

The Mind Behind the Hebbian Synapse *

Donald O Hebb

Shriya Palchadhuri

How do neurons orchestrate behaviour? Let us trace our footsteps back to one of the first hypotheses proposing an alliance between the brain and behaviour. This article trace Donald Hebb's life, and how, in the neurophysiology milieu of the 20th century, he revolutionized the way psychologists and neuroscientists viewed the brain, bringing a unifying concept of the mind with his publication of *The Organization of Behaviour*. In closing, the legacy of Hebb in the after-years of the monograph is discussed in the context of contemporary discoveries in the realm of neural plasticity and learning that revisit Hebbian concepts.

Introduction

Donald Olding Hebb is a Canadian psychologist most famously known for his unifying theories attempting to understand the physiology of the mind and its experience-driven learning processes. He described his supremely influential monograph, *The Organization of Behaviour* (1949), as “a general theory of behaviour that attempts to bridge the gap between neurophysiology and psychology, as well as that between laboratory psychology and the problems of the clinic” [1]. It became the groundwork for future scientists attempting to use computational models to understand the cognitive abilities of the mind; and in this regard, he is rightfully called the ‘Father of Neuropsychology’. Hebb’s core postulate that gave the neuroscience world eponymous expressions such as the *Hebbian synapse* and *Hebbian learning rule*, were strikingly ahead of his time, and his theories remained untested for long, for want of investigative capabilities at the time. Nevertheless,



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Keywords

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70 years after his book, Hebb remains an often, and indeed more reiterated name than in his time, because of the relevance his theories still hold to modern neuroscience and his understanding of the brain. This article traces Hebb's life, his presence in the neurophysiology milieu of the 20th century and the influences that formulated his revolutionizing contribution in changing the course of neuroscience. In closing, the legacy of Hebb in the after-years of *The Organization of Behaviour* is discussed in the context of contemporary discoveries about neural plasticity and learning.

1. Biography

1.1 *Early Life*

Donald Hebb was born on 22 July 1904 in Chester, a small coastal village in Nova Scotia, to Arthur Morrison Hebb and Mary Clara Olding. Both his parents were medical practitioners in Chester, with Mary, his mother, being only the third woman in Nova Scotia to have obtained a medical degree. Of the four Hebb children, Donald was the eldest. In 1920, the Hebb family moved to the town of Dartmouth, and Mary died of cancer in the same year.

1.2 *Education*

In 1921, Hebb entered the Faculty of Arts at Dalhousie University in Halifax, and with the intention of becoming a novelist, he graduated with a B.A. English in 1925. He soon began teaching at his old school in Chester, but within a year, disillusioned with academic life, he quit. Hebb was especially stirred by the works of the time such as Freud and was becoming increasingly more interested in psychology. In 1927, Hebb applied to do graduate work in the psychology department of McGill University in Montreal, but he was given a reading list and told to come back next year. In 1928, he was accepted for his M.A. at McGill. Two years down, however, he was bedridden with a tubercular hip, forcing him to write a theoretical thesis in bed. Hebb drew immense inspiration from the works of Sherrington and Pavlov and wrote his thesis on



the role of intrauterine learning in spinal reflexes, titled *Conditioned and Unconditioned Reflexes and Inhibition*, which he was later quite critical of. One of the two examiners of the thesis was Professor Boris Babkin, a former student of Ivan Pavlov. Babkin arranged for Hebb to gain some practical experience in classical conditioning techniques from Leonid Andreyev of Pavlov's Institute in St. Petersburg, a time clouded in Hebb's waning enthusiasm and scepticism about Pavlovian learning concepts.

1.3 *PhD With Karl Lashley*

Hebb initially wrote to Robert Yerkes at Yale, and was offered a position to study for a PhD. Babkin, however, urged him to apply to Lashley if he wanted to learn about physiological psychology, and in July 1934 Prof. Karl Lashley accepted Hebb to work with him at the University of Chicago. Hebb began his work on spatial orientation and learning, but had to move to Harvard University, when Lashley accepted a position there. His initial project remained incomplete (until much later) but Hebb earned his PhD degree in 1936 from Harvard on his work on the visual perception of rats reared in darkness.

