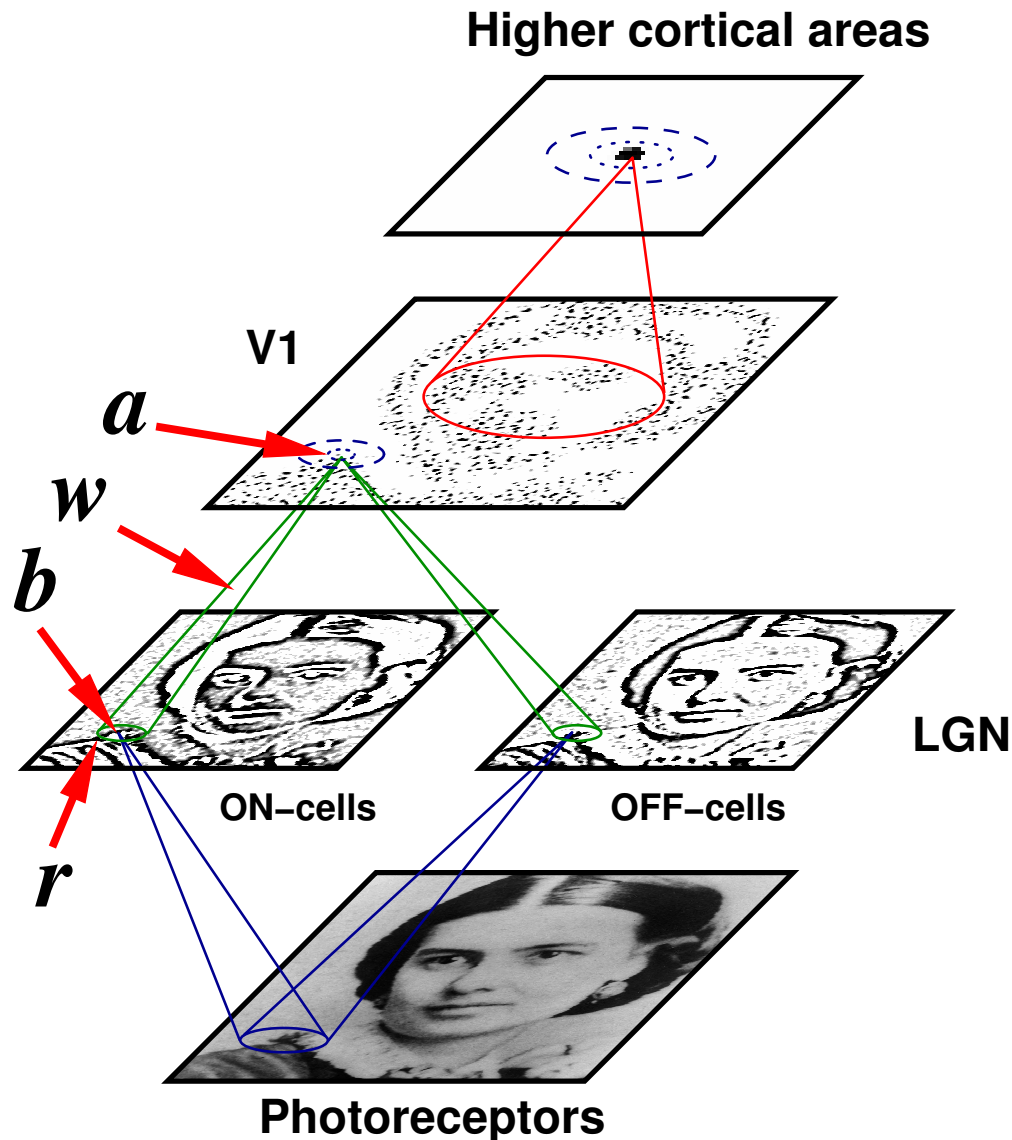
**Activity:** thresholded weighted sum of all receptive fields

