The science of memory: A comprehensive analysis of declarative and non-declarative memory mechanisms and updates (Review)

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Abstract. Memory is a vital cognitive process of the acquisition, storage and retrieval of sensory information, which is essential for understanding and learning. It is classified as sensory, short-term and long-term memory. A particular type of memory may be associated with different areas of the brain, such as navigational memory, which may be regulated by the basal ganglia, cerebellum, hippocampus, entorhinal cortex, as well as associated parahippocampal and retrosplenial cortices. Emotional learning is associated with the auditory cortex and amygdala. Memory is also regulated by epigenetic mechanisms; for example, hypomethylation enhance memory by enhancing the expression of memory-associated genes. The present review provides a comprehensive overview of memory mechanisms and updates, as well as the molecular mechanisms involving the procedure of memory formation. The molecular mechanisms involve long-term potentiation and the cyclic AMP signalling pathway. The role of nitric oxide in memory formation is also discussed. The present review also expands to the cellular level, exploring how dopamine neurons orchestrate the balance of memory formation and forgetting. Neurogenesis is associated with the formation of new memory. The hippocampus is associated with learning and memory formation. The dentate gyrus is a unique area of the hippocampus that can generate new neurons throughout adulthood, a process known as adult neurogenesis. Memory modulation is a high-priority research topic for future research, as modern research reveals that memory can be modulated by specific receptor agonists, such as glutamate receptor agonists and deep brain stimulation techniques. However, the exact mechanisms involved need to be further elucidate and validated by clinical evidence in human studies.

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1. Introduction

Memory can be defined as the acquisition, storage and retrieval of sensory information, and the ability to recall past events at the conscious and unconscious levels. Recent advancements in both molecular neurobiology and information technology are prompting a re-evaluation of how memory is defined (1). There are three stages of memory processing: Encoding, storage and retrieval. Encoding denotes the transformation

of sensory information into neural code. The second stage of memory processing is storage or consolidation, which is the process of retaining encoded information over time. and the third is retrieval, the process of accessing and recalling stored information and bringing it back into conscious awareness.

Memory can be influenced by different factors that affect the three stages of memory (encoding, storage, and retrieval). Pain can impair memory function; both animal and human studies have indicated that physical discomfort can negatively affect the memory process (2,3). Pain selectively hinders the consolidation of object recognition memory (a type of declarative memory), without affecting its initial acquisition or later retrieval (4). Memory encoding is highly dependent on attention. In the event that an individual distracted or does not allow sufficient focus to new stimuli, the amount of information successfully converted for storage will be diminished (5). The capacity to store memories can be compromised by physical damage to a specific area of the brain. For instance, trauma to the hippocampus, a structure critical for memory consolidation, can corrupt the storage process (6). Even after information is stored, the ability to retrieve it from long-term memory can be disrupted. This can occur due to normal decay over time, where the neural traces weaken, making the memory difficult or impossible to access (7).

The crucial difference between short- and long-term memory is that long-term memory requires the synthesis of new proteins for its stabilization. A critical newly synthesized protein in the process of long-term potentiation (LTP) is protein kinase C (PKC) ζ type (PKMζ), an autonomously active variant of the enzyme PKC (8). PKMζ is essential for sustaining activity-dependent strengthening of synaptic connections. Inhibiting PKMζ effectively erases established long-term memory without harming short-term memory.

Furthermore, once the inhibitor is removed, the ability to form new long-term memory is completely restored. Another crucial molecule is brain-derived neurotrophic factor (BDNF), which is crucial for the persistence of long-term memories. The enduring stabilization of synaptic modifications requires a simultaneous increase in both pre-and postsynaptic structures, specifically the axonal bouton, dendritic spine and postsynaptic density. This structural growth is supported at the molecular level by an increase in postsynaptic scaffolding proteins. For example, higher levels of PSD-95 and HOMER 1c have been shown to be associated with the stabilization of synaptic enlargement (9).