1.4 *Montreal Neurological Institute (MNI) with Penfield*

At the time, the famous neurosurgeon Wilder Penfield was directing the Montreal Neurological Institute (MNI) and had a vacancy to fill. On Babkin's recommendation, Penfield accepted Hebb as a Fellow at the MNI for two years to work on psychological changes of patients who had undergone brain surgeries, such as removal of their frontal lobe/s. Psychologists of the time had placed the frontal lobe of the cerebral cortex as the seat of human intelligence, from observations that this area was relatively small in animals with lesser intelligence. Hebb's research with patients, however, left him deeply intrigued—large frontal lobe lesions had little or no effect on intelligence scores [1].



1.5 Academic Appointments

Hebb's observations, and the influence he drew from Spanish neuroanatomist and psychologist Rafael Lorente de Nó's work, led him to speculate that intelligence is not an innate skill but acquired through life experiences.

Hebb never studied patients again, but he continued to pursue this question as a lecturer and then an assistant professor at Queen's University, Kingston, Ontario. Through collaborations with Penfield over the following years, he delved deep into post-lesion effects on personality and behaviour, later to understand that he was relying too heavily on standard tests of intelligence that failed to measure subtle changes in personality. Hebb's observations, and the influence he drew from Spanish neuroanatomist and psychologist Rafael Lorente de Nó's work, led him to speculate that intelligence is not an innate skill but acquired through life experiences.

While in Ontario, Hebb also theorized that adult intelligence was crucially moulded by experiences during early childhood, basing his argument on the results of his research at the MNI. The paper was ignored at the time, although it gained much popularity later and was even embodied in pre-school enrichment programs like 'Head Start' [3]. But the concept was too advanced in 1940 and against the views of most psychologists who defined intelligence as an innate attribute. In an attempt to explain his observations on childhood influences on adult behaviour as well as the efficiency of frontal lobe lesions, Hebb hypothesized that the region's role was not to think but rather to acquire immense knowledge during infancy and childhood. However, experiments to determine the relative effects of early and late brain lesions failed to support this idea. Nevertheless, they provided a stepping-stone to Hebb's later theories.

Hebb joined Lashley again, when Lashley was appointed the head of the Yerkes lab of primate biology at Florida. It is Hebb's studies here that led to publication of *The Organization of Behaviour* (1949).

In 1947, Hebb was appointed Professor at McGill University, and within a year, he took over as the Chairman of the Psychology Department. He soon became the President of the Canadian Psychological Association, and then that of the American Psycho-



logical Association. Upon his retirement, he moved back to Dalhousie. Donald Hebb passed away on 20 August 1985. He was posthumously inducted into the Canadian Medical Hall of Fame in October 2003.

2. The Neuroscience Climate of the 20th Century

Hebb's move to Florida was primarily to test his theories of intelligence as an outcome of experience and not just as an innate characteristic in chimpanzees in which Lashley wanted to study learning. However, primate experiments proved much more difficult than rodent experiments, and the first surgery ever done on chimpanzees only happened towards the end of Hebb's five-year stay, but this didn't deter Hebb from theorizing and publishing heavily on emotionality and behaviour (of dolphins) during this time [4]. Hebb was increasingly becoming convinced that psychology and behaviour were closely tied with brain function—an idea that was surprisingly novel for psychologists of the time—and this gradually led to the genesis of *The Organization of Behaviour* (1949).

The 20th century was one of the most admirable phases of neurophysiology, even though the field was still suffering from the lack of appropriate investigative tools to study function. In the previous century, Ramón y Cajal had argued in favour of discrete neurons as the unit of the brain, and with his illustrations, had beautifully demonstrated the various types that spanned the cerebral cortex and the cerebellum. From this, together with studies from other histologists, the complexity of the brain was quite evident, not just in terms of subtypes and regions, but also connections. Neurophysiologists began studying the reflex arc, as it provided a simpler model to link structure with function. Soon after, Sherrington introduced the concept of the 'synapse' as the points of contact between neurons (*Figure 1b*) for explaining spinal reflexive behaviour, shifting the focus of physiological learning theories toward synaptic transmission. More work by Keith Lucas and Edgar Adrian and others, established neural firing and con-