$$\eta_a =$$

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

- Response high when input matches weights

# HLISSOM Architecture



## Learning:

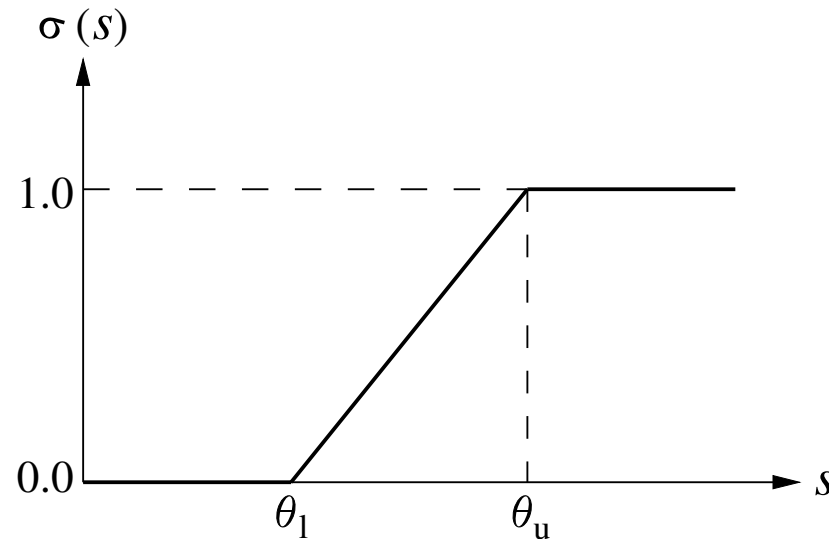
normalized Hebbian

$$w_{a,rb}(t + 1) =$$

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

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

# Neuron activation function $\sigma(s)$

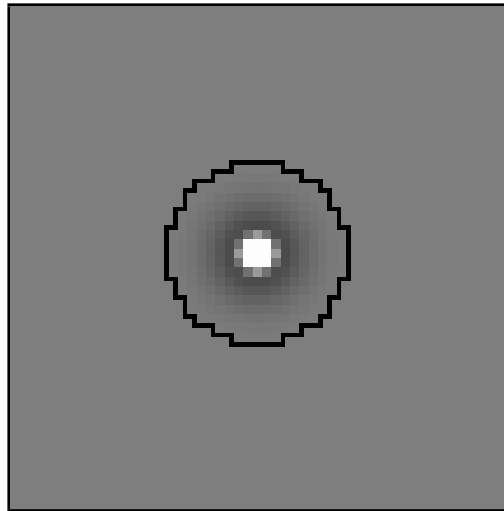


CMVC figure 4.5

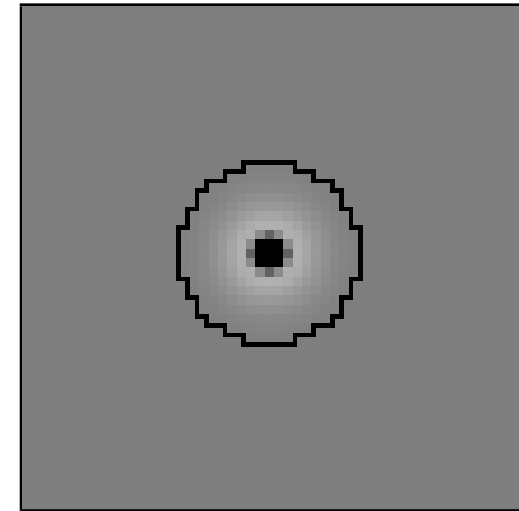
- Piecewise-linear approximation to a sigmoid
- Easy to compute
- Speeds up computation, since most neurons are truly off
- Strongly sensitive to threshold  $\theta_l$



