Structure and function of human visual cortex following peripheral disruption

Professor Brian Wandell
Stanford Center for Cognitive and Neurobiological Imaging (CNI)
Department of Psychology
Stanford University

These slides can be downloaded from
http://vistalab.stanford.edu/~brian
Plasticity and stability in human visual cortex

• What does functional magnetic resonance imaging (fMRI) reveal about the stability and plasticity of human visual cortex across the lifespan?

• What are the consequences of abnormal development for V1 maps and responses?

• What are the consequences of acquired retinal dysfunctions for V1 maps and responses?
Plasticity and stability in human visual cortex

• In congenital and developmental studies, human fMRI measures substantial cortical reorganization

• In adult retinal dysfunction produces
  A. Stable feedforward mapping
  B. Atypical cortical interactions
Progress in measuring human visual cortex


PET images were exciting but of limited quality.

These data were striate cortex mapping with PET.

“Little is known about the organization of extrastriate visual areas in the human brain. Therefore, to construct our proposal we must draw upon data from experimental work in monkeys. (Horton and Hoyt, 1991, Brain)”
Human eccentricity mapping
(DeYoe, 1994; Engel et al., 1994, 1997; Sereno 1995)
Pseudo-color representation of visual field map
Eccentricity Map
Flattened Representation

The large foveal V1 projection zone, outlined in yellow, is several square centimeters.
Angular measurements sharply delineate visual field map boundaries
More than sixteen visual field maps

Rod monochromacy

Control responses: Cone-mediated

Normal control FMRI time series

- BOLD signal (%)
- Time (sec)

- Curves displaced for clarity
- Typically rod-free
- Near periphery
Control responses: Rod-mediated


Rod vision

V1

Missing foveal activation

32°
Rod monochromats develop a visual response in the rod-free zone

*Baseler et al., (2002)*

32° cm

V1
Rod monochromat FMRI time series amplitude is large

Typically rod-free

Near periphery

Curves displaced for clarity
The population receptive field

The receptive field
Stimulus referred measurement

‘Responses can be obtained in a given optic nerve fiber only upon illumination of a certain restricted region of the retina, termed the receptive field of the fiber (Hartline, 1936)’.
Population RF estimation

Stimulus

Predicted BOLD (including HRF)

% BOLD

Parameters

(x_1, y_1, s_1)

Observed

1 cycle

Time (sec)
Population RF estimation

Stimulus

Parameters $(x_2, y_2, s_1)$

Predicted BOLD (including HRF)

Observed

% BOLD

1 cycle

Time (sec)
Population RF estimation

Stimulus

Population RF model

Predicted BOLD (including HRF)

% BOLD

Parameters $(x_2,y_2,s_2)$

Observed
Population receptive field size varies significantly between maps (Dumoulin and Wandell, 2008)
Population RF increases with eccentricity in each map (Kay et al.)
Achiasma

Visual pathways

- Right visual field
- Left visual field

LH
Conventional optic chiasm

Control
Missing optic chiasm

Achiasma
Molecular guidance at chiasm mediated by Ephrin-B2 and EphB1 (Williams et al., 2003, Neuron)
Achiasmic

Apkarian et al., E.J.N. 1994
Apkarian et al., Brain, 1995
Victor et al., Cer. Cortex, 2000
Jansonius et al., JNO, 2001
Prakash et al., JNO, 2009

Right visual field
Left visual field
Subject AC2 characteristics

- Slight decrease in visual acuity
- Slightly reduced peripheral visual fields
- No stereopsis
- Prominent infantile and see-saw nystagmus (resolved)
Right and left hemifield maps are overlaid in both hemispheres.
Right and left hemifield maps are overlaid in both hemispheres.
Modeling the time course (1 Gaussian)
Modeling the time course (2 Gaussians)
Achiasmic - Folded representation

Retina

V1

(No clear evidence of an inserted map)
Early disruption of retinal signals

Stem cells, curing blindness

Levin et al., 2010, Neuron

IMAGES COPYRIGHT MICHAEL MAY
Contrast sensitivity functions

- Similar to controls at low spatial frequency
- Substantially worse above 0.25 cpd
- Constant for the 7 years following surgery
Why can’t Mike see?
Missing signals to visual cortex

Levin et al., 2010, Neuron
Hypothesis: MM is selectively missing V1 neurons with small receptive fields.

- Poor spatial resolution in psychophysics
- In typical development, there are many neurons with small receptive fields; these compete with the large receptive field population and extrastriate feedback for synaptic space in V1.
- If the small receptive field neurons are absent in MM’s V1, and their synaptic space is given over to these large receptive field neurons, we expect to find large V1 pRF sizes.
Retinal dysfunction

Human subjects with deafferented V1: JMD
(Masuda et al., 2008)
Passive: No signal in LPZ
One-back: Signal in LPZ
JMD onset age — (Masuda et al., ARVO poster, Wed.)

Drifting contrast pattern
Fixation, no task

Teen age onset
Current: 32

Acquired JMD

Control

BOLD coherence

1.0
-1.0
JMD onset age – (Masuda et al., ARVO poster, Wed.)

Drifting contrast pattern
Fixation, no task

Birth onset
Current: 35

Congenital JMD  Control

BOLD coherence
JMD onset age – (Masuda et al., ARVO poster, Wed.)

Moving bar
Fixation, no task

Congenital JMD

Control

14
0

pRF size (deg)
Summary and conclusions
Plasticity and stability in human visual cortex

- What does functional magnetic resonance imaging (fMRI) reveal about the stability and plasticity of human visual cortex across the lifespan?

- What are the consequences of abnormal development for V1 maps and responses?

- What are the consequences of acquired retinal dysfunctions for V1 maps and responses?
Summary

1. In controls, rod signals do not elicit responses in foveal cortex.

2. In rod monochromats, normally foveal cortex is active.

3. A genetic defect that disrupts cone signaling causes a developmental reorganization in visual cortex.
Summary

1. A genetic defect that disrupts crossing at the chiasm signaling causes a developmental reorganization in visual cortex.

2. Despite the entirely disrupted maps in V1, the rest of the brain figures out what to do.
Summary

1. Eliminating fine resolution images at a young age interfered with normal development. Restoration of the image for the adult did not succeed in producing high quality vision.

2. The inability to restore high quality vision may be caused by a specific system – the high resolution foveal cells – that is late-developing.
Summary

1. The fMRI measures individuals with macular (or peripheral) loss are consistent with a stable feedforward signal.

2. Interactions with cortex introduced by task demands, however, are atypical.

3. The stability of the feedforward signal is a hopeful sign for the viability of retinal prosthetics.
Plasticity and stability in human visual cortex

• In congenital and developmental studies, human fMRI measures substantial cortical reorganization

• In adult retinal dysfunction produces
  A. Stable feedforward mapping
  B. Atypical cortical interactions
To Mike:
Thanks from all of us for your advice and support
Structure and function of human visual cortex following peripheral disruption

Professor Brian Wandell
Stanford Center for Cognitive and Neurobiological Imaging (CNI)
Department of Psychology
Stanford University

These slides can be downloaded from http://vistalab.stanford.edu/~brian