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Integrated Overview of the Memory System

Ishanee Das Sharma¹, Dr. Somi Patranabis²

¹Student, ²Assistant professor, Amity University, Kolkata

Abstract: *This review aims to clarify and classify memory from psychological and neuroscientific point of view, delving into the molecular mechanisms taking place as well. The main forms of memory are sensory memory, short term memory and long-term memory. We also try to specify the flow of information through various memory models. The concept of synaptic plasticity and long-term potentiation is highlighted, with special focus on the physiological parts of the brain that are involved in memory storage. Overall, this study will help expand our knowledge on the intrinsic details of memory storage and the functioning of our brain.*

Keywords: *sensory memory, short-term memory, long-term memory, memory models, synaptic plasticity, long term potentiation, memory storage.*

I. INTRODUCTION

Our memory plays a major role in making us who we are, by recognizing our associates, recalling past experiences or just by remembering what we have learnt. It is like a string that connects our past to our present. If it is untethered, we become incapable of living our present and are also unable to embrace the future because we remain stuck in the loop of the same memories. Some memories are processed automatically, compared to our personal and more factual memories.

Memory is the workforce of the mind by which information or data is encoded, put away, and recovered when required. It is the maintenance of all the data that we have come across to impact our future actions. If past occasions and memories related to them cannot be recalled then, it would be unimaginable for language, relationships, or individual character to develop and grow. Forgetfulness or unable to recall memories (memory loss) is termed as amnesia. [1][2][3][4][5][6]

The memories stored in our brain are typically accessed in three ways; through **recall**, **recognition**, and **relearning**. Recall is basically reaching into our mind and trying to bring up information. Though the terms are quite self-explanatory, recognition means the power of our brain to identify old facts again and relearning is like refreshing or reinforcing old ideas. But how? So, the basic question that one must ask is that daily we perceive so many things and objects, so how do these become our memory?

Memory is frequently comprehended as an information handling framework with explicit (when we store information consciously) and implicit functioning (all those information on which we do not concentrate but irrespective of that it gets stored in the brain) that is comprised of a sensory processor, short-term (or working) memory, and long-term memory. The sensory processor permits data from the outside world to be detected in the form of chemical and physical stimuli and stores the information according to the varying levels of focus and intent that has been paid while being involved in a certain activity. The main job of the working memory is encoding of the data perceived and its timely retrieval. Data in the form of stimulus is translated in accordance with the implicit and the explicit functions by the working memory. The working memory likewise recovers data from recently stored material. The capacity of long-term memory is to store information permanently through different straight models or systems.

Memory is certifiably not an ideal processor and is influenced by numerous components. The ways by which data is encoded, put away, and recovered can all be corrupted. The measure of attention given to the stimulus perceived, can determine whether the data is encoded for storage or not. Also, the storage capacity can get undermined by physical harm to regions of the mind that are related with memory storage, for example, the hippocampus. [7][8] Finally, the recovery of data from long term memory can be disrupted because of the early decay of that memory. Normal working, memory decay after some time, and unexpected damage to the brain all influence the precision and limit of the storage procedure. [9][10]

II. MEMORY AND ITS SUBDIVISIONS

The Atkinson–Shiffrin "modal model" shapes the establishment of our comprehension of human memory. It comprises of three stores: Sensory Memory (SM), whose visual part is called iconic memory, Short-Term Memory (STM; additionally, called working memory, WM), and Long-Term Memory (LTM). Since its origin, deficiencies of every one of the three segments of the modal model have been distinguished. While the hypotheses of STM and LTM experienced huge alterations to address these weaknesses, models of the iconic memory remained to a great extent unaltered. The fundamental problem of iconic memory model is that, since the stimulus or vision is encoded in retinotopic coordinates, the iconic memory is not able to hold information perceived under normal conditions when the objects or the subjects are in motion with respect to one another.

Thus, 50 years after its plan, it stays an uncertain issue whether and how the principal phase of the modal model serves any valuable capacity and how ensuing phases of the modal model get contributions from the environment. So before discussing about the traditional modal model of human memory and the modern take on it we will study its different components and understand their functioning and individual roles.

A. Sensory Memory

During every second of our existence, our senses are taking in an enormous amount of information of what we see, feel, hear and perceive. While all this information is significant, in no way can we retain all of it. So, our memory creates a quick snapshot of it and helps us focus on the minute and the relevant details only. [12]

Sensory memory holds information for only one second after an item is perceived. The capacity to take a gander at a thing and recall what it resembled with only a moment of perception, or remembrance, is the case of sensory memory. It is out of subjective control and is an automatic reaction. With extremely short introductions, members regularly report that they appear to "see" beyond what they can really report. The primary trials investigating this type of memory were decisively led by George Sperling (1963). [8] Subjects were given a framework of 12 letters, arranged into three lines of four. After a short introduction, subjects were then played either a high, medium or low tone, prompting them which of the lines to report. Considering these report tests, Sperling was able to conclude that the limit of sensory memory was around 12 things, yet that it decayed rapidly (inside a couple hundred milliseconds). Since this type of memory decays so rapidly, members would see the display however not be able to report the entirety of the things (12 in the "entire report" strategy) before they decayed. This kind of memory cannot be prolonged via practice.

