The visual System

Retina
⇓
LGN
⇓
V1

Primary Visual Cortex V1
Background: In the retina exist multiple parallel systems of gangion cells which are specialized for different aspects of the visual world, the most prominent being the M (magno)- und P (parvo)- System.

M-cells
large dendritic fields, phasic response, motion detection

P-Cells
small dendritic fields, tonic response, form detection
The LGN is a conspicuous brain structure with an organization of the cells in 6 clearly defined layers.
The M- and P-pathway are kept separately in the LGN
Selective lesions of the M-P-System

Parvocellular lesion

Magnocellular lesion

0.5 mm
Specific deficits after lesions of the M- and P-System

- **Contrast Spatial frequency**
  - High
  - Low

- **Static**
  - Lesion M
  - Lesion P

- **Moving**
  - Lesion M
  - Lesion P

- **D**
  - Chromatic contrast sensitivity
  - Control
  - P alone
  - M alone
Overview processing in M- and P-System
Primary visual cortex (Area17, V1)

Line of Gennari specifies primary visual cortex
Cell types and afferent fibers in V1

A Inputs from lateral geniculate nucleus

B Resident cells

- Pyramidal
- Spiny stellate (local)
- Smooth stellate (local)
Information flow in V1

P-Pathway: 4Cbeta 2,3 5,6, V2

M-Pathway: 4Calpha 4B MT

To other (extrastriate) cortical areas (e.g. V2, 3, 4, 5, MT)

To subcortical areas:
- to superior colliculus, pulvinar, pons
- to LGN, claustrum
Receptive fields in V1, Layer 2/3 (P-System)

Orientation selectivity in V1

Retina, LGN
Construction of a receptive field in V1 from concentric ON-center receptive fields

Exact mechanism, by which orientation selectivity is generated, is still unknown and subject of current research.
Mechanisms of orientation selectivity

Layer 4 neurons are orientation selective in their spike output, but not in their dendritic depolarization.

Two photon Ca2+ imaging of small dendritic compartments throughout the dendritic tree.

From Jia et al. 2010
Mechanisms of orientation selectivity

Orientation selectivity is present at different dendritic sites, but can be tuned to different orientations.

From Jia et al. 2010
Mechanisms of orientation selectivity

One orientation direction is present on several dendrites, but one dendrite can have areas of varying orientation preference.

Orientation preference must be generated within this neuron, is not preimposed by synaptic inputs.

From Jia et al. 2010
Orientation columns are found in men, monkeys, cats, but not in rats and mice. But neurons in V1 of rats and mice are also orientation selective, although the cortex is not organized in orientation columns.
Orientation columns in V1

Pin wheel like organisation of orientation columns
Ocular dominance columns in V1
Blobs: for processing of colour
V1: Parallel processing
Borders in the center of the pinwheels are precise

From Ohki et al. 2006
V1: Parallel processing

Ocular dominance
M - P
Within P:
Orientation
Colour (blobs)
...

Lateral geniculate nucleus
6(C) 5(I) 4(C) 3(I) 2(I) 1(C)
Lateral intracortical connections
Lateral intracortical connections couple columns with similar orientation selectivity.
V1 is surrounded by V2

Thick stripes: M-system
Interstripes: Form analysis
Thin stripes: Colour analysis (blobs)
The M- and P-pathway in the visual cortex

Dorsal (parietal) pathway, M-system

Ventral (temporal) pathway, P-system
Motion analysis: Random dot movies

A  No correlation

50% correlation

100% correlation
Random dot movie: 5% correlation
Random dot movie: 30% correlation
Random dot movie: 100% correlation
Motion: critical role of area MT

A

No correlation

50% correlation

100% correlation

B

Monkey

Postlesion

Prelesion

Human

Brain damaged patient

Normal subjects
Random dot movie: 0% correlation
Motion: microstimulation in area MT
Illusory contours in V2
When we see what is not there!
What do you see ???
Ahaaa !!! Beebee!!!
Seeing ≠ Recognition
The inferior / temporal pathway continues with V4 and the inferior temporal cortex (IT)
Form analysis in V4 und IT
V4 und IT: Form and colour together