2. Role of epigenetics in memory

Studies on rats show that learning triggers DNA hypermethylation in the hippocampus. Although the hippocampus serves as the initial storage site for these memories, they are eventually relocated to the anterior cingulate cortex (10,11). During memory formation, in the hippocampus, the activity of the DNA methyltransferase enzyme increases. This leads to the hypermethylation of genes that inhibit memory, effectively turning them off. Simultaneously, genes that promote memory often undergo hypomethylation (removal of methyl groups), keeping them active (12).

DNA methylation at specific base pairs affects synaptic plasticity by altering gene expression and splicing patterns. DNA methylation can influence memory by altering synaptic circuits (13,14). The epigenetic regulation silences memory repressors and promotes memory enhancers. Eventually, this balance contributes to the formation, consolidation and long-term storage of memories by stabilizing synaptic plasticity.

3. Balance between memory formation and forgetting: How dopamine neurons orchestrate memory

For effective adaptation in the environment, animals need a balance between neurogenesis and the vanishing of old memories. Dopaminergic neurons may be responsible for both the formation of new memory and the forgetting of unconsolidated memory by different signalling pathways. Research on *Drosophila* has revealed that cyclic AMP (cAMP) signalling plays a crucial role in creating memories, while cyclic guanine monophosphate (cGMP) signalling actively triggers the forgetting of unsolidified memories through these dopaminergic neurons (15).

Through genetic screening and proteomic analysis, researchers have discovered that neuronal activation leads to the formation of a complex that includes Kdm4B, a histone H3K9 demethylase, and Bur, a GMP synthetase (15). This Kdm4B/Bur complex is crucial and sufficient for the forgetting of unconsolidated memories. Nitric oxide (NO)-dependent cGMP signalling activates the Kdm4B/Bur complex through phosphorylation via dopamine neurons. This activation is essential for gene expression of *kek2*, which codes for a presynaptic protein. Consequently, activating Kdm4B/Bur brings about changes at the presynapse (15).

The present review discusses the association between cGMP signalling and synaptic changes, mediated by gene expression, in the process of forgetting. It suggests that the opposing functions of memory formation and forgetting are achieved by different signalling pathways via dopamine neurons, ultimately influencing synaptic integrity and balancing adaptive animal behaviour.

Bidirectional memory regulation and dopamine neuron signalling. Animal behaviour is determined by memories stored in the brain; however, survival depends on the ability to forget negative memories. Since environments are always changing, and in a changing environment, an efficient system is essential to weaken older memories, as it provides the way for more adaptive, updated behaviours (16,17). At the same time, the brain must also solidify key memories through a process known as consolidation, often involving gene expression (18).

The active process of memory decay, or 'forgetting', has been extensively studied at the molecular level in *Drosophila*. A classic example is observed in the olfactory aversive training paradigm, where a specific odour is paired with an electric shock (19). This creates an associative memory within the olfactory memory centre of the fruit fly, known as the mushroom body (MB) neurons (20,21). This memory formation is facilitated by the simultaneous activation of dopamine neurons (22,23).

Notably, this olfactory aversive memory in fruit flies rapidly fades within ~3 h. This rapid forgetting is actively driven by

specific molecular players: Rac1, a small G protein belonging to the Rho GTPase family, and Raf, an upstream kinase of mitogen-activated protein kinase (24,25). While the blocking of this Rac1-mediated forgetting pathway can extend memory duration, the memory still eventually fades. This raises the question of whether memory simply fades away over time, or whether are there additional active processes contributing to its decay.

The processes by which memories are formed must inherently interact with the mechanisms that cause them to fade. A fundamental principle in learning and memory involves dopamine and its G-protein-coupled receptors activating adenylate cyclase (AC), which in turn elevates the levels of cAMP. This pathway is a common thread in memory creation (20,26).

At the synaptic level in *Drosophila*, the connections between MB neurons and their downstream partners, the MB output neurons, experience synaptic depression (27,28). However, a key question remains: Namely, whether this synaptic plasticity within the MBs is linked to memory decay. Specifically, it remains still unclear how synaptic depression may be reversed or restored by either active forgetting mechanisms or any other processes that lead to memory decay.