Sensory memory can be subdivided into three types: **Iconic memory** also known as visual sensory memory is a quick decaying store of visual data; a sort of sensory memory that briefly stores a picture which has been seen for a little length. **Echoic memory** also known as auditory sensory memory is a quick degrading store of sound-related data, another sort of sensory memory that quickly stores sounds that have been seen for short durations. **Haptic memory** also known as tactile memory, involves a brief memory of touch.

B. Short Term Memory

Short term memory is the capacity to keep a small quantity of data accessible for a brief timeframe. It permits recall for a time of a few seconds to a moment without practice. Its ability is additionally very limited. Short term memory is otherwise called working memory. Working memory is often utilized synonymously with short term memory; it is a relatively recent term as scholars now believe that the two are functionally different and that short-term memory is a part of the working memory. They believe that while short term memory only helps in momentary storage of data for a fleeting second, working memory can help in data manipulation and its retrieval. [12]

C. Working Memory

In 1974 Baddeley and Hitch proposed a "working memory model" that replaced the general idea of short-term memory with a functioning upkeep of data in the short-term storage (Figure 1).

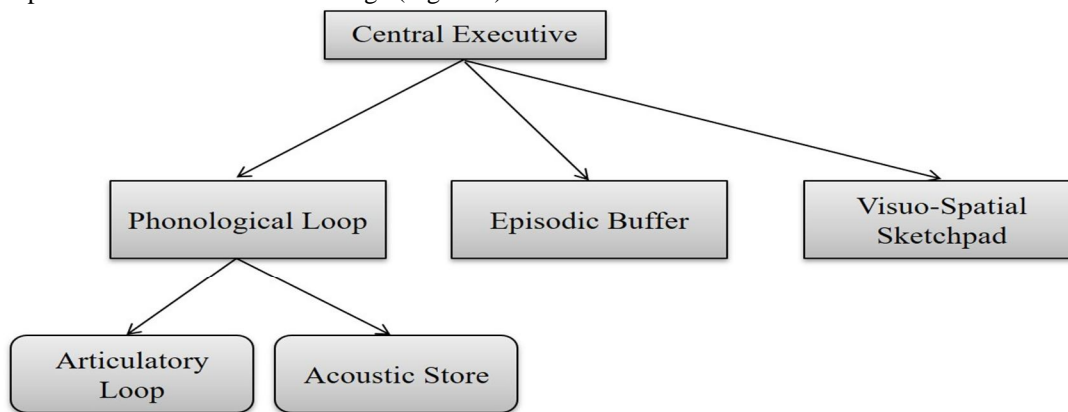


Figure 1. The working memory model

In this model, working memory comprises of three fundamental stores: central executive, phonological loop and visuo-spatial sketchpad. In 2000 this model was extended by adding another component to it known as the multimodal episodic buffer. [13]

The central executive essentially acts as the control unit or the intermediary of information flow. It channels data to the three segment forms: the phonological loop, the visuo-spatial sketchpad, and the episodic buffer.

The phonological loop stores sound-related data by quietly practicing sounds or words in a persistent loop: the articulatory procedures (for instance the reiteration of a phone number again and again). A short list of information is simpler to recall.

The visuospatial sketchpad stores visual and spatial data. It is used while performing spatial assignments, (for example, deciding about distances) or visual ones, (for example, tallying the number of windows on a house or envisioning pictures).

The episodic buffer is devoted to connecting data across spaces to frame coordinated units of visual, spatial, and verbal data and sequential ordering (e.g., the memory of a story or a film scene). It is also said to have established links with the long-term memory.

The working memory model clarifies numerous perceptions, for example, why it is simpler to do two distinct errands (one verbal and one visual) than two comparable undertakings (e.g., two visual). Working memory is likewise the reason for allowing us to do regular exercises including thought. It is the segment of memory where we complete our daily introspection and use them to learn and reason about topics. [12][13]

Working memory is a critical brain function, which is responsible for maintenance and manipulation. The role of sensory cortices (present in the parietal lobe, comprising of four distinct region named as Brodmann's areas) is debated. While the essential theory stresses the importance of these cortices in working memory since damages to these sensory cortices could very well hamper the functioning of the working memory, the non-essential theory stressed that continued activity of the working memory was due to an association of cortices and not singularly because of sensory cortices.

Thus an experiment was performed in APC (anterior piriform cortex), an olfactory sensory cortex, in mice performing several olfactory working memory tasks, and it was found that the APC was important for dynamic information maintenance in olfactory working memory. [14]

From the perspective of neuroscience, it has been observed that working memory apparently leads to the activation of the fronto-parietal brain regions which includes the parietal and prefrontal cortices. Recent studies have also highlighted the involvement of sub cortical regions such as the cerebellum and the midbrain in working memory.