A. Shape selectivity

B. Color selectivity
The inferior / temporal pathway and the dorsal / parietal pathway
The M- und P-Systems
M- and P-Systems are separated, but not isolated

Transsynaptick tracing studies using the rabies virus system

3 day survival (2 synapses)
2) Retrograde label in 4B, 4Cα

6 day survival (3-4 synapses)
3) Retrog. Label in 4B, 4Cα, 4Cβ

1) Injection into Area MT

From Nassi and Callaway 2006
The P-Pathway going through Layer 4Cβ in V1 reaches area MT via V2 (thick stripes) and V3.
Plasticity in the visual system: Monocular deprivation in the young

C₁ Normal area 17

C₂ Area 17 after monocular closure of contralateral eye
The pattern of ocular dominance columns in V1

normal

after monocular deprivation
Development of ocular dominance columns
Terminal arbors in visual cortex after monocular deprivation

open eye

closed eye
Amblyopia

Critical period in humans from 6 - 8 years
NMDA-receptors involved

A  Normal development
Low power view

B  NMDA receptor blockade

C  NMDA receptor activation
High power view
Sleep enhances plasticity after monocular deprivation

MD for 6 hours

MD for 6 hours + 6 hours sleep

MD for 6 hours + 6 hours awake (sleep deprivation)

MD for 12 hours no sleep

From Frank et al. 2001
Plasticity in the adult visual system: Response after focal retinal lesions

The defect in visual cortex representation after a focal retinal lesion is filled within 3 weeks.

The filling of the defect correlates with the emergence of new persistent spines on the dendrites of Layer 5 neurons in V1.

From Keck et al. 2008
Bye, bye – and keep moving!
The visual cortex


B: The retinal projection

The axonal projection from the retina to the brain: Retinal axons form the optic fiber layer of the retina, run up to the optic disk, then form the optic nerve, partly cross in the optic chiasm, then form the optic tract which runs to

1) the lateral geniculate nucleus (lgn), where information is relayed to a projection neuron running to the

2) primary visual cortex V1

In the human the projection is crossed to about 50%, with the ganglion cell axons from the temporal half of the retina remaining ipsilateral, and those from the nasal half of the retina crossing in the chiasm (Attention: The crossed projection from the nasal half of the retina represents the temporal half of the visual field of that eye and vice versa). Lesions in the visual projection give rise to characteristic visual field deficits.

In rodents the projection is crossed 85-90%, in reptils and birds close to 100%.

There are additional CNS nuclei receiving direct retinal input:
- The superior colliculus: main retinal target in reptiles, amphibia and birds, receives retinotopic projection, in primates and human mainly for saccadic eye movements and head movements
- The suprachiasmatic nucleus (hypothalamus) for day night rhythm
- The pretectal nuclei: for pupillary reflex and eye movements.

The lateral geniculate nucleus: As for most sensory pathways, a part of the thalamus serves as relay nucleus for the projection to the cortex, for the optic axons this is the lateral geniculate nucleus. Receptive fields in the LGN are similar to those of retinal ganglion cells – little information about processing going on there. Strong recurrent projection from cortex and other visual centers: control of information flow to cortex. Organized into 6 layers: segregation of axons from the two eyes (3 layers each) and segregation of axons from M- (2 layers) and P-ganglion cells (4 layers). the principles of segregation of visual processing from the retina are reflected in the histological structure of the LGN!

C: The visual cortex

The primary visual cortex, V1, Area 17: General characteristics of V1: very special type of cortex, can be recognized on macroscopic sections due to white line in gray matter (line of Gennnari).

Information fed into 6-layered cortical structure, Information flows within cortical layers: axons from Iggn end in layer 4 on spiny stellate cells.