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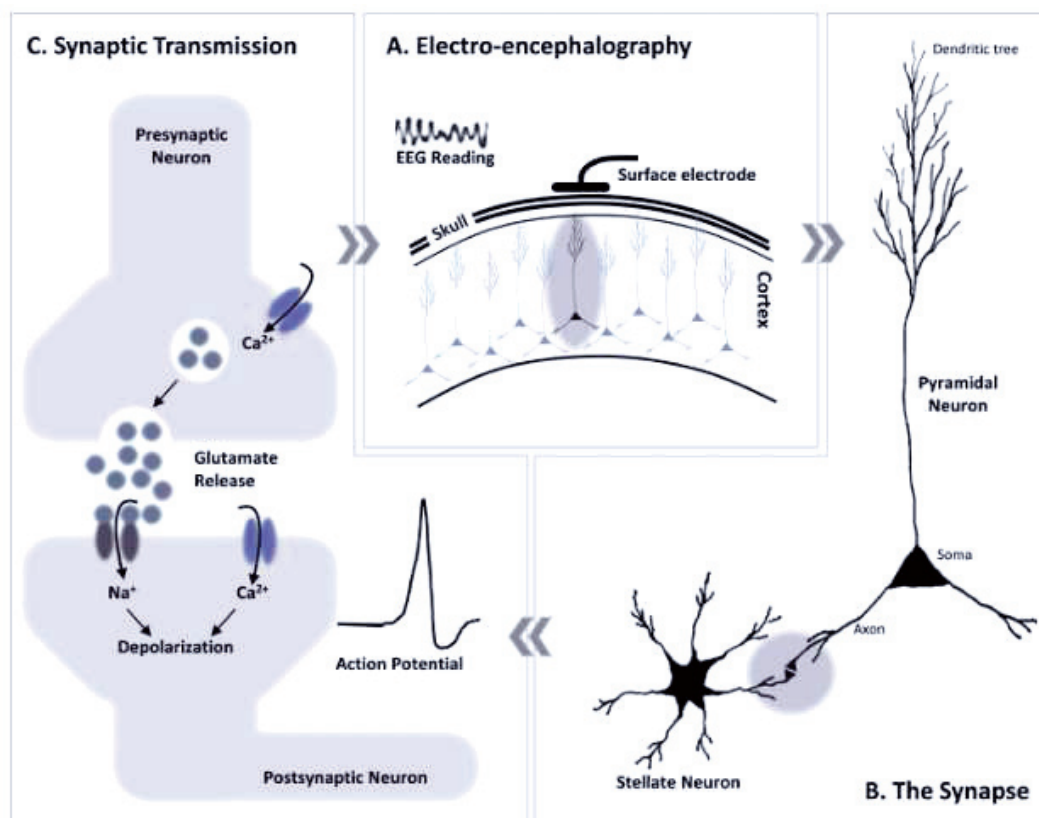
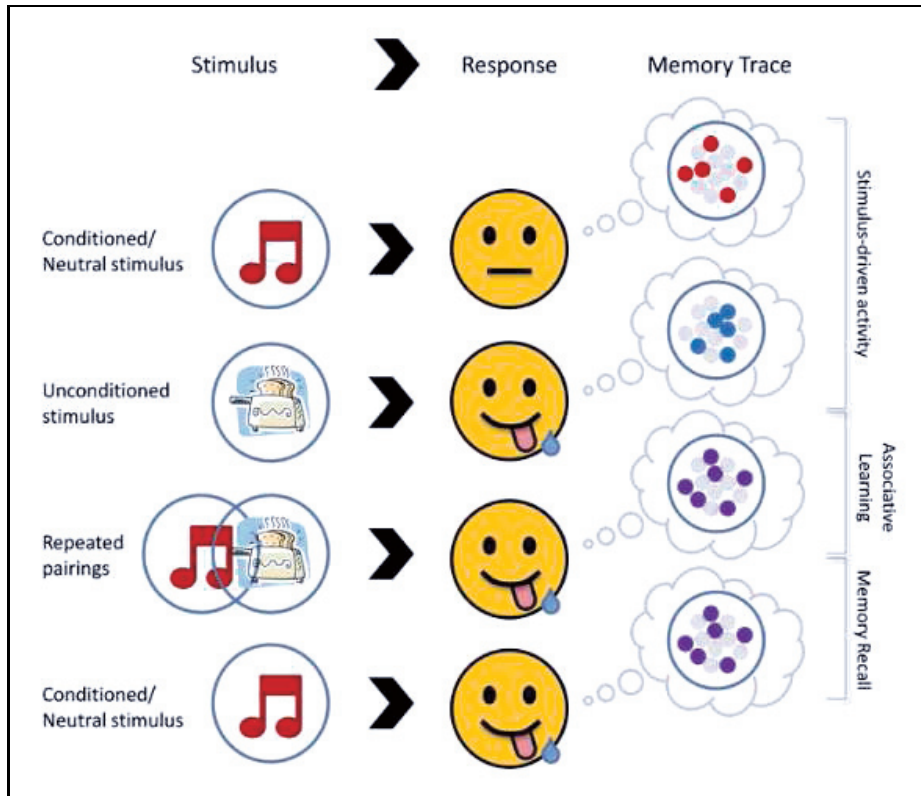


