ARC Journal of Addiction
Volume 1, Issue 1, 2016, PP 1-2
www.arcjournals.org

Memory, Addiction & Modelling

Karan Bhatt

Dept. of Earth & Environment Science,
KSKV Kachchh University, Bhuj-India

Abstract: *Recent cellular and molecular studies of memory storage suggest that experience dependent modulation of synaptic strength and structure is a fundamental mechanism bywhich the diverse forms of memory are encoded and stored. For memory storage, sometype of synaptic growth is thought to represent the stable cellular change that maintainsthe long-term process. In its most general form, the synaptic plasticity and memoryhypothesis states that activity-dependent synaptic plasticity is induced at appropriatesynapses during memory formation and is both necessary and sufficient for the information storage underlying the type of memory mediated by the brain area in which thatplasticity is observed.*

MEMORY FORMATION

The capacity to form, retain, and use memories is a fundamental property of the brain essential for survival in all organisms. Humans have a rich array of memories associated with emotion, acquired skills and habits, facts about life and addictions. How do we form memories; how are they encoded and stored in the brain? To process and store a lifetime of memories, some form of plasticity in the brain is required. Following Hebb's dual-trace theory [1], it is now believed that memories are encoded as dynamic spatiotemporal patterns of synchronized cellular activity within widespread neural networks and that this dynamic activity progressively results in altered patterns of connectivity among the neurons. Within this framework, any memory representation would correspond with specific sets of patterns of activity in overlapping networks.

As was fully recognized by Hebb, a major problem in the neurobiology of memory is discovering how the activation of neurons in the brain leads to the formation of knowledge and actions. How, that is, do cells collude with brain systems to produce memories that enable changes in behaviour? He proposed that experience-induced changes in neuronal firing could provide a starting point for an explanation. Part of the answer was given by Bliss and Lomo [2] who showed that brief activation of hippocampal cells induced a change in the connectivity of existing synaptic connections with other cells — a finding now well-known as long-term potentiation (LTP). Various forms of LTP and the reverse effect, long-term depression (LTD) have been the subjects of extensive investigations for several decades. The quest of such research is to find synaptic mechanisms mediating the creation of Hebb synapses that may provide cellular bases for memory.

LTP & LTD

Many features of LTP as a phenomenon make it a compelling candidate for the synaptic processes underlying neural information storage. First, LTP is induced rapidly. Soon after its induction, LTP appears within minutes. Hanse and Gustafsson [3] suggested that it develops incrementally, reaching asymptotic levels by approximately 5 to 20 s, depending upon the synapse studied. Another feature is that LTP is associative. If high frequency stimulation of one set of afferents induces LTP, individual active synapses can also be recruited to express LTP — provided that the synapse is coactive. Another feature of LTP is that it is remarkably persistent. LTP in the hippocampal formation can persist from hours to weeks or months, depending upon the stimulation parameters. In intact animals, LTP is decremental and usually decays within 1 to 2 weeks [4].

Other mechanisms that permit either the reversal or the inverse of LTP are likely to be necessary. Such a phenomenon is observed at the same synapses that display LTP and is termed LTD. LTD was noted in early studies, although its possible role in information storage was only suggested by Barrionuevo et al. [5] in the early 1980s. In contrast to LTP, distinct forms of LTD were noted early on in these studies, as evidenced by the distinct mechanisms of their induction. Homosynaptic LTD is used todescribe LTD that follows synaptic activity and typically is induced by repetitive low

frequency (0.5 to 5 Hz) stimulation. LTD also is observed when either synaptic activity or LTP occurs at neighboring synapses. This form of LTD is referred to as heterosynaptic in that it is observed at synapses that are not potentiated. The diversity of types or forms of LTD induction mechanisms may reflect distinct roles for these forms of plasticity in hippocampal function and memory.

ADDICTION & MEMORY

It is not surprising that the evidence accumulated over the last decade demonstrates that drugs of abuse can co-opt synaptic plasticity mechanisms in brain circuits involved in reinforcement and reward processing. Indeed, an influential hypothesis is that addiction represents a pathological, yet powerful, form of learning and memory [6,7]. Although the brain circuitry underlying addiction is complex, it is unequivocal that the mesolimbic dopamine system, consisting of the ventral tegmental area (VTA) and nucleus accumbens (NAc), as well as associated limbic structures, are critical substrates
for the neural adaptations that underlie addiction. It is also clear that the interactions between addictive drugs and synaptic plasticity in different brain regions will contribute to specific aspects of addiction, such as craving, withdrawal and relapse. Moreover, because of advances in our understanding, and the societal importance, of the neurobiology of addiction, this topic
has been the subject of numerous reviews in both the basic science and clinical literatures.

CONCLUSION

Although various studies try to show the mechanisms of addiction, still there is a need for much more studies. This gap between what we know and what we don't know can be rapidly decreased by computational approach in neural addictive biology. Models depending on the current knowledge can be developed and the voids of uncertainties can be filled. Recently, Bhatt and Kumar [8] described how morphine is able to block LTP_{GABA} and induce addiction. Thus, studies in this direction will lead to better and faster understanding of addiction.

REFERENCES

[1] D. O. Hebb, The Organization of Behavior, John Wiley & Sons; New-York, 1949.

[2] T. V. Bliss, T. Lomo, Long-lasting potentiation of synaptic transmission in the dentate area of the anaesthetized rabbit following stimulation of the perforant path, The Journal of physiology 232 (2) (1973) 331–356.

[3] E. Hanse, B. Gustafsson, Onset and stabilization of NMDA receptor-dependent hippocampal long-term potentiation, Neuroscience research 20 (1) (1994) 15–25.

[4] C. A. Barnes, Memory deficits associated with senescence: a neurophysiological and behavioral study in the rat, Journal of comparative and physiological psychology 93 (1) (1979) 74–104.

[5] G. Barrionuevo, F. Schottler, G. Lynch, The effects of repetitive low frequency stimulation on control and "potentiated" synaptic responses in the hippocampus, Life sciences 27 (24) (1980) 2385–2391.

[6] S. E. Hyman, R. C. Malenka, Addiction and the brain: the neurobiology of compulsion and its persistence, Nature reviews. Neuroscience 2 (10) (2001) 695–703.

[7] J. A. Kauer, Learning mechanisms in addiction: synaptic plasticity in the ventral tegmental area as a result of exposure to drugs of abuse, Annual review of physiology 66 (2004) 447–475.

[8] K. Bhatt, A. Kumar, Mechanism of morphine addiction by inhibiting the soluble Guanylate Cyclase-Nitric Oxide (sGC-NO) pathway, Mathematical Biosciences 266 (2015) 85-92.