It has been experimentally proved, and is clearly understood that working memory efficiency decreases with age. Hence older participants are expected to perform poorly in comparison to young participants when it comes to a working memory task at hand. In a study, healthy participants with an average age of 70 with no visible neurodegenerative disease (such as dementia or Alzheimer's) were given to carry out a working memory task while their MRI scans were obtained. It was observed that a decrease in cortical surface areas was found to be prevalent in the participants whose performance was low, implying an association of decreased functioning of working memory with loss of brain structural integrity. [15]

Aren't we always told to pay attention while doing any kind of work? Apparently scientifically as well, there is a broad agreement that working memory is closely related to attention. There are two kinds of theories pertaining to attention. The first one conceptualises attention as a resource which leads to a limited capacity of working memory. Three different versions of this idea proposed were: Attention as a resource for processing and storage, a shared resource for memory maintenance and perceptual attention, and a resource used for the control of attention. The other theory considers attention to be a selection mechanism helping in filtering out irrelevant stimuli and asserting a role in cognitive control. [16]

Age is actually not the only factor found to be solely influencing the functioning of the working memory. Many recent studies were made regarding the effect a mental or neurological disorder can have on the working memory of an individual. Having a clear idea now, that working of this system is dependent on the activation of the frontal and parietal regions of the brain in a healthy individual, targeting and studying these regions might help understand and differentiate the differences found in case of a diseased brain, and help observe the deficits at a behavioural level. For instance it was found that individuals with social anxiety disorder were seen to perform poorly in working memory tasks, giving rise to speculation that it was due to a division in cognitive resources resulting in working memory deficits due to not being able to disengage attention from persistent anxiety related thoughts. Similar speculations can be made about people suffering from schizophrenia.

Working memory of patients having suffered traumatic brain injuries were also seen to be similarly affected. Hence decoding the underlying neural mechanism of working memory can help us curate targeted rehabilitation strategies for the ones suffering from related impairments. [15]

D. Long Term Memory

The capacity of sensory memory and short-term memory for the most part has a carefully constrained limit and length, which implies that data isn't retained for an indefinite time. In contrast, long term memory can store bigger amounts of data for conceivably boundless length of time (sometimes for our whole life). Its ability is inconceivable. For instance, given an irregular seven-digit number, one may recollect it for just a couple of moments before overlooking, proposing it was stored in short term memory. Then again, one can recall phone numbers for a long time through repetition; this data is supposed to be put away in the long-term memory (Figure 2). There are two kinds of long-term memory: declarative or explicit memory and non-declarative or implicit memory. [12]

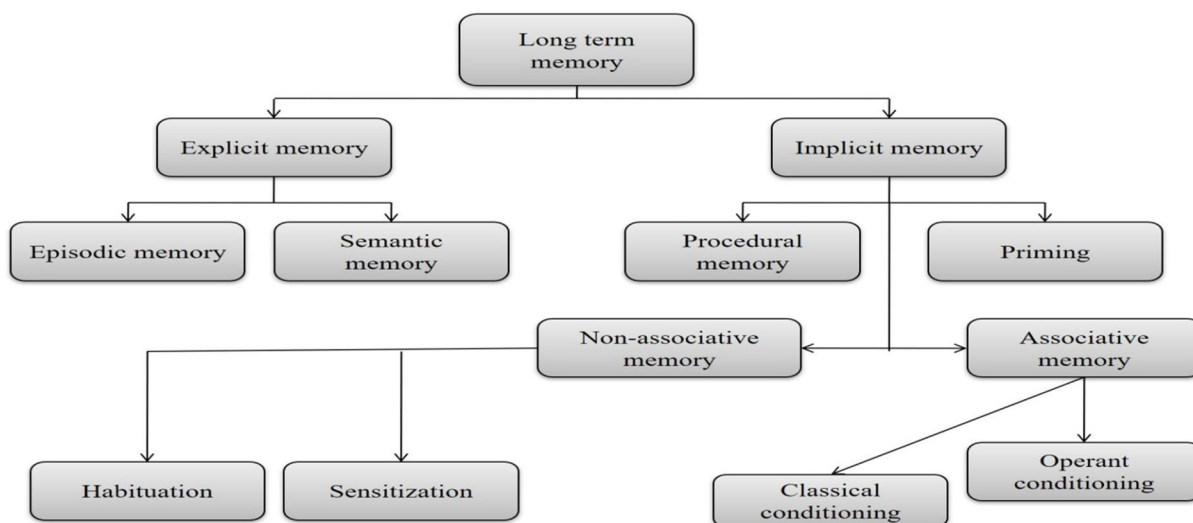


Figure 2. Sub-divisions of the long-term memory

E. Declarative Memory

A historically significant event in the study of declarative memory was done by Tulving in 1972. He proposed two subdivisions of this memory into episodic and semantic memory. While he said that episodic memory consisted of memories of experiences linked to specific times or places, semantic memory was said to store general knowledge of the world. Although there exists, long lasting debates regarding the strong distinction between these two memory types, this study was supported by early studies of amnesic patients being able to learn new semantic memory without having any memory of learning it. Even though this independence has led to many subsequent research works, this perspective is beginning to change with the onset of newer representation models. [17] Explicit memory alludes to data that can be deliberately retained. Therefore, we can tell that are two kinds of declarative memory (explicit memory): one is semantic memory and the other is episodic memory.