Traditionally, researchers linked gene expression mainly to the strengthening of memories, a process known as consolidation. However, research reveals a direct connection between gene expression and the weakening of memories through synaptic plasticity (29). Furthermore, this memory decay occurs independently of the Rac1 or Raf pathways (30). Since this particular decay occurs after Rac1-dependent forgetting and uses different mechanisms, it is termed 'gene expression-based forgetting'. This type of forgetting is driven by cyclic guanine monophosphate (cGMP) signalling within the MB neurons, which receives a boost from dopamine neurons. The results in altered gene expression in the MB neurons and changes at the presynapse (15).

The specific role of cGMP signalling via dopaminergic neurons helps bridge a crucial gap in the understanding of how memories are both formed and forgotten.

4. Classification of memory

Memory can be classified as sensory memory, short-term memory or working memory, and long-term memory according to the multistore (or modal) model proposed by Atkinson and Shiffrin in 1968 (31). As regards sensory memory, in cognitive psychology, sensory memory is defined as the primary stage of the memory process. It functions as a modality-specific buffer that preserves a high-fidelity, isomorphic (unprocessed) representation of environmental stimuli. It essentially acts as a highly detailed, fleeting snapshot of the environment that allows the brain a fraction of a second to process incoming sensations before they are lost or moved to short-term memory (32). Its range may from milliseconds to 4 sec. Sensory memory can be classified as iconic memory (vision). echoic memory (sound), haptic memory (touch), olfactory memory (smell) and gustatory memory (taste). Short-term memory and long-term memory are discussed in more detail in the sections below.

5. Short-term memory

Short-term memory, also known as primary or active memory, refers to the cognitive system responsible for holding a limited amount of information for a brief period, generally up to 30 sec (33). This contrasts with long-term memory, which can store an unlimited amount of information indefinitely. The distinction between these two memory systems lies not only in their duration, but also in their distinct functions. Despite their differences, short-term memory and long-term memory are closely interconnected.

Short-term memory refers to the active retention of information for a brief duration, without any manipulation of that information. When information is actively maintained and manipulated, it is typically categorized as working memory, as defined by Baddeley (34).

Research into short-term memory commonly employs tasks where participants are presented with a series of items and subsequently asked to either recall or recognize the information they just encountered. This raises fundamental questions regarding its operation: These questions pertain to how short-term memory functions, and whether there are distinct processes and storage mechanisms for different types of information, or for information received through various sensory modalities, such as visual or auditory input.

Investigating short-term memory across modalities and materials. Much of the research on short-term memory has focused on how verbal or visuospatial stimuli are processed, with less attention paid to other types of information. Similarly, the impact of sensory modality on short-term memory has been primarily explored within the verbal domain.

The study by Talamini *et al* (35) aimed to bridge existing gaps by comparing visual and auditory short-term memory across diverse material types. Specifically, it investigated whether sensory modality and the nature of the stimuli interact to influence memory performance. Talamini *et al* (35) examined whether musical expertise can modulate memory performance. As previous research has demonstrated, musicians often have superior auditory memory and visual memory to a certain extent, compared to non-musicians (35). To test this hypothesis, the researchers employed a consistent recognition paradigm across various stimulus types, and in every trial, the researcher presented two sequences of events separated by a silent delay before the participants (musicians and non-musicians), and they were asked to identify whether both sequences were identical or different. They compared performance for auditory and visual materials, which were categorized into three distinct groups: i) First, a verbal group, which included syllables; ii) a second non-verbal with contour group (these were stimuli that could not be easily named but possessed a distinct contour (patterns of rising and falling variations in loudness for auditory stimuli or luminance for visual stimuli); and iii) a third non-verbal without contour group; this category included pink noise sequences for auditory stimuli and Kanji letter sequences for visual stimuli, characterised by the absence of an obvious contour or pattern of up-and-down changes based on non-pitch features (35). The results revealed a significant advantage for musicians, mainly with auditory non-contour stimuli and contour stimuli (visual or auditory).

The findings of that study suggest that musical training can enhance short-term memory associated with musical skills. It was also concluded that the encoding strategies play a crucial role in short-term memory performance (35).