# DoG LGN RFs



ON neuron

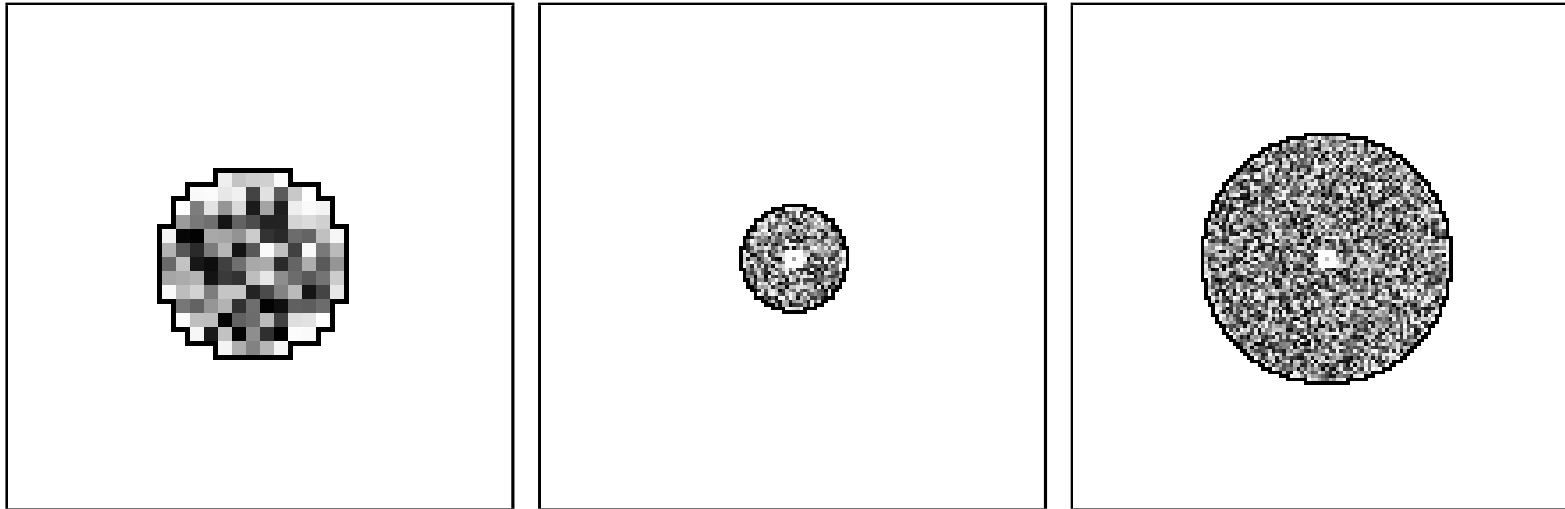


OFF neuron

CMVC figure 4.2

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

# Initial V1 weights



Afferent (ON and  
OFF)