Figure 1. Neuroscientific Principles Across Scales. (For details see *Box 1*)

duction properties, such as the refractory period, all-or-none firing, and firing frequency adaptation. Nevertheless, neurophysiologists were left with the lasting question of whether these properties applied to central synapses.

Recording neurophysiology brought on a new era. Gasser and Erlanger's experiments of toad optic nerves led to the idea of action potentials or 'spikes' as the message carriers in the brain but it is important to remember that synaptic transmission was still thought to be 'electrical' in nature rather than chemical. It would take another few years for chemical transmission (*Figure 1c*) to be accepted widely. In 1929, Hans Berger, professor of psychiatry and neurology at the University of Jena, discovered electrical rhythms in the human brain, the electroencephalogram (*Figure 1a*). He described synchronous brain waves that persisted





when unperturbed, but any stimulus such as a visual, could bring a range of excitations, breaking this rhythm and creating asynchrony. With this arose the possibility of recording the combined electrical activity of neural populations in the cortex, representing a level of analysis well above that of the single neuron or single synapse. However, the main neurophysiology riddle of the twentieth century remained—how does an excitable neural population orchestrate behaviour?

Orthodox behaviourists rejected any idea about intrinsically driven behaviours outside the realm of stimulus-driven responses, which were being studied in extensive detail. The neo-Pavlovian learning theory (*Figure 2*) assumed a unidirectional journey of information from sensory receptors (because neuronal conduction was unidirectional) such as the eye or ear to the brain, which then relayed outputs to effectors and muscles. Hebb wasn't convinced

Figure 2. Pavlovian Conditioning. (For details see *Box 1*)

Intrinsic neural activity must be as important if, as seems obvious, the behaviour is affected by variables like attention and motive. Hebb claimed that electroencephalic data was a clear sign that the physiological data on which classical behaviourism relied was outdated.

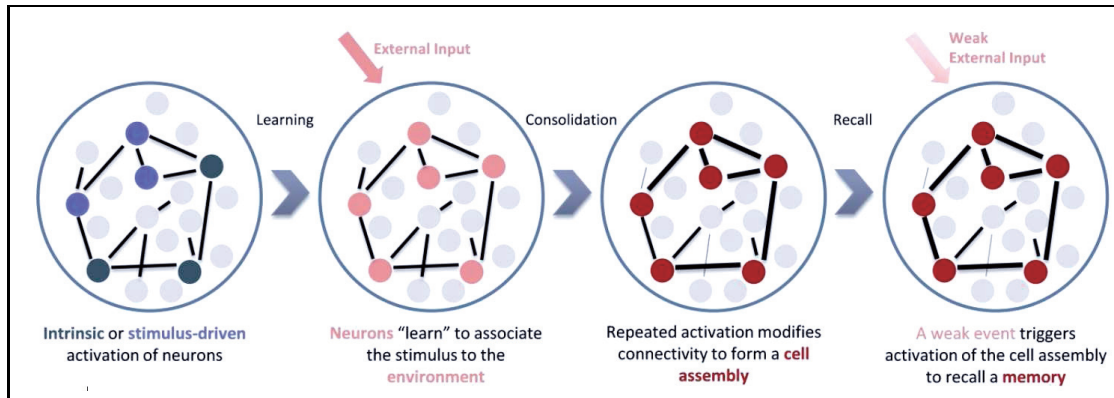
by this unidirectionality or the lack of any modifying factors such as attention or intent and the discovery of Berger's brain waves provided a possible explanation for variables he tried to describe. The intrinsic neural activity must be as important if, as seems obvious, the behaviour is affected by variables like attention and motive. Hebb claimed that electroencephalic data was a clear sign that the physiological data on which classical behaviourism relied was outdated. This was Hebb's justification for adopting a neural theory based on more current neurophysiological data.

