

## PSYCO 452

### Week 4: Exploring Distributed Memory

- The Cell Assembly
- Simulating Hebbian Plasticity
- Holographic Memories
- Biological Mechanisms Of Association

## Course Trajectory

When	What
Weeks 1-3	Basics of three architectures (DAM, perceptron, MLP)
Weeks 4-6	Cognitive science of DAMs and perceptrons
Week 7	Connectionism and Cognitive Psychology
Weeks 8-10	Interpreting MLPs
Weeks 11-13	Case studies (interpretations, applications, architectures)

## Chapter 11 Discussion

- Questions?
- Important Terms
  - Hidden unit
  - Credit assignment
  - Backpropagation of error
  - Generalized delta rule
  - Instructionism
  - Selectionism
  - Parity problem



## Hebb's Cell Assembly

- In his 1949 *The Organization Of Behavior*, Canadian neuroscientist Donald Hebb developed a neural theory of perceptual learning
- His core idea was the [cell assembly](#)
- A cell assembly is a dynamic network of multiple neurons
- Activity reverberates and persists in the assembly over a period of time
- The activity pattern can be dynamic – there is a temporal flow of activity through an assembly

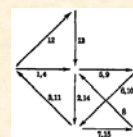


FIGURE 10 Arrows represent a simple "assembly" of several chains being according to the numbers on each (the pathway "1, 4" fires first and fourth, and so on), illustrating the possibility of an "alternating" reverberation which would not extinguish as readily as that in a single closed circuit.



Donald Olding Hebb

## Biology Of Cell Assemblies

- Hebb provided a neurophysiological postulate to explain how cell assemblies developed
- His postulate: “When an axon of cell A is near enough to excite a cell B and repeatedly or persistently takes place in firing it, some growth process or metabolic change takes place in one or both cells such that A's efficiency, as one of the cells firing B, is increased” (Hebb, 1949, p. 62)



Donald Hebb

## An Excitatory Theory

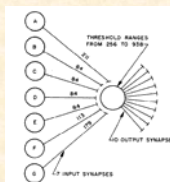
- Hebb's theory is essentially one of increased excitation
- “Any two cells or systems of cells that are repeatedly active at the same time will tend to become ‘associated’, so that activity in one facilitates activity in the other” (Hebb, 1949, p. 70)
- In the 1940s many physiologists were opposed to the notion of inhibition because it caused trouble for the electrical theory of synaptic transmission (Milner, 1957)



Donald Hebb

## Early Simulation #FAIL

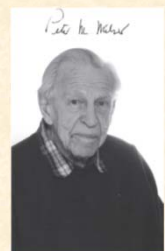
- Rochester, Holland, Haibt and Duda (1956) attempted to bring Hebb's cell assembly theory to life as a computer simulation
  - 69 different neurons, with random connections from each neuron to 10 others
  - Connections were updated according to Hebb's 1949 theory
- Rochester et al. (1956) witnessed “weak reverberation” in which there was a tendency for larger numbers of simulated neurons were simultaneously active
- However, many elements of Hebb's theory did not clearly emerge!
- “This kind of investigation cannot prove how the brain works. It can, however, show that some models are unworkable and provide clues as to how to revise the models to make them work” (Rochester et al, 1956, p. 88)



An example neuron from the Rochester et al. simulation

## Inhibitory Revision

- Peter Milner revised Hebb's theory with his “Mark II” cell assembly published in *Psychological Review* in 1957
- Milner's key contribution was to add inhibitory signals to Hebb's theory
- “The model differs from Hebb's in that an inhibitory regulatory system is postulated which limits (to a minute fraction of the total) the number of cortical neurons that can fire simultaneously, and insures that those firing are dispersed as widely as possible” (Milner, 1957, p. 252)