Still separation of M- and P-channels: M-pathway goes to layer 4Cα, P-pathway goes to layer 4Cβ

Basic scheme of intracortical connections: layer 4 ⇒ layers 2/3 ⇒ layers 5/6
Receptive fields in V1: Round spots of light (retina) produce little activation in V1, except in layer 4 cells. Cells show “orientation selectivity”. Respond best to light bars with particular orientation. The border of light is again an important aspect of the stimulus.

Organization of V1: Orientation columns: neighboring cells have similar orientation preference, form columns of 30 - 100μm diameter, then comes a new orientation. Orientation angles shift systematically from column to column, covering the whole range, resulting in a pin wheel like organization of orientation columns. Ocular dominance columns: only exist, where the visual field is covered by both eyes. Large part of cortex in human, only little area in rodents, do not normally exist in frogs and fish. Form stripes 300-500μm in width, areas which predominantly respond to input from one eye. Blobs: originally identified by Cytochrome-oxidase staining, contain many cells responding to color stimuli (Interblobs: cortex between blobs), show little or no orientation specificity. Ocular dominance columns and orientation columns overlap freely: one orientation column can extend over border of ocular dominance column, Blobs are typically in the center of an ocular dominance stripe.

Lateral cortical connections in V1: Blobs are specifically linked by horizontal connections. Orientation columns with a similar orientation are linked by horizontal connections. Distance between labeled patches approx. 1mm, horizontal connections extend for several mm.


Motion analysis (M-Pathway): Motion sensitivity already present in V1 (moving bars). Area MT (=mediotemporal) is specifically concerned with motion analysis. Many motion sensitive cells in area MT, which detect more complex aspects of motion. Lesions of MT interfere with motion detection, and microstimulation of MT can induce motion detection.

Form analysis (P-pathway): Cells in V2 respond to illusory contours. Illusory contour: interrupted line of figure which is recreated by the brain. The cell responds as if the line was there, although there is no line in receptive field of the respective neuron. Cells in V4 respond to simple shapes, cells in the inferior temporal cortex (IT, = TEO + TE) respond to complex forms, faces. ⇒ Hierarchical organization of visual areas, with higher areas responding to more complex features, at the same time the size of the receptive field of the neurons increases.
**Color analysis (P-pathway):** Most color sensitive cells in V1 are located in blobs. Most color sensitive cells in V2 are located in the thin stripes. Shape sensitive cells in V4 are often also color sensitive. Further color processing in V4 (e.g. color constancy).

Cerebral achromatopsia = Loss of color vision through brain damage.

The different pathways are of course not totally separate, but are interconnected in particular at higher cortical levels.

**D: Plasticity in the immature visual system**

**Amblyopia after visual deprivation:** one eye closed in cat or monkey after birth for several weeks ⇒ deprived eye has very poor vision, although the eye is fully intact. Similar observations in children after cataract removal, strabismus (squint), or with refractive errors in one eye. Blindness due to problem in brain: amblyopia. Only occurs after visual deprivation in newborn animals, not in adult animals ⇒ a critical period exists. Amblyopia is due to changes in ocular dominance columns. Electrophysiology: most cells only driven by undeprived eye Tracing: ocular dominance stripes of undeprived eye much wider, those of deprived eye very narrow.

- Single fiber analysis: open eye: large well branched terminal arbors.
- closed eye: small poorly branched terminal arbors.


Also works in the lgn, but there mainly prenatally, changes driven by spontaneous activity of retinal ganglion cells during development.

**Plasticity in the adult visual system:** There is substantial plasticity present also in the adult visual system, but changes are more localized, less dramatic and require more time.

Example: focal retinal lesions in the adult eye induces substantial changes of the visual representation in the visual cortex.

**Molecular mechanisms of activity dependent plasticity:** depends on NMDA-receptor activation. NT-4 activity and trk-B receptor activation are involved. Sleep is required for the consolidation of activity-dependent plasticity.

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