The Organization of Behaviour

Hebb was on the search for an activity that could last beyond the presence of the stimulus and be reinstated during thought, which itself was rather autonomous. Hebb's major goal in his book was to speculate on the neurophysiological substrates of the process of thinking (cognition) and its role on incumbent behaviour. It is in this sense that he was truly the forerunner of theoretical cognitive neuroscience.

In February 1944, he came across a reference in *Conditioning and Learning*, to the work of Lorente de Nó, a neurophysiologist at the Rockefeller Institute for Medical Research. Nó's hypothesis of closed chains of neurons consisted of multiple parallel pathways reciprocally connected through 'interneurons' (a term he first introduced), capable of firing well beyond the stimulus, by self excitement of reverberating circuits [5], providing Hebb with the foundation for his postulate. Hebb envisaged that this reverberatory network of closed chain neurons was an 'irregular arrangement of cells' which he called 'cell assemblies'. These cell assemblies might provide a transient, unstable neural 'trace', encoding a sensory event which could be modified into a permanent representation as a result of repeated experience. This process of transformation to permanence would involve structural changes resistant to disruption or erasure—what we now call as 'consolidation'. Hebb's 'dual-trace mechanism' assumes "that the persistence or repetition of a reverberatory activity (or 'trace')





tends to induce lasting cellular changes that add to its stability” creating a permanent structural form of storage. Cell assemblies are, therefore, acquired upon developmental experience, stored in a distributive way in the cerebral cortex, and built from physiological processes described in his neurophysiological postulate. In short, cell assemblies originate from Hebbian synapses (*Figure 3*). Hebb went on to elaborate that multiple cell assemblies may associate to form phase sequences at a higher level of hierarchy that then form the neural basis of higher-order percepts—the brain’s realisation of thoughts.

Hebb’s postulate, therefore, states: “When an axon of cell A is near enough to excite a cell B and repeatedly or persistently takes part 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 [1].”

Hebb extended his postulate to fit his observations to the sparse neurophysiological data of the time. His reliance on the cell assembly in learning implied that visual discrimination was learned, and not innate as Hebb had believed. He viewed visual perception as a constructed mosaic of small features, assembled through associative learning that he inferred from the experimental data of the visual capabilities of chimpanzees reared in the dark. Hebb also argued that similar processes occur during infancy and child development for the acquisition of perceptual capacities.

Figure 3. Hebbian Cell Assemblies. (For details see *Box 1*)

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In his view, any form of learning is an acquired (and constructed) percept, distinct from the initial sensation, but without a unique spatial localisation. He rather argued that learning activated a highly distributed neural network with considerable redundancy. This redundancy, congruent with N6's anatomical descriptions, led Hebb to posit many information relays converging on the same postsynaptic site. Thus the same sensory percept, acquired through previous experience, can now be stimulated, not just by various areas of the visual field but also by only a part of the initial stimulus. Hence, an observer can recognize a previously seen object, now partially hidden from his view.

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The beauty of Hebb's postulate is the universality of its terms; there are no exclusions or constraints. "It is, itself, memorable" [5]. In this context, it is worth mentioning psychologist Jerzy Konorski, who first described classical and instrumental conditioning. Konorski's hypothesis in explaining plasticity was conceptually close to that of Hebb. Nonetheless, Hebb succeeded in presenting his postulate in a precise, proverbial fashion, as the foundation of his more elaborate theoretical scheme. The neurophysiological postulate is a general rule (the 'Hebbian learning rule') for defining the conditions leading to the establishment and maintenance of neural connections. He rejected the notion of a strong localization-of-function, thus not completely rejecting cortical equipotentiality, but still allowing for use-dependent changes in synaptic efficacy through structural modifications. More importantly, he was able to validate his view that intelligence was an acquired developmental trait involving the slow maturing of cortical cell-assemblies and phase sequences through learning processes [6]. Later, Milner updated Hebb's cell-assembly construct, including inhibitory neurons, to make the cell/assemblies physiologically feasible.