F. Semantic Memory

Semantic memory alludes to general world information that we have gathered all through our lives. This general information (realities, thoughts, facts and ideas) is interwoven in experience and reliant on culture. Semantic memory is distinct from episodic memory as in it basically concerns principles and facts irrespective of the context and helps in gaining abstract knowledge. For example, semantic memory may contain data about what a cat is, while episodic memory may contain a memory of petting a specific cat. We can find out about new ideas by applying our insight gained from things in the past. [18] An activity such as reasoning, planning for our future or thinking about the past depends on our usage of semantic memory. [19]

G. Episodic Memory

Episodic memory includes the capacity to learn, store, and recover data about individual encounters and experiences that happen in day to day life. These recollections commonly incorporate data about the time and spot of an event and contain detailed information about the event itself. [20] It is typically the memory of events (for example, times, area topography, related feelings, and other relevant who, what, when, where, why information) that can be invoked. For instance, the memory associated with ones 10th birthday party is an episodic memory. It basically allows us to travel back in the past and relive the moments that have already taken place.

III. PROCEDURAL MEMORY

Procedural (implicit) memories include all those which are unconsciously registered in our brains. It is of four types: including procedural, associative, non-associative, and priming. [12]

- 1) *Procedural Memory*: (or implicit memory) does not depend on the conscious review of data, however on implicit learning. It can best be summed up as recollecting how to accomplish something. Procedural memory is fundamentally utilized in learning motor abilities and ought to be viewed as a subset of implicit memory. It is uncovered when one improves in a given activity due just to reiteration – no new explicit memories are formed, rather the person acts on the basis of what he has learnt from previous experiences. An attribute of procedural memory is that the things recollected are automatically converted into activities, and subsequently at times hard to depict. A few examples include how to ride a bicycle or tying shoelaces.
- 2) *Associative Memory*: Associative memory alludes to the capacity and recovery of data through relationship with other data. The collection of associative memory is done by two processes: classical conditioning and operant conditioning. Let us talk about an example of such associative memory to properly comprehend the concept. Two stimuli if used together can help us develop associations between them. If we repeat a conditioned stimulus before an unconditioned stimulus regularly, then the conditioned stimulus eventually becomes necessary for the unconditioned stimulus. Pavlov's Dog [21] is a reasonable model. The canine produces salivation when it recognizes the presence of food (unconditioned stimulus). If the sound of a bell goes off (conditioned stimulus) at the time of providing the dog with food, then the dog learns to expect food whenever he hears that sound. He learns to associate an unconditioned and conditioned stimulus and hence the production of saliva is the motor response of it to the stimuli. [12]
- 3) *Non-associative Memory*: When we are not pairing an environmental stimulus with any behaviour then the learning is said to be non-associative. Non associative learning is further divided in two procedures habituation and sensitization. While repeated occurrence of the stimulus leads to decrease in efficiency of the motor response in case of habituation, for the same case, sensitization produces an opposite effect and results in an increase in original response to the stimuli. [12]
- 4) *Priming*: It is a phenomenon where exposure to one stimulus determines the response to the subsequent stimulus presented to us, where the second response is almost automatic and made unconsciously. [22] For example the word MEDICINE is recognized more quickly following the word DOCTOR than the word BREAD. Research has not been able to determine the exact duration of priming effects yet.

IV. MEMORY MODELS

Memory models are basically a conceptual representation or a created framework of how the different types of memory that we have learned about so far actually function inside our brain. The very first framework was given by Atkinson-Shiffrin [23] and was named as the **multi store model** or **the modal model** (Figure 3).

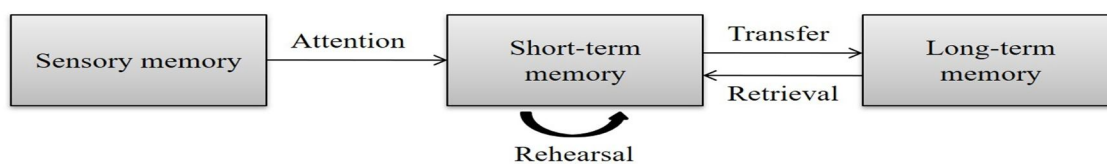


Figure 3. The multi-store model

It suggested that the memory framework was distinctly divided into three segments. The sensory register (buffers), short term compartment and the long-term compartment. The sensory register prevented huge amount of sensory stimulus from overwhelming the cognitive processes while rehearsal of the short-term memory was said to be the primary method for data transfer to long term memory. Even though this model received a lot of criticism it laid the foundation for studying memory structures in the brain. The Craik and Lockhart model suggested that information could be stored easily by analysing it properly, comparing it and adding it with the existing knowledge. It stressed on deep learning for storage of information into the long-term memory. Baddeley's model of working memory helped improve the previous modal model. Instead of integrating short term memory and working memory they expanded the theory of working memory and postulated the presence of three components the phonological loop, the Visuospatial Sketchpad and the episodic buffer.