Investigating short-term memory mechanisms. Short-term memory is a fascinating, complex cognitive function that involves intricate processes of perceiving information, forming memories, and then using that stored information. Despite its critical role in daily life, the precise mechanism by which short-term memories are formed, retained and retrieved remain unclear.

To shed light on these mechanisms, a previous study examined 41 healthy college students who participated in a challenging memory task (36). The researchers used functional near-infrared spectroscopy (fNIRS) to monitor real-time changes in haemoglobin concentrations within specific areas of the cortex in the brain. Alongside this, the facial expressions and vital signs of the participants throughout the memory tests were continuously recorded (36). The results revealed increased activity in several key brain regions during the memory tasks: The inferior prefrontal gyrus, the visual association cortex, the pre-motor cortex and the supplementary motor cortex. They observed a significant increase in functional connectivity between these regions during task engagement, with differences in brain activity among participants becoming less pronounced over time (36). Of note, participants who demonstrated superior short-term memory performance exhibited fewer negative emotional expressions and had higher heart rates compared to those with weaker memory (36). These findings strongly suggest that the robust interconnectivity within the cortex of the brain and sufficient cerebral blood oxygenation are crucial factors in boosting short-term memory capacity.

The influence of proprioceptive short-term memory on passive motor learning. When a physical trainer guides the limbs of an individual to teach a specific movement, they are providing proprioceptive information, the sense of body position and movement. For the learner to perform the same type of movement, they have to accurately perceive this information and store it for later performance. This indicates two key abilities: Proprioceptive acuity (the ability to accurately sense movement) and short-term memory (the ability to hold that perceived information for a short time). These two functions are required for active motor learning (where the learner performs the movement themselves). The role of these two functions is minimal in passive motor learning (where the movement is physically guided).

To investigate this, a previous study experimented on 21 healthy adults (aged an average of 25 years). That study was performed to determine whether an the efficiency of an individual in passively guided learning is linked to their proprioceptive acuity and short-term memory capacity (37). That study revealed a significant positive significance about learning efficiency. Learning efficiency was strongly associated with short-term memory capacity. Specifically, individuals who had good recall capacity of older sensory stimuli demonstrated superior learning efficiency. However, the researchers found no significant association between learning efficiency and proprioceptive acuity. Using a causal graph model, they observed that memory directly influenced learning, while proprioceptive acuity had an indirect effect on learning, operating through memory. These findings underscore the critical importance of a learner's short-term memory for effective passive motor learning (37).

6. Long-term memory

Long-term memory involves the capacity of the brain to store information for extended periods, ranging from days to a lifetime. It is the vast mental archive that allows the recalling of past events, facts, skills and experiences. John Robert Anderson, Professor and Psychologist at Carnegie Mellon University, Pittsburgh, PA, USA, classified long term memory into two parts: One is declarative (explicit), and the other is non-declarative memory (implicit) (38). Declarative memory can be divided into two parts: The first is semantic memory, and the second is episodic memory, while non-declarative memory can be classified into several types, such as procedural memory, emotional memory, classical conditioning, priming and non-associated memory, as illustrated in Fig. 1.

Certain regions of the brain encode different types of memories. The hippocampus is the most critical region of the brain responsible for declarative memories, semantic facts and events remembrance is coded by the hippocampus (39). The basal ganglia are responsible for spatial navigation memory (direction). Procedural learning, including motor skills, is associated with the basal ganglia, such as cycling, driving and learning a new language (40). The limbic system, including the amygdala encodes emotional memories, such as weddings, birthdays, winning an award ceremony and old school days. Laughing, crying and weeping are associated with the limbic system and are encoded by the amygdala (41). Short-term memory is encoded by the cortex region, the prefrontal cortex, the neocortex, sensory and motor cortex. Short-term memories are often termed working memories (33,34). The various parts of the brain responsible for different types of memories are depicted in Fig. 2.