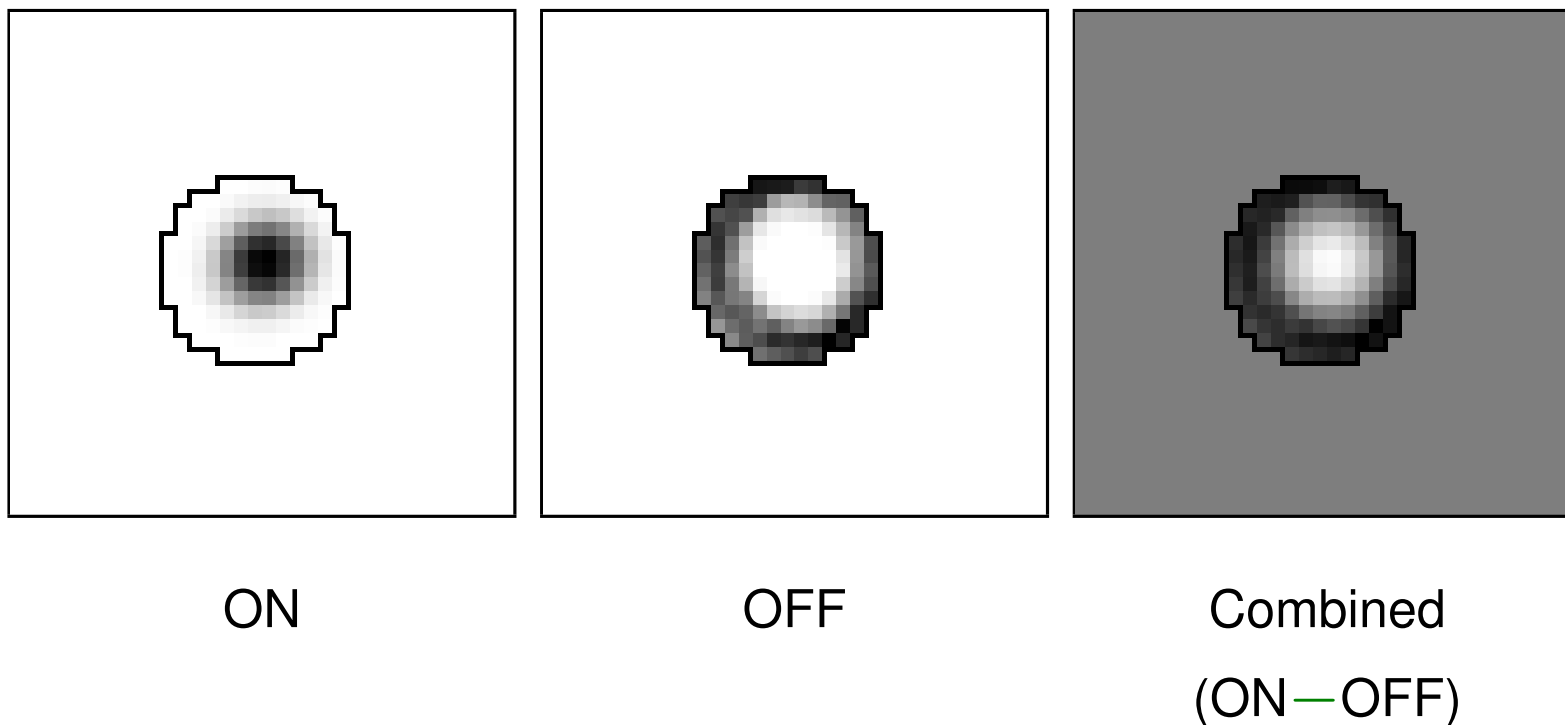
Lateral excitatory

Lateral inhibitory

- Initial rough topographic organization
- Explicit lateral connections

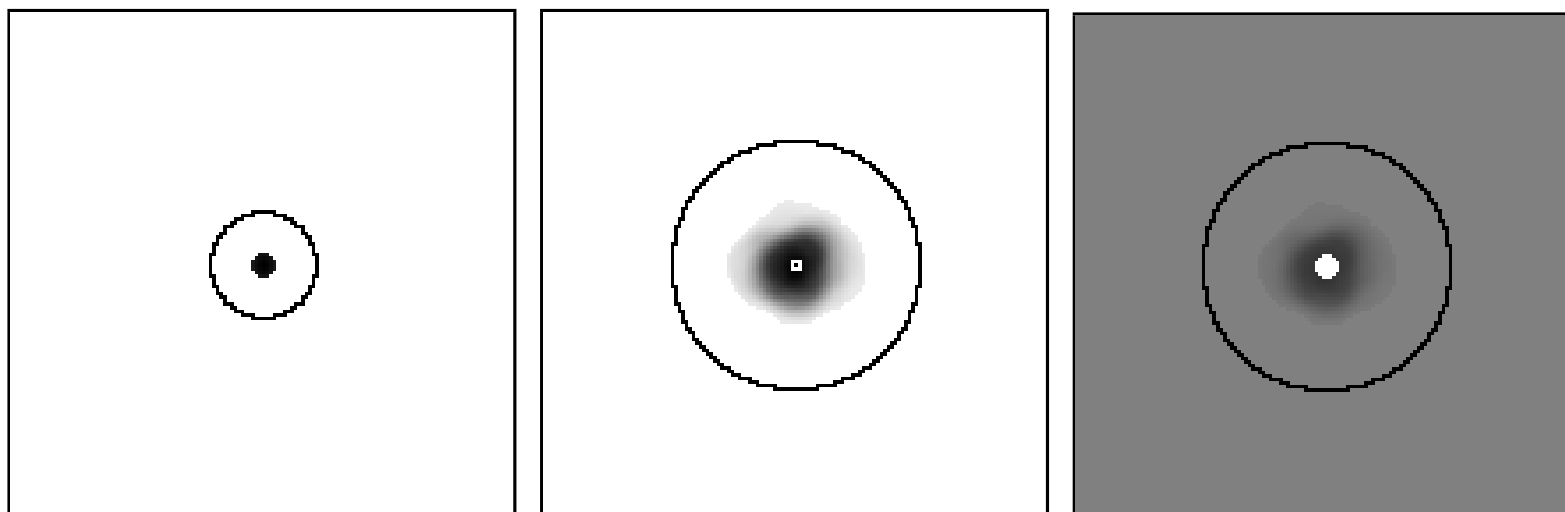
CMVC figure 4.3

# Self-organized V1 afferent weights



Given isotropic Gaussians, learns isotropic Gaussians

# Self-organized V1 lateral weights



Lateral excitatory