Nevertheless, the major difficulty with Hebb's hypotheses was that it was impossible to experimentally test them with the available tools of the time, and until much later. Even though it was possible to study synaptic modifications, it was impossible to isolate cell assemblies to study their mechanistic roles. At a time



when neurophysiologists were still struggling with the fundamentals of synaptic transmission, and even synaptic plasticity was ahead of its time, cell assemblies and phase sequences were to all effect, completely abstract. The sheer neuroanatomical complexity of the cerebral cortex, including the notion of random interconnectedness, had deterred experimental physiologists away to simpler systems and more basic issues of neural plasticity in development and learning. Seventy years since Hebb's monograph, neuroscience has come a long way; remarkably, however, the Hebbian synapse and Hebbian learning rule attract far more interest than they ever did in Hebb's own time.

3. The Legacy of Hebb

Today, the Hebbian synapse and Hebb's neuropsychological postulate has attained the status of dogma in neuroscience, with unaccountable references made to it by studies spanning the entire breadth of the neuroscientific investigation. The postulate was the first to place the synapse at the centre of psychological theory, as the principal site of modification in encoding the experiences of learning and memory, development and ageing.

"Almost 50 years later, much of Hebb's neurophysiology is also out of date, but the part it played in extricating North American psychology from its self-imposed straitjacket is history" [7]. In retrospect, unexpectedly, Hebb's avant-garde approach to a neuropsychological theory of behaviour did not create an immense stir in the field of psychology but he provided a crisp scientific solution to one of the most difficult problems psychological research was facing. Following Hebb's publication, the latter half of the 20th century saw a sudden surge of cognitive psychology research that can at least in significant part be credited to Hebb.

Remarkably, Hebb's novel treatment of the brain as a thinking machine was published coincidentally with the arrival of the first computers. Hebb's cell assembly, therefore, almost immediately became the model for computer simulations of thinking and visual perception. It was only some years later when neuroscientists

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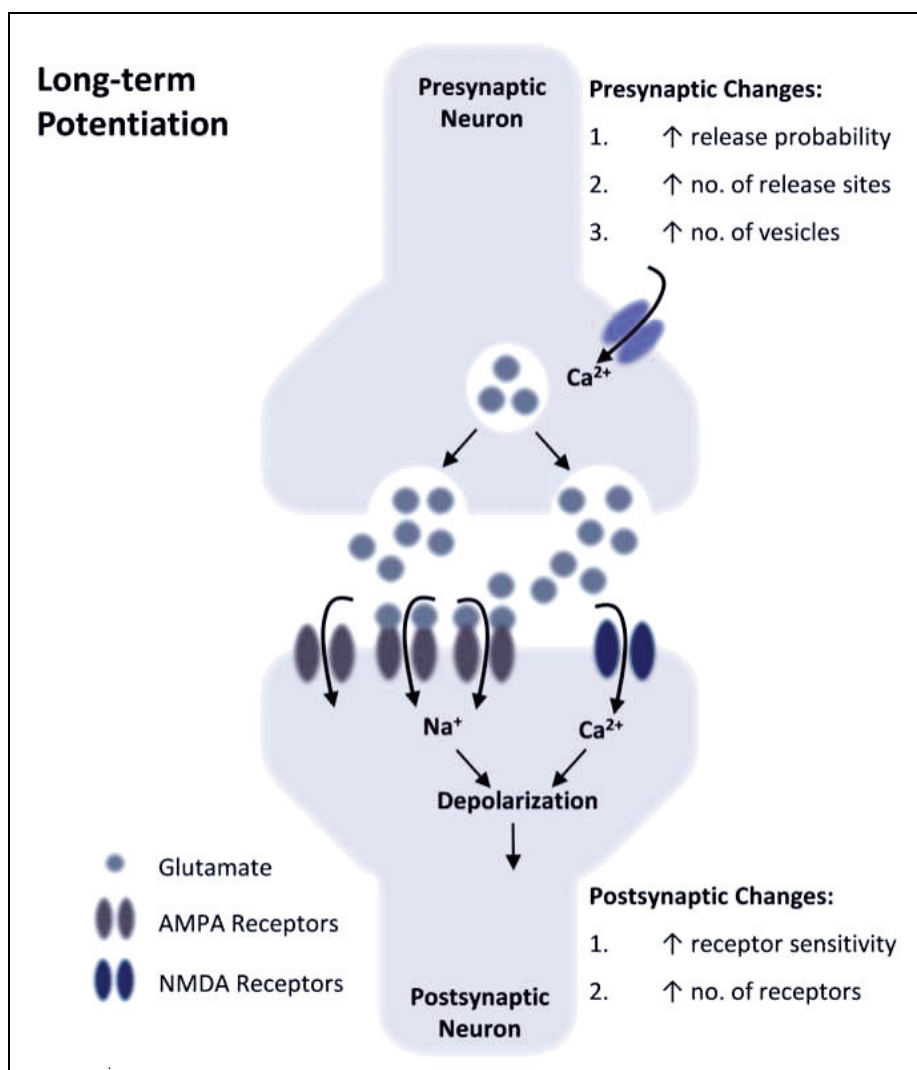
began to make some progress towards understanding learning-induced neural changes that they began to rely heavily on Hebb's postulate, referring to synapses having the required learning characteristics as 'Hebbian synapses'.