## Distributed Associative Memory

- From 1956 on there was a great deal of simulation research that explored the standard pattern associator
- “Versions of Figure 1 have a long history (e.g. Taylor, 1956, Figs 9 & 10; Steinbuch, 1961, Fig. 2; Kohonen, 1977, Fig. 1.9; Rumelhart et al, 1986c, Chap. 1, Fig. 12, Chap. 9 Fig. 18, Chap. 12 Fig. 1, Chap. 18 Fig. 3; Schneider, 1987, Fig. 1; McClelland & Rumelhart, 1988, Cap. 4, Fig. 3). Physiological analogs of this figure have appeared in sources ranging from Anderson et al. (1977, Fig. 1) to James (1890/1950, Fig. 40)” (Dawson & Schopflocher, 1992, p. 205)
- Inhibition is a general characteristic of this model, particularly if processor activity can be negative

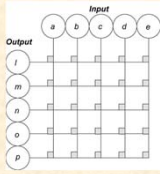


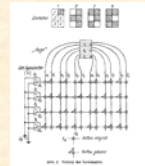
Figure 1: The standard pattern associator

## Die Lernmatrix

- One of the first distributed memories was Steinbuch's (1961) Lernmatrix
- It consisted of a set of input binary switches and an output set of switches (pairs, excitatory and inhibitory)
- The learning matrix associated patterns of input switch positions with output switch positions, permitting the system to associate input/output responses

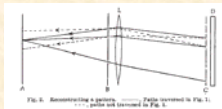
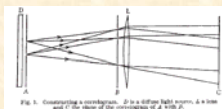


Karl Steinbuch



## Holographic Associative Memory

- The mathematics of holograms suggest an alternative approach for the creation of distributed associative memories
- The process of convolution is used to associate two patterns together; multiple associations can be stored in the same hologram
- When a cue is correlated with the hologram, what the cue was associated can be recalled

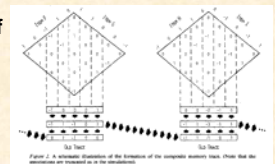
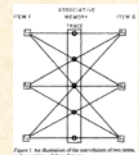


## CHARM: Convolution

- Metcalfe Eich's (1982) CHARM model is a holographic associative memory that creates associations using the holographic operation of convolution
- Convolution involves taking the outer product of two to-be-associated vectors, summing the diagonals of the result, and adding the sum to a vector that represents the memory trace

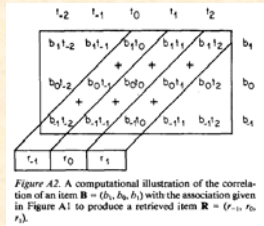


Janet Metcalfe



## CHARM: Correlation

- Recall in CHARM is accomplished by the holographic operation of correlation
- In essence, correlation involves taking the outer product of a cue with the memory trace
- Then, all of the longest diagonals are summed to produce the retrieved vector



## CHARMed Behavior

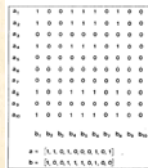
- CHARM simulates a number of regularities that govern human associative memory
- "Nearly all of the predictions and applications depend on the interactive nature of the holographic association and of the composite trace. The events stored in such a memory combine and interfere with one another so that the output from memory is different from the input to it" (Eich, 1982, p. 657)

Prediction or phenomenon	Simulation	Favorable		Evidence
		Yes	No	
Interaction between correct recall and similar associates depending on whether pattern-retrieval or remote memory of similar or of unrelated items	1	X		Experiment 1
Equal frequency of retrieval and response time latencies	1	X		Experiment 1
Control properties of the iterations when cue and target items are similar (but when they are unrelated)	2	X		Experiment 2
Stimulus generalization	3	X		Experiment 4, (Eich, 1982), Experiment 4
Retrieved item similar to target, even if recall of cue better than target	3	X		Experiment 4
Response of a particular pair learned first will be a heterogeneous list	—	X		Taking, Note 3
Interaction between number of category exemplars presented and cue salience of prototype as compared to exemplars	4	X		Hertz, Cross, Gauthier, and Schwartz, 1973
Prototype more resistant to forgetting than general exemplars	5		X	Peters and Kavel, 1970; Gauthier, Schwartz, Kavel, and Patten, 1972
Non-associative-specific interference: A-B-C gives worse recall than A-B alone	Minsky and Marston, 1961	X		McClelland, 1964
Transfer of learning: A and B recall in A-B-X-D paradigm with MNEF testing	6	X		Bates and Underwood, 1970
Independence of B and D responses in the A-B-C-D paradigm	6	X		Martin, 1971
All of the transfer or retention effects are in the original surface	7	X		Ogden, 1965; Martin, 1965