The three units are said to work harmoniously under the supervision of the central executive. The fourth recognised model is the Serial-parallel independent model by Tulving. This model is an improvement on the previous models as it accounts for two primary systems of memory representation. The first one is called the cognitive representation system which helps in remembering facts, events and life episodes, while the action system stores learning based memory such as dance moves, swimming etc. After looking at these various memory models we use each concept to develop the latest provisional memory model which is the culmination of all these findings and is known as the MNESIS: Memory Neo-Structural Inter-Systemic model proposed by Francis Eustache and Beatrice Desgranges. It has various input pathways and accounts for all sorts of learning that can be automatic or deliberate in response.

A. Physiological Parts Of Brain Involved In Memory Storage

When we talk about storage of memories, what we need to figure out is whether memories are stored in each part of the brain or is it always stored in specifically designated spaces? While finding the evidence for the same Karl Lashley used rats and monkeys to do experiments on them. He taught the rats to run through a maze and find the exit. After the achievement of this task he then used soldering iron to create lesions in the brain of the rats specifically in the cerebral cortex area to try and erase the memory which would help them get out of that maze, but despite the size and shape of his lesions the rats were still able to find their way out. Based on his research Karl gave the equipotentiality hypothesis: if one part of the brain is damaged the other part can take over its memory function. Since his research many scientists have argued and concluded that the main parts of the brain involved in memory storage are Amygdala, the Hippocampus, the Cerebellum, and the Prefrontal Cortex. [24] The amygdala is thought to be involved in emotional memory. [25] Because of its role in the processing of emotional memories the storage process is at a deeper level because the memory is attached to us emotionally. The hippocampus is believed to be involved in spatial learning and declarative learning, although it helps us in forming explicit memories, if we lose it, we can still form implicit memories thanks to our cerebellum. Finally, the prefrontal cortex helps us in retaining semantic memories (Table 1).

PART OF THE BRAIN	FUNCTION IN MEMORY PROCESSING AND STORAGE
HIPPOCAMPUS	Spatial learning and declarative learning. Formation of explicit memories.
CEREBELLUM	Formation of implicit memories.
AMYGDALA	Processing of emotional memories.
PREFRONTAL CORTEX	Storage of Semantic memories.

Table 1. The parts of the brain associated with memory processing and storage

For now, damage to certain brain areas in patients and animal models are the only evidences that are supporting the claims made by the scientists. Further, rather than talking about a single specific area, the actual damage may have happened in the adjacent brain areas or a brain pathway travelling through the area may be responsible for the observed deficit. Learning and memory are usually due to changes in neuronal synapses, thought to be brought out by long-term potentiation and long-term depression. It has been observed in general that the more emotionally charged an event is, more easily it is remembered, this is known as the memory enhancement effect. However, for obvious reasons people with Amygdala damage do not show this effect.

B. Gill Withdrawal Experiment: Discovery Of Involvement Of Synapse In Memory Related Processes

While studying about the mechanism of memory storage, scientists started regarding the process at a molecular level. However, there were two conflicting theories about the same: one was given by Karl Lashley that information was stored due to the aggregate activity of different neurons while Ramon Cajal said learning resulted due to the changes in strength of synapses. Finally, Eric Kandel discovered the central role that synapses play in memory and learning with the help of his **gill withdrawal experiment in Aplysia**. With this experiment he was able to establish the relation between mechanism of memory storage with the involvement of neurons, transmitters, messengers and proteins at the molecular level.

Aplysia has an external respiratory system consisting of the organ gill that is covered by a sheet of skin called the mantle, which finally ends in a fleshy spout called the siphon. When a light stimulus was applied to the siphon, it responded by withdrawing its siphon and gill. Now just like other defensive mechanisms, the gill withdrawal reflex could also be modified through different types of learning including habituation, dishabituation, sensitization, and classical conditioning. [26]

The reason for choosing Aplysia was because it provided them with **three** advantages. While the human brain is made up of trillion numbers of nerve cells Aplysia has only 20,000 and the ones involved in memory formation maybe less than 100 nerve cells. Also, these cells are the largest found in the animal kingdom, hence recording information from these cells became very easy. Finally, these cells could be easily injected with labelled compounds, antibodies; procedures that helped study these cells at a molecular level. [27]

When a small tactile stimulus was applied to the siphon, both the siphon and the gill are withdrawn into the mantle cavity as a reflex for protection. This simple behaviour exhibits three of the most elementary forms of learning familiar in vertebrates: habituation, sensitization, and classical conditioning.

