

Chapter 13 Memory and the Brain

Carl Lashley's experiment:

Purpose: To find the location of memory traces in the brain

Memory Traces/Engrams – The physical and biochemical changes in the brain that form that allow memories to be encoded, stored, and recalled

Method: Lesions of varying size and location were made in the brain

Result: **Mass Action Principle** - The reduction in learning observed is proportional to the amount of tissue destroyed, and the more complex the task, the more disruptive the lesion

The location of the lesion seems inconsequential
(See Figure 13.1)

Conclusion: Memory is distributed in the brain. This is in accordance with the connectionist model and the theory of parallel distributed processing (See Box 2B)

Donald Hebb

Theory: Memories are stored as networks of neurons called **cell assemblies** and formed via Hebbian learning – “cells that *fire* together, *wire* together”

Habituation – refers to a reduced response when the same stimulus is repeated over and over.

Sensitization – refers to an increased response to a stimulus when it is paired with an aversive stimulus e.g. electric shock.

Long-term potentiation – long lasting synaptic enhancement; occurs with high frequency stimulation over a brief period

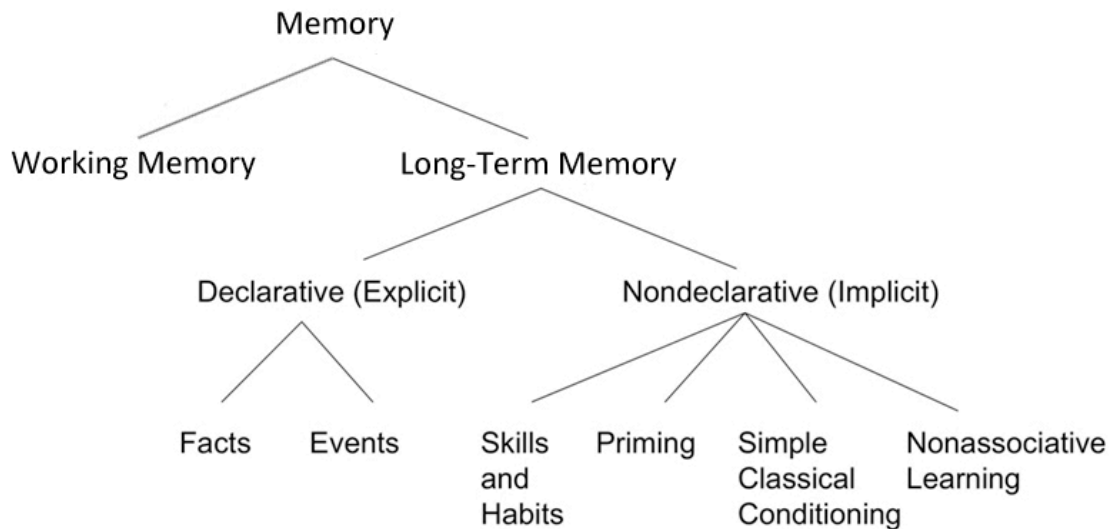
Specificity – only those synapses activated during stimulation are enhanced

Associativity – For two pathways converging on a single neuron: if one pathway is weakly activated while the other is strongly activated; then both pathways show LTP

Long-term Depression – a mechanism of long lasting synaptic inhibition
(See Box 13A)

What morphological changes in **dendritic spines** are associated with LTP?
(See Figure 13.6)

Memory Systems



(See Figure 13.10)

- Be able to give an example and explain each type of memory

Patient H.M.

Lesion: Anterior temporal lobe

Characteristics of H.M.'s memory loss:

1. No deficits in cognitive domains other than memory
2. **Anterograde Amnesia** - cannot remember events that occurred *after* the surgery.
Retrograde memory is intact (memory of events *before* the surgery).
H.M. does not have **Retrograde Amnesia**
3. All types of **explicit memory** are impaired – regardless of sensory modality
4. H.M.'s **Working memory** is intact
5. Can form new **Non-declarative** memories (intact)
e.g. H.M. can pass the **Mirror Drawing Task**

- What conclusions can be drawn from this information?

(See Page 341)

Patient M.S.

Lesion: removal of right occipital regions – blind in the left eye
Shows a **double dissociation** of working and long-term memory

(See Figure 13.14)

Three stages of memory Processing:

1. **Encoding**
2. **Storage**
3. **Retrieval**

Systems level heterogeneity of memory v. Cellular level homogeneity of synaptic change
(See Figure 13.15)

Memory **Consolidation** – progressive stabilization of long-term memories; a post-encoding process

Synaptic consolidation – occurs within a few minutes after encoding; cellular changes occur

System consolidation – days, months, even years; system level reorganization to support memories

Standard consolidation theory – a theory of consolidation for declarative memory; suggests a mechanism by which memories formed in the hippocampus are transferred to the cortex

(See Figure 13.17)

Retrieval of consolidated memories

(See Figure 13.18)

- Encoding is not completed immediately, but requires a process of synaptic consolidation
- Storage involves a process of system consolidation
- Retrieval is the activation of the a stored memory trace (engram)

Note:

A *Double Dissociation* is defined as a situation in which:

A impairs the function of X but not Y; while,

B impairs the function of Y but not X

This strengthens an argument that A is responsible for function X, but not function Y