Long-term memory formation is a multi-stage journey, transforming fleeting sensory experiences into lasting recollections. It begins with encoding, the initial conversion of what is seen, heard or felt into a neural language the brain can understand and store. Then this newly encoded, fragile memories stabilize, a process known as consolidation (42). This involves the following: i) Cellular consolidation, which occurs rapidly, within minutes to hours after learning, involves structural changes at individual synapses (43). ii) System-level consolidation, which is a much slower process, taking over days, weeks, months, or even years (44,45). System-level consolidation occurs across different brain regions. In this consolidation, memories gradually move from the hippocampus to the neocortex (46). Sleep plays a critical role in the consolidation process, as the hippocampus reiterates recent events, helping to strengthen and update these cortical connections (47). Once memories are consolidated, they become stored across various brain regions. Finally, retrieval is the act of accessing and recalling this stored information whenever it's needed. Declarative memory (semantic and episodic memory) and

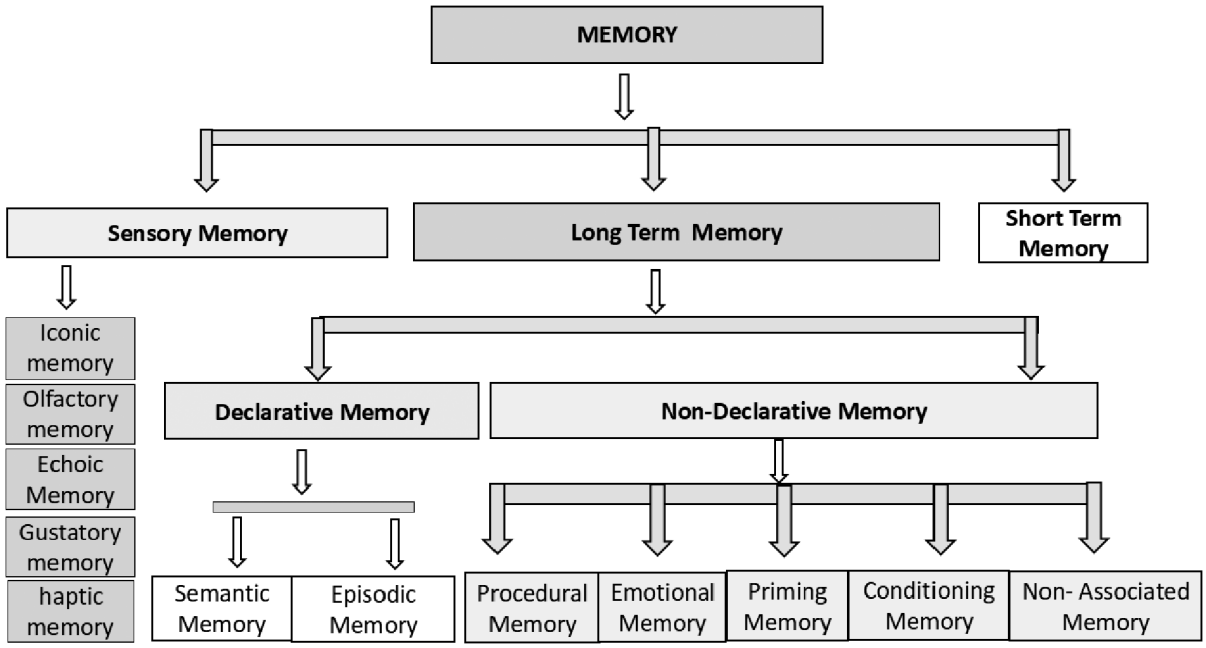

Figure 1. Illustration of memory classification: Memory is classified into sensory memory, short-term memory, and long-term memory. Long-term memory is divided into declarative and non-declarative memory. Declarative memory is classified into semantic and episodic memory, while nondeclarative memory is classified into procedural, emotional, priming, conditioning, and non-associated learning.