Note: MNEF = modified free recall.

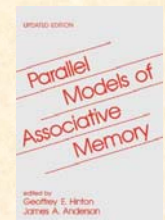
## Holography And Hebb Learning

- Pike (1984) developed a proof that established the formal equivalence of holographic associative memories and distributed associative memories that use Hebb-style learning
- Pike went on to claim that holographic memories had a number of properties that made them biologically implausible
- He argued that Hebb-style learning is more consistent with the neuroscience of memory



## The Rise Of Associative Memory

- Minsky and Papert's publication of *Perceptrons* in 1969 led to the demise of old connectionism
- Very little research on artificial neural networks was being published in the 1970s
- However, in the early 1980s there had been enough developments in the study of distributed associative memory to open the door for the connectionist revolution
- Hinton and Anderson's 1981 book on such memories has become a classic, and led the charge of the connectionist revolution





## Biological Mechanisms Of Association

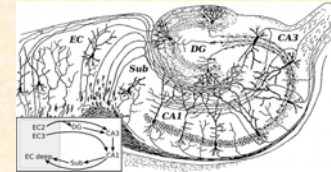
- Hebb made specific proposals concerning the biological mechanisms that caused cell assemblies to form
- “The most probable suggestion concerning the way in which one cell could become more capable in firing another is that synaptic knobs develop and increase the area of contact between the afferent axon and the efferent soma” (Hebb, 1949, p. 62)



Donald Hebb

## The Hippocampus

- The hippocampus is a component of the limbic system
- There are reasons to believe that it is a locus of Hebb-like learning



Hippocampus pathways

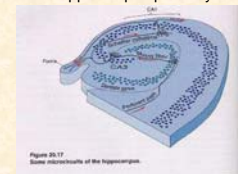
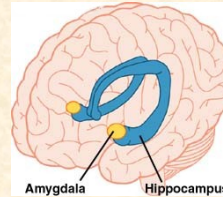


Figure 28.17 Basic microstructure of the hippocampus.

## The Hippocampus And Memory

- After surgery that resulted in the bilateral removal of his hippocampus, Henry Molaison was unable to form new memories, though existing memories were intact



Henry Molaison

- Dr. MILNER: Do you know what you did yesterday?
- H.M.: No, I don't.
- Dr. MILNER: How about this morning?
- H.M.: I don't even remember that.
- Dr. MILNER: Could you tell me what you had for lunch today?
- H.M.: I don't know, to tell you the truth



Brenda Milner

## Long-term Potentiation

- Bliss and Lømo (1973) discovered long-term potentiation in the hippocampus
- Under particular conditions of stimulation, the effectiveness of a synapse would increase
- The increase in potentiation could last for surprisingly long periods of time
- In general, long-term potentiation occurs when there is elevated activity in both the pre-synaptic and the post-synaptic neuron
- This is exactly like Hebb learning!

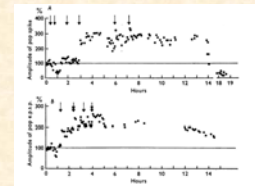
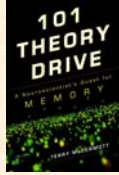


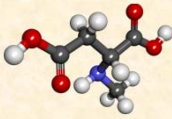
Fig. 12 Two examples of potentiation lasting for many hours. A, the amplitude time course of the potentiation of the hippocampal synapse for the experiment presented in Fig. 4-6. A total of six conditioning pulses, each at 150ms for 15 sec were given. B, the time course of a p.p.s. potentiation in another experiment in which conditioning pulses at 150ms for 10 sec, and 100ms for 5 sec were given alternately (single and double-headed arrows respectively).