We find that if the siphon of the sea slug is successively provided with a stimulus on its tail then its reaction intensity is decreased to about one third of its initial intensity. This decrease has been attributed to the process of habituation and is said to happen due to a decreased release of the glutamate transmitter in siphon sensory neurons at their synapses with the motor neurons. It has been noticed that the release of neurotransmitters is enhanced with the increase of intracellular concentration of Ca^{2+} . Basically, in this case continuous stimulus leads to decrease in the number of voltage-gated Ca^{2+} channels that open, thus reducing the amount of glutamate neurotransmitter released and decreasing excitation of the post synaptic potential.

To study the process of sensitization a shock was given to the tail region of the Aplysia, it identified it as an aversive stimulus and further enhanced its defensive mechanism. Now if another stimulus would be applied to its tail it would react with a far greater intensity than what it had done previously. Aplysia sensitization is arbitrated by interneurons called **facilitator** neurons, which are subsequently activated by shocks to the head or tail. Stimulation of the facilitator neuron causes the release of more neurotransmitter (glutamate) at its synapse with the motor neuron, thus increasing the magnitude of the gill-withdrawal response. The effect of facilitator-neuron stimulation is mediated by cAMP and a cAMP-dependent protein kinase in the siphon sensory neuron terminal. Short-term sensitization exists if kinase is activated and the concentration of cAMP is elevated, about 1 hour after each sensitizing stimulus.

The memory attributed to sensitisation of the gill withdrawal reflex is graded as a function of training. While a single tail shock is seen to give rise to a short term sensitisation that lasts for about few minutes, a train of shock produces medium level sensitisation that lasts for hours and repeated trains of shocks delivered at fixed intervals can lead to sensitisation that stays for days or even weeks. [26]

While a single shock can give rise to a short-term memory lasting for only few minutes hence it does not include the formation of a protein which technically cannot happen in such a short time frame. In contrast shocks given over a spaced time frame say few days can result in the formation of long-lasting memory which will also require protein synthesis.

The reflex also displays classical conditioning and associative form of learning. Here the actions are carried out in a paired manner. The stimulus is applied to the siphon just few seconds before delivering the tail shock so that the animal learns to make an association between the two stimuli. It has been observed that the enhancement of the withdrawal reflex was observed to be greater and lasted for a longer time in case of classical conditioning when compared to that of only sensitization.

The Byrne lab studied another form of associative learning, which is operant conditioning of feeding behaviour in Aplysia, which occurred due to changes in excitability. [26]

The study concluded interestingly that the mechanism of neuromodulation that was taking place to carry out the operant conditioning was quite similar to those observed in the sensory neurons of Aplysia during classical conditioning of reflexes, displaying the similar mechanistic activities of the two forms of associative learning finally leading to behavioural learning. [26]

Another very important observation made on these Aplysia models was the concept of nociceptive plasticity. Nociceptive plasticity is defined as cellular and behavioural changes produced by the activation of nociceptors, which happens when there is a sensation or perception of pain. Noxious stimuli such as a pinch or shock which was used on the aplysia, generated defensive responses and repetition of the same led to gradual sensitisation. It was seen that such noxious stimuli enhanced the withdrawal responses and the closeness of the following stimulations also lead to different kinds of observations (more dramatic response was observed when the following stimulus was closer to the site of application of the noxious stimulus). The concept was further complicated when several groups discovered that that noxious stimulus could inhibit as well as enhance defensive responses. However Marcus et al. gave the most complete report of such type of plasticity by stating that the inhibition of siphon withdrawal happened following a noxious signal but not when the stimuli applied was innocuous (harmless) in nature. So the study of injury related mechanisms and learning related mechanism in different organisms can help confirm the importance of nociceptive plasticity and its apparent significance in evolution of some forms of memory. [28]

Since the work of Cajal even though it has been largely assumed that learning and memory are thought to involve changes in synaptic connections of the neurons. However in many cases it has been seen that learning involves changes in excitability of the neurons and recent findings suggest that this excitability maybe one of the reasons for the formation of memory engrams (an engram is said to be a unit inside the brain containing cognitive information, theorised to be the means by which memories are stored in the brain, in response to external stimuli.) in vivo. [29]

C. Long Term Potentiation: Molecular Basis Of Memory Storage

It has been years since Donald Hebb published his theory related to the involvement of synapses in the formation of memory. Hebbian theory claims that repeated and persistent stimulation of the post synaptic cell by the pre-synaptic cell leads to a subsequent increase in the synaptic efficiency. This theory was an attempt to explain the phenomenon of synaptic plasticity. Hebb's work foretold some of the greatest discoveries of the following decades, including the discovery of LTP (Long term potentiation) and more recently the existence of memories in synaptically connected neuronal assemblies. This concept is now named as the synaptic plasticity and memory (SPM) hypothesis. [30]

So one of the most dominant finding related to the encoding of memory has been explained by the synaptic plasticity and memory hypothesis in the field of neuroscience.