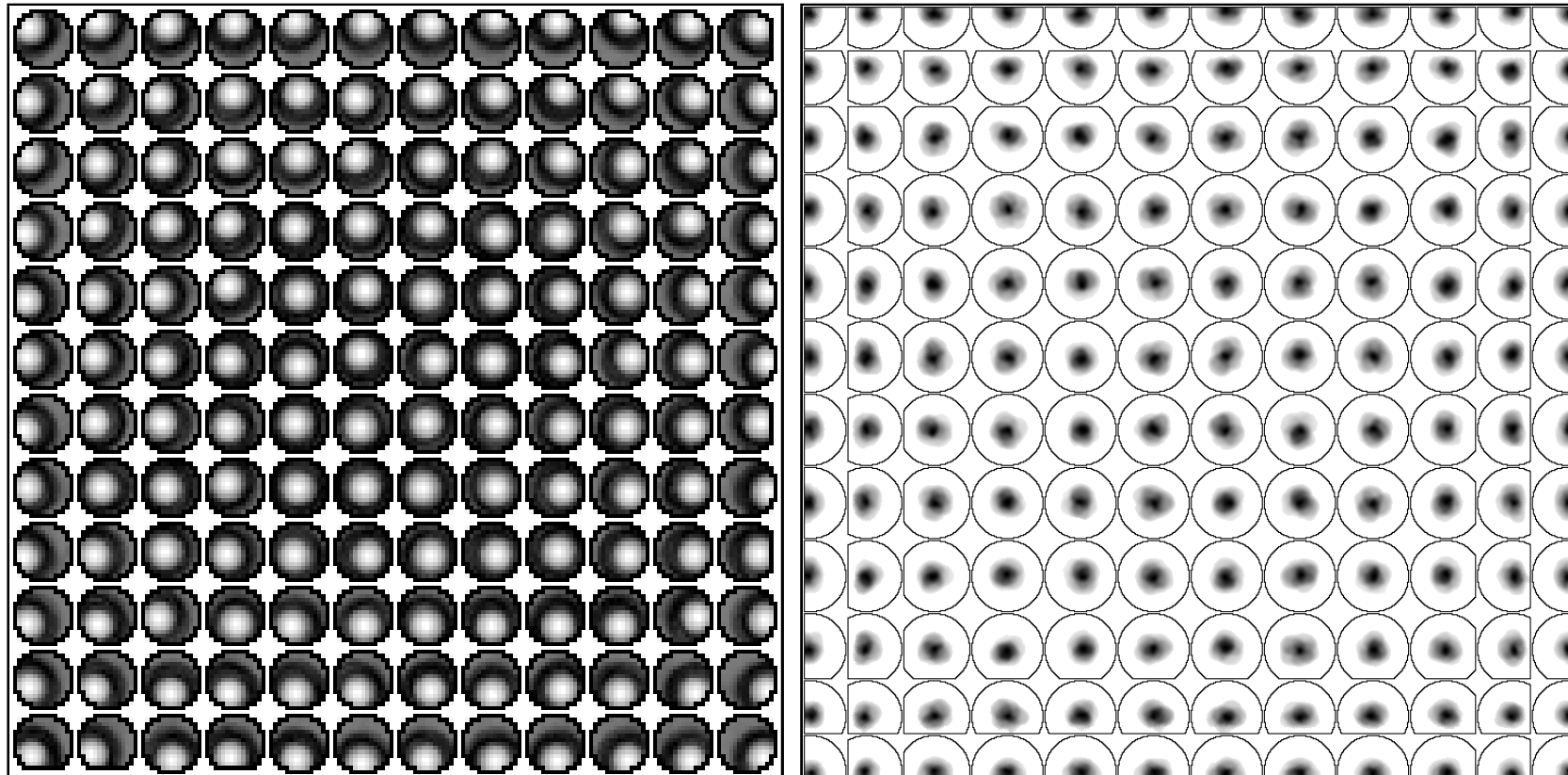
Lateral inhibitory

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

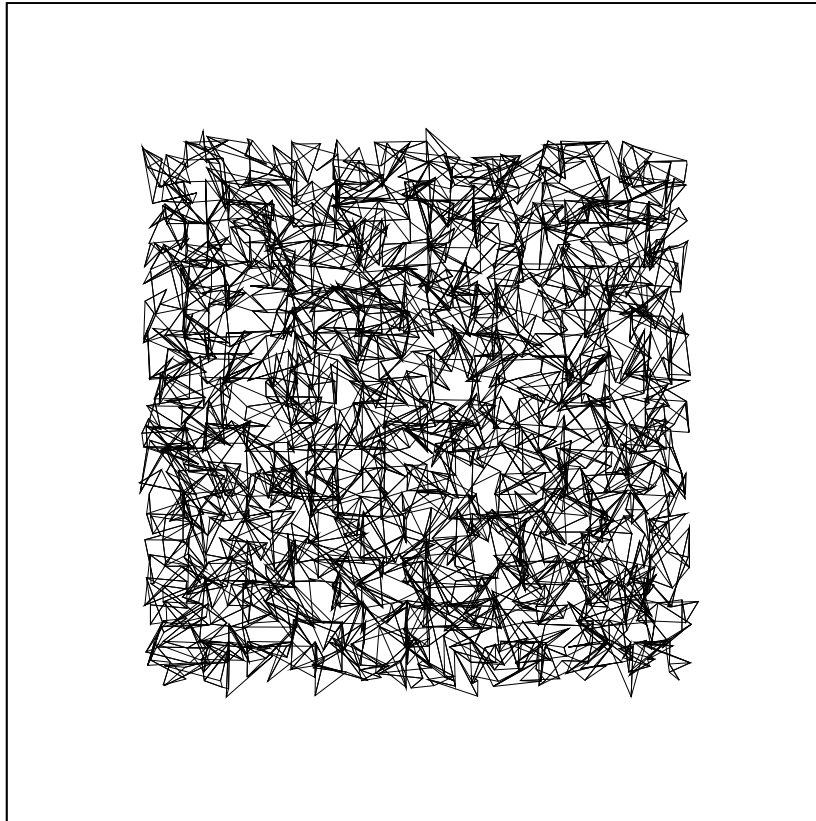


CMVC figure 4.7

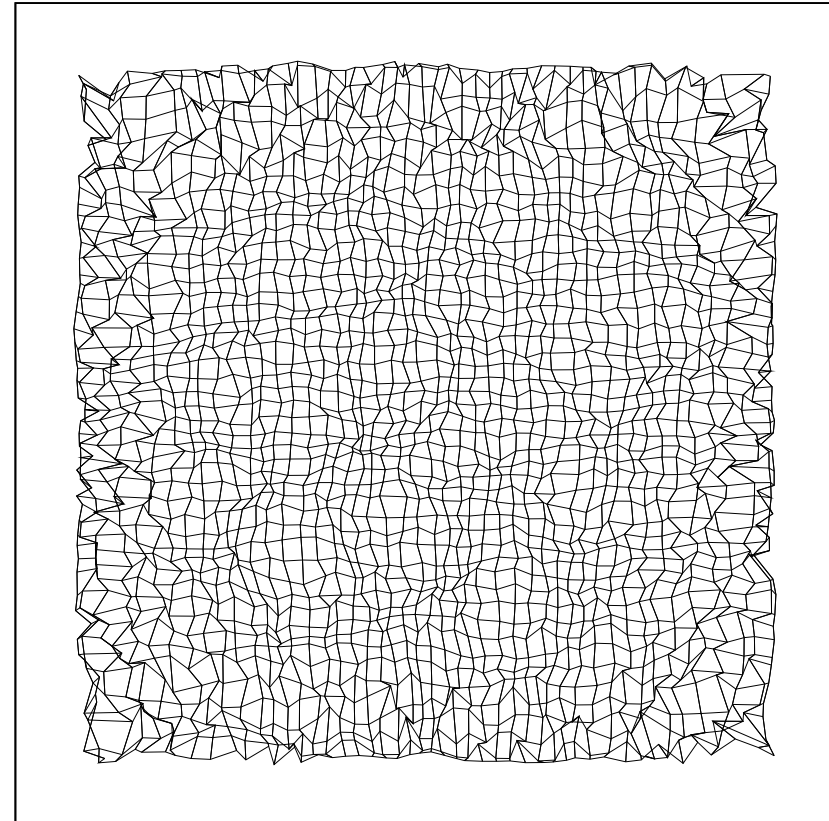
Afferent (ON—OFF)

Lateral inhibitory

# Self-organization of the retinotopic map



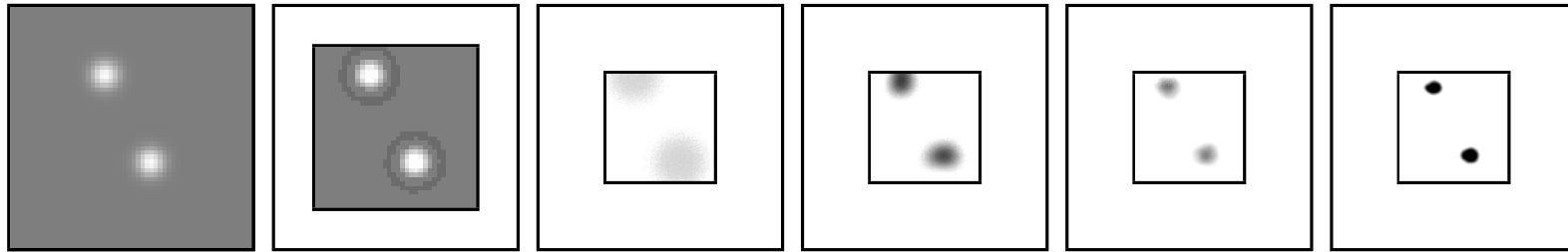
Initial disordered map



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

- Bednar, J. A. (2002). *Learning to See: Genetic and Environmental Influences on Visual Development*. Doctoral Dissertation, Department of Computer Sciences, The University of Texas at Austin, Austin, TX. Technical Report AI-TR-02-294.
- Bednar, J. A., & Miikkulainen, R. (2003). Self-organization of spatiotemporal receptive fields and laterally connected direction and orientation maps. *Neurocomputing*, 52–54, 473–480.
- Bednar, J. A., & Miikkulainen, R. (2004). Prenatal and postnatal development of laterally connected orientation maps. *Neurocomputing*, 58-60, 985–992.
- 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.

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