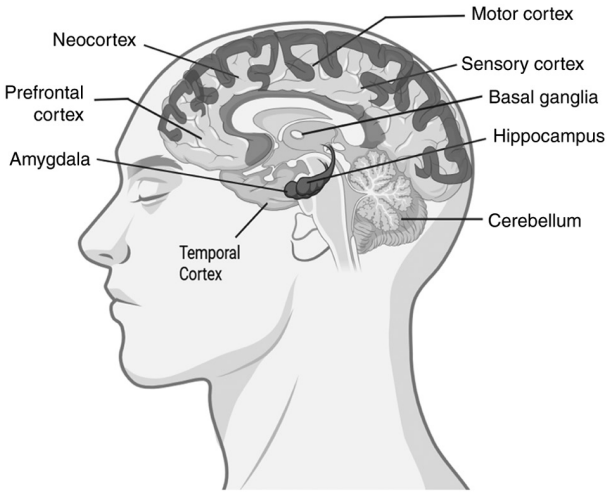

Figure 2. The illustration of different memory areas of the human brain: Semantic memory is coded by the prefrontal cortex, anterior and lateral temporal cortex; the hippocampus, medial temporal cortex, and neocortex code episodic memory. The basal ganglia code procedural memory. Conditioning memory related to the amygdala, cerebellum, and priming memory by the neocortex. Short term memory is encoded by the cortex region, the prefrontal cortex, the neocortex, sensory and motor cortex. The figure was created in BioRender (https://BioRender.com).

non-declarative memory (including procedural memory and classical conditioning) are depicted in Fig. 3.

Mechanisms of long-term memory. The more in-depth understanding of long-term memory formation, achieved through decades of research, provides exciting new avenues for improving cognitive function. Researchers believe that memory can be enhanced by targeting the mechanisms involved in memory formation, including activating cAMP response element-binding protein (CREB), managing (alpha-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid (AMPA)/N-Methyl-D-aspartic acid (NMDA) receptor trafficking, adjusting neuromodulators such as dopamine, adrenaline, cortisol, or acetylcholine, and influencing metabolic processes through substances, such as glucose and insulin (48).

The fundamental basis of long-term memory at the cellular level is synaptic plasticity, the ability of connections between neurons (synapses) to change in strength and structure. The most well-studied mechanism is LTP. LTP is a persistent strengthening of synapses that occurs when two neurons are repeatedly and simultaneously activated. This phenomenon is often summarised by 'neurons that fire together, wire together'. The first step of LTP is the induction of LTP. This typically involves the activation of NMDA receptors (a type of glutamate receptor) on the postsynaptic neuron. When these receptors are activated by coincident presynaptic activity and strong postsynaptic depolarisation, they allow an influx of calcium ions into the neuron. Manipulating NMDA receptors has been shown to improve both working memory and the consolidation of long-term memory (49). The second step is downstream signalling. The increase in intracellular calcium triggers a cascade of molecular events, activating various protein kinases (such as PKA, PKC and CaMKII) (50). The third step involves gene expression and protein synthesis (51,52). For true long-term memory (lasting hours to days or longer), these signalling pathways often lead to changes in gene expression and the synthesis of new proteins, such as CREB and immediate early genes (IEGs) (52).

CREB is activated in various signalling pathways, contributing to memory consolidation and even enhancement. For instance, when growth factors stimulate tyrosine kinase receptors, CREB initiates a chain reaction involving

