Summary for developmental plasticity

normal adult V1

- . rough balance between cells driven by L and R (above/below L4)
- . alternating L and R OD columns with equal width
- . L and R LGN axons terminate in distinct territories in Layer 4

newborn V1

- . no ocular dominance column
- . LGN axon terminals branch extensively, fibers from two eyes overlap

V1 of MD animal

- . physiological change: OD shifts toward the non-deprived eye
- . Anatomical change: deprived eye columns shrink, open eye columns expand
- . LGN axon terminals from the open eye take over more area

Summary for developmental plasticity

- mechanism of segregation
 - TTX , no segregation (complete lack of activity)
 - BD (binocular deprivation), normal OD (weaker but <u>balanced</u> input from both eyes)
 - activity-dependent competition
 - three-eyed frog experiment (competition leads to segregation)





Molecular mechanism of cortical plasticity

2. Neurotrophins

First neurotrophin, NGF (nerve growth factor), is discovered by Rita Levi-Montalcini, winner of the 1986 Nobel Prize





Criteria for neurotrophins to function as molecular signals in synaptic plasticity:

1) expressed in the right places and at the right times

2) expression and secretion are activity-dependent

3) regulate aspects of neuronal function

Summary	
- a	dult cortical plasticity
	lesion induced changes in cortical map
	- somatosensory cortex area 3b
	- primary auditory cortex
- ce	ellular mechanism of cortical plasticity
ŀ	Hebb's hypothesis
	- developmental plasticity
	- adult cortical plasticity
- N	Iolecular mechanisms of cortical plasticity
	- NMDA receptor
	- coincidence detector
	- block NMDA receptor, block segregation of OD columns
	- neurotrophins
	- signaling molecules in synaptic plasticity