In neuroscience, long term potentiation (LTP) is a persevering strengthening of synapses dependent on recent levels of activity. These are examples of synaptic action that produce a durable increase in signal transmission between two neurons. [31] Something contrary to LTP is long term depression, which delivers a steady decline in synaptic strength.

LTP has been found to be one of the several phenomena involving synaptic plasticity. As recollections are believed to be encoded by alteration of synaptic strength, [32] LTP is generally viewed as one of the major cell instruments that underlies learning and memory. [31][32]

The process of learning begins from the sensory signals transcribed at the cortex; they are then transferred to the hippocampus where new memories are believed to form. If the signal is strong or repeated, then it leads to the formation of a long-term memory which is transferred back to the cortex for storage. Lesions in the hippocampus takes part in the formation of new memories but do not affect the old ones.

Synaptic plasticity follows the use it or lose it rule: frequently used synapses are strengthened and the ones which are rarely fired, their connections are diminished. New memories are formed when neurons establish new connections or strengthen their synaptic connections. If a memory is no longer needed or rarely recalled, its corresponding synapses will gradually weaken and then disappear. The strength of a synapse is measured by its level of excitability or responsiveness of the post synaptic neuron in response to a given stimulus from the presynaptic neuron. High frequency signals or repeated stimulations can strengthen synaptic connections overtime; this is known as long term potentiation or LTP and is thought to be the cellular basis of memory formation. It is best studied at the glutamate synapse of the hippocampus. When a glutamatergic neuron is stimulated action potentials travels down its axon and triggers the release of glutamate into the synaptic cleft. As per the procedure the released glutamate then binds to the receptors present on the post synaptic neuron. The two main glutamate receptors that often coexist in a synapse are AMPA and NMDA receptors. Following the binding of glutamate to them, these ion channels are activated. When the presynaptic neuron is stimulated by a weak signal only a small amount of glutamate is released, although both receptors are bound by the glutamate only AMPA is activated by weak stimulation. Sodium influx through the AMPA channel leads to a slight depolarization of the post synaptic membrane. The main reason that the NMDA channel remains closed is because its pore remains blocked by magnesium ions.

When the presynaptic neuron is stimulated by a strong or repeating signal, a large amount of glutamate is released into the synaptic cleft. Hence the AMPA receptor stays open for a longer time admitting more sodium ion influx resulting in a greater depolarization. Increased influx of positive ions causes the expulsion of the magnesium ion from the NMDA channel which now gets activated allowing not only sodium but also influx of calcium ion into the post synaptic neuron. The calcium ion is the mediator of LTP induction which has two distinct phases. In the earlier phase (early LTP) calcium initiates signalling pathways that activates several protein kinesis (like PKC and CaMKII). These proteins enhance synaptic connections in two ways: they phosphorylate the existing AMPA receptors thereby increasing AMPA conductance of sodium and help to bring more AMPA receptors from the intracellular store to the surface of the post synaptic membrane. This phase is thought to be the reason for the formation of short-term memory which lasts for a limited time period. In the late phase of LTP new proteins are made and gene expression is activated to further enhance the connection between the two neurons. These include newly produced AMPA receptors and expression of other proteins that influence the growth of new dendritic spines and consolidation of synaptic connections. The late phase may correlate with the formation of long-term memory.

We can finally conclude that the increase and decrease in the both pre and post- synaptic cells can accompany most elementary forms of learning. This learning induced synaptic growth represents the most final and stable stage of long-term memory storage bringing up a possibility that long-term memory can be stored fully or maybe partially, depending on the strength and stability of the synaptic structure.

D. Synaptic Plasticity And Memory Hypothesis

Several lines of evidence have been proposed by Martin et al. to confirm the role of synaptic plasticity as a mechanism involved in memory storage. These include: detectability (in which learning should result in perceivable changes in synaptic weight), mimicry (in which inducing those similar kinds of synaptic weight changes in the animal should create the same memory), anterograde intervention (where prevention in synaptic weight changes should halt learning process) and finally retrograde intervention (where any interference with the synaptic weight changes should erase the memory previously stored. From these various approaches and culmination of isolated evidences there had arisen support for this hypothesis.

By far the most crucial evidence comes from anterograde intervention approaches using plasticity induction or genetic manipulations used to block both learning and LTP. In retrospect there is very little evidence of mimicry available, the nearest attempt till date being the reappearance of auditory fear upon the reinstatement of long term potentiation of certain auditory inputs to the amygdala. Studies have shown very specific LTP occurs in the amygdala during fear conditioning, and depotentiating the active synapses subsequently lead to memory loss. Also when two distinct memories share overlapping neuronal assemblies, potentiation or depotentiation of the synapses encoding for one of those memories has an effect on only, that one selective memory without harming the recall and storage of the other one. Together all these finding form a strong supporting basis for the SPM hypothesis.