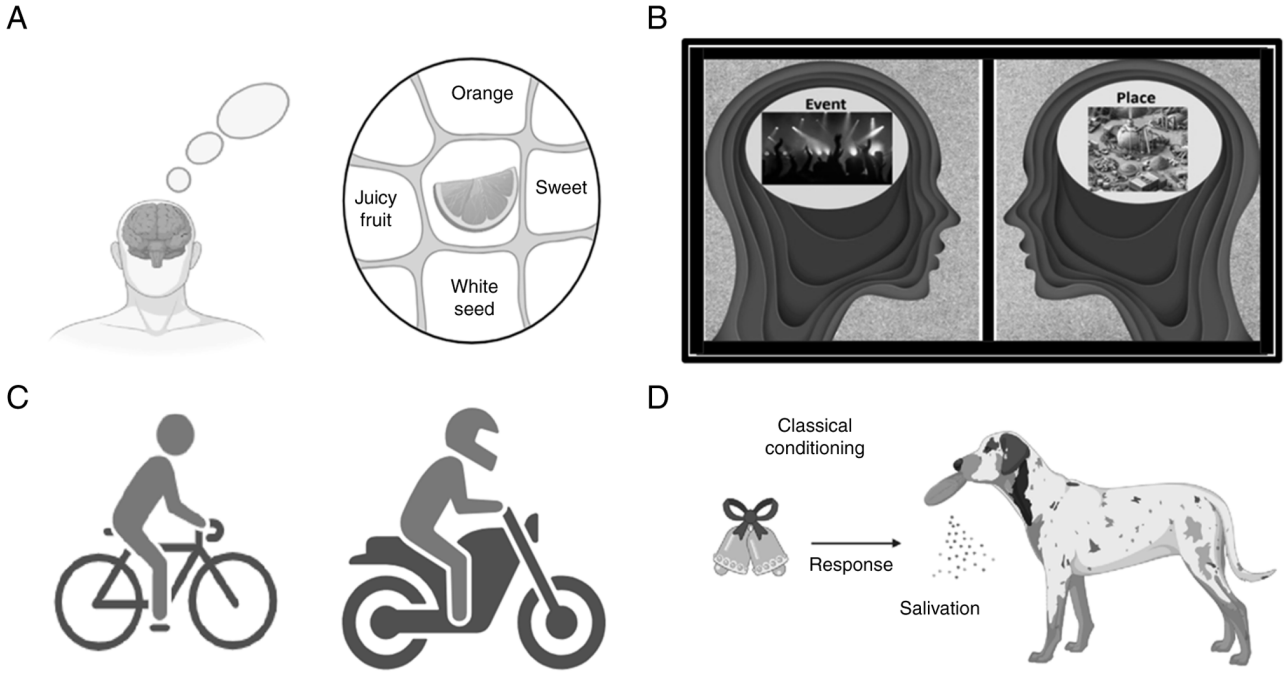

Figure 3. Illustration of declarative and nondeclarative memories (A) Semantic memory: Thinking about the facts of an orange. (B) Episodic memory, thinking about specific places and events. (C) Procedural memory, showing learning of cycling. (D) Classical conditioning memory of Pavlov.

Ras and extracellular signal-regulated kinase (ERK) (53). CREB plays a crucial role in long-term potentiation, which is a form of synaptic plasticity and a fundamental mechanism for storing memories. CREB also contributes in reconsolidation process (54).

GPCR activation induces adenylyl cyclase enzyme, which converts ATP to cAMP, which activates PKA. Even stress pathways and glutamate release can trigger CREB by causing the release of intracellular calcium and subsequent events (55). Ultimately, all these pathways can lead to the activation of CREB1, an active form of CREB. This activation controls the expression of numerous target genes, including IEGs. Some of these IEGs are themselves transcription factors, which indicates that they regulate other genes responsible for the synaptic changes that underpin synaptic plasticity. A good example is the IEG and transcription factor C/EBPβ, a gene regulated by CREB that is essential for memory consolidation (56). Altering the function of CREB1 has a direct impact on memory: When CREB1 is disrupted, memory impairments occur, whereas boosting its activation leads to improved memory (57).

These transcription factors then regulate the expression of effector genes that lead to lasting structural changes at the synapse (57). These include the growth of new synaptic connections, changes in the number and type of neurotransmitter receptors (e.g., more AMPA receptors), and modifications to the shape of dendritic spines (small protrusions on dendrites that receive synaptic input) (58). These structural changes are essential for the long-term stability of memories.

Role of NO in memory consolidation. The role of NO in memory consolidation has been recently reviewed (59). NO plays various roles in the central nervous system, including blood vessel dilation and neurotransmission to immune responses. The production of NO in the brain is mainly catalysed by the NO synthase (NOS) enzyme, which is found in three forms: Endothelial NOS (eNOS), neuronal NOS (nNOS) and inducible NOS (iNOS). While eNOS and iNOS are found in the central nervous system, nNOS is responsible for generating NO within neurons (60).

