Lecture 8: Brain Plasticity

Clinical and Experimental Neuropsychology
Developmental Plasticity

• Neuroplasticity is any change in neuronal form or function

• Primate fetal brains contain 30-60% more axons than adult primate brains. Corpus callosum: 400% more axons

• During gestation there are widespread changes in type and location of cells and their interconnections

• Cell migration largely ends at birth but changes in local cortical connectivity continue

• Simple exposure to particular stimuli (i.e. learning not required) determines brain organisation during critical period of infancy
Developmental Plasticity

Impact of brain lesions

Before age 1 – greatest impairment

Age 1-5 – reorganisation of brain function

Age 5+ - little or no sparing of function
“The brain was constructed to change” (Merzenich, 2003)

• Marked lack of plasticity once the ‘sensitive periods’ of development have passed

• Long held assumption that the mature brain is hardwired

• Major advances in cognitive ability were ascribed to alterations in connectivity between existing neurons

• Growing realisation that plasticity still occurs to some degree – recovery from injury, learning, memory

• From childhood onwards the brain modifies its machinery in a more selective way, refinement.

• Selective representation of behaviorally relevant input
Exploring Neuroplasticity

1. Effects of Practice

2. Recovery from Brain Injury
Sensory and Motor Maps can be modified with experience

Merzenich et al (1980s) Primate Studies
Pascual-Leone et al’s (1993) Braille readers

• Mapped motor cortex for representations of reading finger in Braille readers and controls bilaterally
Findings

- Cortical representation of reading finger significantly enlarged at the expense of representation of other fingers in Braille readers only

- Can even observe changes within a day when Braille is practiced for 4-6 hours (Pascual-Leone et al., 1995b)

- Replicated with piano players & other learning experiments

• Training on a **specific piano exercise** with one hand, 2 hr/day.

• Increased motor output maps after 5 days compared to untrained hand.

• Control group **played piano at will** for 5 days – intermediate map
Enhanced Representational Fidelity

Structured, goal-directed practice is key
• **Second Experiment** – asked Ss to practice piano exercises *mentally* instead of physically

![Physical exercise](image)

![Mental exercise](image)

• **Thinking is doing!** Uncovers a fundamental link between thought and action

• Major implications for rehabilitation, sports psychology
• Plasticity changes are inherently competitive

• As a result, they are not always positive
Simultaneous stimulation of 5 fingers – reduced differentiation in cortical maps
Phantom Limbs

- Reorganisation in somatosensory cortex following amputation / denervation

- De-afferented cortex becomes functionally innervated by surrounding cortex

- Phantom limbs: awareness of a nonexistent body part in mentally competent individuals

- 6 months post-op: 90% experience phantom limb sensations, 67% phantom limb pain (Jensen et al, 1983)
• “referred sensations” on the phantom limb when adjacent sites are stimulated

• Topographical effect: when moving a stimulus across adjacent area, speed, distance & direction of movement are felt in phantom limb
Examples of labile body image

- Driving a car – extension of body boundaries
- Sense of balance following lengthy bus or boat journey
- Body image illusions…
  - Rubber hand illusion
Changing the body image with illusions

• Based on the Pinocchio illusion (Lackner, 1988) – sensation that body parts change size & shape

• These illusions make use of the fact that vibration of the skin over the tendon of a joint extensor muscle elicits a vivid kinaesthetic illusion that the joint is passively flexing:

(From Ehrson et al, 2005)
Neural Substrate of Body Size: Illusory Feeling of Shrinking of the Waist

Lackner (1988) – if vibrated body part touches another body part, the sensation arises that the touched part changes size/shape:
Results

• activity in the cortices lining the left postcentral sulcus and the anterior part of the intraparietal sulcus reflected the illusion of waist shrinking, and this activity was correlated with the reported degree of shrinking

• Suggests that altered body images are computed by higher-order somatosensory parietal areas, through integration of more elementary somatic signals
Mechanisms underlying neuroplasticity

1. Change in balance of excitation and inhibition - can happen very quickly
   - If inhibition is removed, regions of functional influence are increased

2. Strengthening of existing synapses, e.g. through long-term potentiation (LTP)
   - Weakening of synapses through long-term depression (LTD)
3: change in neuronal membrane excitability