While this hypothesis still dominates over the neuroscience behind memory storage there are still experimental ideas against the fact that this hypothesis leads to the long term memory storage which gave rise to an alternative model called the cell-intrinsic storage of memory. The most likely candidate for this kind of cell intrinsic storage was proposed by Holliday, who said that the phenomenon of DNA methylation might be a reason for learning and memory storage. [30]

E. Spontaneous Thought As An Unconstrained Memory Process

Having thoughts and introspecting is something that comes very naturally since thinking and being able to have opinions, is what sets us humans apart. The spontaneity of thoughts is random and takes place without much guidance or cognitive control but it has been found that the formation of spontaneous thoughts shares many commonalities with the memory processes we have come across. William James first described this continuous and unstoppable stream of thoughts nearly a century ago and nowadays scientists name this phenomenon as “spontaneous thoughts” which follow our mental whimsy whenever we are not paying much attention on our goals, tasks or outside stimuli. It has been seen that spontaneous thoughts derives its content from our memory stores and arises from the same neural establishments involved in memory storage and searching. Researcher’s state that maximum component of our thoughts is occupied by episodic memories. During periods of rest, people tend to revisit recent experiences a phenomenon known as **offline memory replay** and researchers state that 60% of these wandering thoughts are episodic in nature. While episodic memory acts as a fodder to such thoughts, semantic memory extend structure and meaning to them, resulting in recombination of past occurrences, scaffolded by semantic representations to create novel situations of the past and future scenarios.

How do our memory systems help in this kind of imagination? We have already learned how our hippocampus and prefrontal cortex help in significant memory storage and processing. The hippocampus is also seen to be the source of “offline” memory replay. For instance in a rodent study, after a rodent explores a new maze or a new track, neurons in the hippocampus that encoded spatial locations were seen to spontaneously reactivate during rest and replay the rodent’s newly learned experience. It can be clearly understood that dreams are the least constrained form of spontaneous thoughts since at that time two key processes i.e. **attention** and **cognitive control** are switched off. These links between our memory and thoughts have helped computer scientists to develop artificial intelligence that imitate the dynamics of daydreaming, opening a wide gate into the future prospect of this study. [33]

Our memory not only impacts our thoughts but also our conscious being. William James’s work on memory cannot be discredited. James linked our conscious memory to both short and long term memory. He asserted that our consciousness of now is impacted by our past experiences and expectations of our future and that, Atkinson and Shiffrin’s modal model is a modern and extended take on his views. [34]

V. CONCLUSION

“Memories are like small islands in the vast ocean of forgetfulness”. The quote tells us how our brain while taking in an outstanding load of information from the outside world can retain only a part of it. While most of the insignificant information merges into the ocean, only the ones which are consciously or unconsciously taken in, followed by frequent rehearsals are converted into islands. We learnt about the different types of memory and their subdivisions which were followed by research of different memory models. It’s been over fifty years since the memory model was initially developed by Atkinson and Shiffrin in 1968 and since then this model has undergone several modifications finally resulting into an integrated version.

While these models helped us understand the direction of information flow, with the help of the latest findings in the field of neuroscience we were able to delve deeper and address exactly how information is encoded within the brain cells. The molecular approach to memory storage first came into attention because of the work of Eric Kandel and his gill withdrawal experiment in *Aplysia*. It showed us how the theoretical knowledge of memory such as the process of habituation and sensitization worked through the release of neurotransmitters such as glutamate and serotonin in between the neural synapses at a molecular level. Finally, we learnt about long term potentiation which is a concept in cognitive neuroscience telling us exactly how the variation of synaptic strength can determine whether a memory is remembered or gradually forgotten. While we have established that synaptic plasticity is one of major reasons for the storage of memory it is important to note that a single change in a particular synapse is not enough for the storage of a complex memory, in that case an ensemble of neurons and their synapses need to produce a specific alteration in the information flow in a given neural circuit. With the development in the genetic tools with time one day man may be able to modify specific neural circuits and hence change stored memories.

But given the huge amounts of information being absorbed by our brain every day, what mechanism residing in our brain helps segregate the new information from the old ones we may ask, instead of getting muddled up or over written? A paper recently published in **Nature Neuroscience** gave quite an interesting rendition to this question. They finally explained how the brain’s protective buffer works. The researchers showed in order to distinguish between already existing and current stimuli, the brain rotated the sensory information to encode it as a memory. In order to explain this orthogonal dimension to memory storage, Buschman compared the situation to taking notes in a hurry in a limited space. Apparently what the brain does is, it starts writing the memory on a piece of paper and once it runs out of space, just like we would, it rotates the paper by 90 degrees and starts writing on the margins. This mechanism basically helps protect and separate information stored in our brain. These daily new researches coming up indicate the sheer vastness of the unknown territories when it comes to our mind and how it operates, which keeps encouraging us to keep delving into its depths.

A research study found that we can increase cognitive function and brain efficiency through simple lifestyle changes such as taking a **good nap** (because it is believed that a good sleep helps in memory reconsolidation), **memory exercises**, **healthy eating** and **leading a stress-free life**.

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