In 1973, Bliss and Lomo reported long-term potentiation (LTP) in the rabbit hippocampus (*Figure 4*). A single tetanic stimulation of the perforant pathway resulted in a very long-lasting enhancement of the response to a test stimulus, well beyond that expected for post-tetanic potentiation. LTP became the key to understanding memory, and finally, a practical occurrence of Hebbian plasticity was realized. Another study showed that whereas high-frequency bursts produced potentiation, as expected, low-frequency bursts actually induced depression (LTD, long-term depression) and other studies demonstrated the persistence of LTP with the persistence of memory in a circular arena task. Together, these reports became the building blocks for linking hippocampal LTP with memory processes, and soon, glutamatergic action at NMDA receptors was revealed.

In the light of the Hebbian learning rule, Magee and Johnson's discovery on rat hippocampal slices is seminal in showing the importance of back-propagating action potentials (bpAP) in LTP. Subthreshold excitatory post-synaptic potentials (EPSPs, a measure of presynaptic activity), paired with bpAPs (a measure of post-synaptic activity), brings a precisely timed associative signal to the dendrites. This amplifies dendritic APs and calcium influx, and induces the LTP, and may thereby contribute to Hebbian enhancement of synaptic strength. The discovery of bpAPs as the retrograde signal that permeates the dendritic tree, brought back the dendrite as an active component of the neural processing component, stirring up new avenues in dendritic integration research.

Another pathway of investigation led by Hebbian views is the mechanism of synaptic homeostasis and stability. Hebbian forms of plasticity can achieve relative stability, but this depends upon a balance between LTP and LTD at particular synapses. Here, spike-timing-dependent plasticity (STDP), comes into play, where





the balance struck in long-term synaptic modification depends critically on the relative timing of pre- and post-synaptic events (temporal sensitivity). STDP has the effect of introducing competition, so that some synapses are privileged and are strengthened, whereas others are disadvantaged and weakened.

Furthermore, some recent work has revised the old concept of reverberatory circuits in the neocortex, with a new emphasis placed on the issue of persistence. During memory tasks which incor-

Figure 4. Long-term Potentiation. (For details see Box 1)

porate a short delay period, (now more popularly referred to as “trace-conditioning”), a short-term memory load is imposed, and persisting activity in cerebral and hippocampal neural circuits appear to mediate learning; another instance of the dual-trace mechanism proposed by Hebb. Recent work in developmental cognitive neuroscience, developmental plasticity across the life span, and some topics in neuro-computation are assuredly leaning on Hebbian views to investigate newer avenues of computational research.

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Seventy years after his seminal monograph, Donald O. Hebb and the Hebbian synapse is more relevant than ever, and more strikingly so, to the neuroscientists of the 21st century, than he was to either the physiologists or psychologists of his era. With the opening line of an article, Paul Adams probably paid Hebb his greatest tribute ever by stating that “two of the most influential books in the history of biology are Darwin’s *On the Origin of Species* (1859/1964) and Hebb’s *The Organization of Behaviour* (1949) [8].”