4: anatomical changes, e.g. sprouting of new axonal terminals & formation of new synapses

- Anatomical changes take longer…traditionally not considered as feasible mechanisms of plasticity
- Mechanisms are not mutually exclusive
- Plastic changes occur on multiple time scales and according to multiple rule-based systems
Hebbian learning

• Important framework for bridging neural and behavioural levels of analysis

• Key principle: two neurons or groups of neurons that are disconnected may become connected if they are repeatedly activated at the same time
  “Cells which fire together wire together”

• Conversely, if the firing pattern between neurons is repeatedly nonsynchronous then the connection may become inhibited
  “Cells which fire apart wire apart”
a. Two small lesions

(b) A single large lesion
Maladaptive connections
• Even in clinical/rehab settings, not all experience is good…

• Some type of stimulation may exaggerate deficits in a vulnerable circuit

• Competitor network may be activated which further inhibits an already malfunctioning network (Kapur, 1996)

• More during rehab lecture…. 
Promotion of Adaptive connections

• Best case scenario: to foster adaptive connections within a lesioned network and minimise the possibility of accidently fostering faulty connections with other networks.

• Taub et al (1993). Patients suffer unilateral strokes leading to poor function of one upper limb

• What is the best way to activate the dysfunctional limb in view of Hebbian principles
Taub’s approach:

– Discourage patients from using their good limb, keep hand at rest in pocket.

– Encourage use of dysfunctional limb

– Findings: significant improvement in motor function after 2 weeks lasting up to two years

– So, positive effects resulting from a specific pattern of stimulation; activation of one limb combined with deactivation of the other
Neurogenesis in the adult human brain?

- Eriksson et al (1998): new cells emerging in caudate nucleus and hippocampus

- Partly experience-dependent: more cell genesis in animals in enriched environments than impoverished (Gould et al, 1999)

- Stress may reduce occurrence of neurogenesis (e.g. Bremner et al 1999, Vietnam Veterans)
Evidence of Structural Plasticity in Adulthood

Maguire et al. (1997) Experienced taxi drivers found to have larger right hippocampi than novices and controls.

FIGURE 2. Reprise of the voxel-based morphometry (VBM) taxi driver result (originally reported in Maguire et al., 2000). A: Increased grey matter volume in the posterior hippocampi in taxi drivers compared with control subjects. B: Area in the right posterior hippocampal region where the longer the time taxi driving, the greater the grey matter volume.
Negative Plasticity and Cognitive Decline?

• Final point – the flipside of this plasticity is that cells and connections may atrophy and die due to **lack of use** – “negative plasticity”

• drive to keep the mind active into old-age to prevent cognitive deterioration like Alzheimer’s Disease and dementia


• Research on plasticity shows we remain amenable to some degree of change throughout the lifespan
Cognitive Enrichment offsets Cognitive Decline

Coq and Xerri (2001) Neuroscience
Memory enhancement in healthy older adults using a brain plasticity-based training program: A randomized, controlled study


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Normal aging is associated with progressive functional losses in perception, cognition, and memory. Although the root causes of age-related cognitive decline are incompletely understood, psychophysical and neuropsychological evidence suggests that a significant contribution stems from poorer signal-to-noise conditions and down-regulated neuromodulatory system function in older brains. Because the brain retains a lifelong capacity for plasticity and adaptive reorganization, dimensions of negative reorganiza-

hanced representational fidelity of, the learned stimulus or behavior (9). For example, in monkeys trained to detect a specific pattern of stimulation to the fingers the somatosensory cortex reorganizes to represent that specific input pattern with large, well organized, and spatiotemporally coherent responses (10–12), whereas violin players have been shown to have stronger and more distinct representations of the fingers in the right hemisphere, corresponding to the individuated finger mov
Open questions remain…

- What are the mechanisms and precise principles behind functional & structural neuroplasticity?
- Useful versus harmful stimulation?
- Are all parts of cortex equally flexible? What about subcortical structures?
- To what extent are higher cognitive functions subject to experience dependent plasticity? Generalisation?
Predictors of recovery?

- Age
- Sparing of frontal lobes
- Awareness of deficit
- Lesion size
- Extent of rehabilitation
- High IQ
References

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