## NMDA Receptors

- Gary Lynch has proposed a theory of how associative memories are created in the brain
- The theory emphasizes the role of N-methyl-D-aspartate receptors in the hippocampus
- When these receptors are active, biological changes occur that increase the efficacy of synapses
- Long-term potentiation!

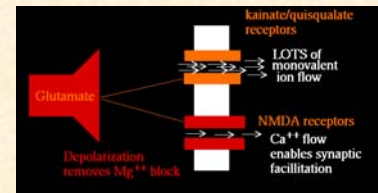
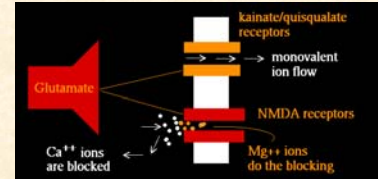


Gary Lynch



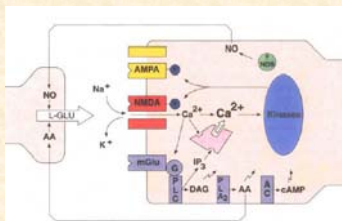
## Neural Mechanism Of Hebb Learning

- With low frequency presynaptic activity, then LTP does not occur because NMDA receptors are blocked
- High frequency resynaptic activity coupled with postsynaptic activity (contiguity!) removes the block, allowing NMDA receptors to function, and allowing synaptic efficacy to be modified



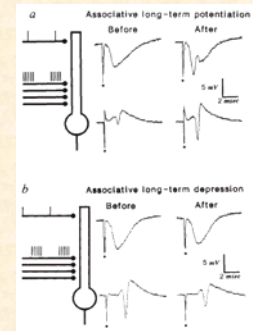
## Importance Of Calcium

- Calcium ions play a critical role in neural processing mediated by NMDA receptors
- It is generally hypothesized that calcium is involved in a variety of enzymes that themselves modify synaptic properties to produce changes in potentiation



## Long-term Depression

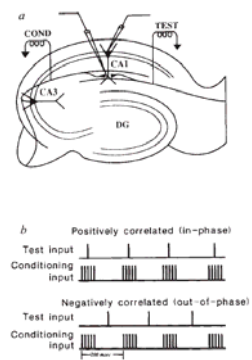
- Hippocampal LTP is an excitatory phenomenon
- There are several reasons to believe that analogous inhibitory effects are required for Hebb-like learning
- Long-term depression (LTD) is such an effect, where lack of correlation between to-be-associated signals causes a decrease in synaptic efficacy



'After' is 30 minutes post experiment

## Producing LTD

- Stanton and Sejnowski (1989) presented two trains of inputs to locations in the hippocampus
- When the trains were in phase, LTP was produced
- When they were out of phase, the result was LTD
- LTD does not involve NMDA receptors – it is not disrupted by AP5



## LTD And Hebbian Synapses

- “These experiments identify a novel form of Hebbian synaptic plasticity in the hippocampus and confirm the predictions made from a covariance model of information storage in neural networks” (Stanton & Sejnowski, 1989, p. 217)



Patric K. Stanton



Terry Sejnowski

## Hebb's Legacy

- “Stemming from the postulate, Hebb's name is increasingly used as an adjective, so that we have the Hebb synapse, Hebbian synaptic plasticity, Hebbian learning rules, Hebbian neural networks and even anti-Hebbian learning. The postulate forms part of Hebb's neural theory of perception, and much of our current understanding of functional neural connections is based on Hebbian concepts” (Brown & Milner, 2003)



Donald Hebb