Box 1. Detailed Description of Figure Legends

Figure 1: Neuroscientific Principles Across Scales: (A) The brain is composed of a complex network of neurons that communicate with each other through electrical signals. The spontaneous activity of a population of neurons can be picked up by surface electrodes placed on the scalp, indicating various brain states and functions, like sleep or attention. This is called electroencephalography (EEG). (B) Each neuron (in A), consists of a soma or cell body, its dendritic tree, through which it receives input from other neurons, and its axonal arborizations, through which it communicates with the next neuron. This zone, where the pre-synaptic neuron connects to the post-synaptic one, is called the synapse. (C) Neurons communicate with each other at the synapse by chemical transmission. An action potential at a presynaptic neuron causes synaptic vesicles to dock at the presynaptic membrane and release a neurotransmitter such as glutamate into the synaptic cleft. Released glutamate binds to ion-channels on the post-synaptic membrane, such as AMPA-receptor and causes an inflow of positive cations into the postsynaptic neuron. Thus the neuron that was at a potential of -70mV at rest, gets depolarized to less negative potentials. When sufficient depolarization occurs, the neuron fires an action potential. *Contd.*



Box 1. Contd.

Figure 2: Pavlovian Conditioning: It is a useful tool to study learning and memory mechanisms in the brain. A neutral or conditioned stimulus (CS) is paired with an unconditioned stimulus (US) repeatedly, till it acquires the characteristics of the US. In his experiment, Pavlov used a bell ring as the CS; and his dog was indifferent to it. As the US, Pavlov used food, which caused the dog to salivate. This salivation is an innate unconditional response of the animal, and hence is called the unconditioned response (UR). Now, when the CS and the US were paired repeatedly, the animal learned to associate them over time. The response that was initially produced in response to the US would now also be produced in response to the CS, even if it was presented alone. Pavlov called this the ‘conditioned response’ (CR). Thus, after repeatedly ringing the bell and then providing the food, the dog learnt to associate the sound of the bell with the impending arrival of food. Now, even if only the bell was rung without giving food, the dog would salivate. This conversion of the neutral sound of the bell to a meaningful one for the animal is the essence of Pavlovian conditioning. In the brain, the bell ring excites a different (or partially overlapping) population of neurons than the food. However, when the two are presented together, they might excite a subset of this population simultaneously. With repetition, this network of neurons become ‘wired together’ such that, ringing the bell alone results in activation of the whole network, causing the animal to retain the memory of a previous event and salivate as a response to it.

Figure 3: Hebbian Cell Assemblies: (Modified from Holtmaat and Caroni, 2016) Co-incident activation of different cells in a random network leads to the formation of cell assemblies, that is, groups of repeatedly co-activated cells become wired together by enhanced excitatory synapses. Such activation results in a memory representation or a ‘trace’ and reverberatory (persistent) activity as a result of repetition, causes this cell assembly to modify its connections and become permanent. Thus, a partial stimulus is capable of generating the same cell assembly, resulting in memory recall.

Figure 4: Long-term Potentiation: Synaptic plasticity or the ability of synapses to alter the strength of their connections is the fundamental basis of learning. Long-term potentiation (LTP) occurs when a particular synapse undergoes an increase in synaptic transmission as a result of recent activity such as a high-frequency stimulation, or repeated use. LTP can occur via different pre- or post-synaptic mechanisms. At the presynaptic end, LTP can result in increased release of neurotransmitter into the synaptic cleft by affecting the probability of the release, or the number of release sites or by increasing the number of vesicles recruited. At the post-synaptic end, there might occur increased insertion of receptors into the membrane for increased diffusion of ions, or increased receptor sensitivity. At a typical excitatory synapse, normal transmission recruits only AMPA-receptors that cause ionic flow of sodium. However, another class of glutamate receptors called NMDA receptors are recruited at a synapse that has undergone LTP. The NMDA-R is usually blocked by a magnesium ion. When the neuron is sufficiently depolarized, the magnesium block is released, and the NMDA-R becomes permeable to calcium, depolarizing the neuron further. Thus, via different mechanisms, the same input is now able to cause a larger depolarization, enhancing the strength of the particular synapse.



Suggested Reading

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