Specificity protein 1 (Sp1) and CREB are crucial transcription factors that regulate NO production by modulating the expression of NOS enzymes related gene. nNOS can interact with Sp1, and the interaction can inhibit Sp1 binding to DNA, providing a mechanism for NO to modulate its own production. Once synthesised, NO can diffuse to the postsynaptic cleft, where it exerts diverse effects on neighbouring neurons (61).

Memory consolidation is the complex process by which short-lived memories transform into a more stable memory (51). NO is crucial to this process, largely through its involvement in synaptic plasticity (51,62,63). Synaptic plasticity is an activity-dependent modification of the efficacy of synaptic transmission at synapses. It plays a crucial role in the capacity of the brain to include transient experiences into persistent memory traces.

NO strengthens synaptic plasticity by enhancing LTP in the hippocampus and cerebral cortex (64). It facilitates the conversion of labile memories into a stable memory through several pathways. NO functions as a retrograde messenger; it diffuses backwards from postsynaptic neuron to presynaptic neuron through the synaptic cleft and targets soluble guanylyl cyclase in the presynaptic neuron, enhancing neurotransmitter release. These versatile roles of NO ensure that neural circuits are optimally configured for the long-term retention of information (65). This comprehensive involvement of NO indicates the significance of cognitive functions and suggests its potential as a target for future therapeutic strategies in treating memory-related disorders.

In the hippocampus, NO functions as a retrograde messenger following the activation of NMDA receptors (NMDARs) during LTP (64). The process begins when NMDARs on the postsynaptic neuron are activated, leading to an influx of calcium ions (Ca^{2+}) (66). This calcium flow activates nNOS, which produces NO. NO rapidly diffuses back from the postsynaptic terminal to the presynaptic terminal, where it activates soluble guanylate cyclase. This enzyme then catalyses the conversion of GTP to cGMP. The resulting increase in cGMP activates protein kinase G (PKG) (67). PKG further phosphorylates various proteins involved in neurotransmitter release, such as glutamate, and finally strengthens the synapse, promoting LTP. The complex signalling cascade plays a critical role of NO in regulating synaptic plasticity and memory consolidation (67).

7. Declarative memory

Declarative memory is also known as explicit memory, a type of long-term memory that involves the conscious recollection of facts, events and concepts (68). It is the type of memory that can intentionally be called to mind and verbalised. It has been indicated that the medial temporal lobe, particularly the hippocampus, plays a crucial role in declarative memory (39). This form of memory is further categorised into two main types: Semantic and episodic memory.

Semantic memory. Semantic memory deals with general knowledge and facts about the world, independent of personal experience or context. It is based on the concepts, principles and abstract information, such as 'the Moon revolves around the Earth' or 'a Square has four sides', relies on semantic memory. It is the repository for all the knowledge which has been accumulated. Research indicates that the semantic memory networks of older adults has greater separation and segregation between concepts, with fewer connections among them. Networks offer a powerful strategy to model the organisation of conceptual representations (69,70). These computationally modelled semantic memory networks go beyond analysing simple word-to-word relationships. They enable researchers to examine complex interactions among multiple words and assess the overall structural characteristics of a network, such as its efficiency. Researchers have applied semantic memory network analyses in diverse fields, including creativity, language acquisition, studies of clinical populations and research on healthy ageing (70,71). Evidence suggests that with advancing age, semantic memory exhibits reduced efficiency, poorer organisation and fewer connections (72,73). Ageing influences certain cognitive functions, including language production, which can result in older adults experiencing more difficulty with word retrieval.

How visual and semantic representations influence memory. A previous study explored how different types of visual and semantic information contribute to memory formation. The researchers used a deep neural network to model three levels of visual processing: Early, middle and late (74). For semantic representations, they drew upon normative features (e.g., 'is round'), taxonomic categories (e.g., 'is a fruit') and encyclopaedic knowledge (e.g., 'is sweet'). The aim of that study was to identify brain regions where each type of representation predicted either perceptual memory (remembering what something looked like), conceptual memory (remembering what something meant), or general memory (remembering both) (74).

During the study, participants viewed objects, while undergoing functional magnetic resonance imaging (fMRI). Afterwards, they completed two memory tests: One conceptual (word-based) and one perceptual (picture-based), and found that visual representations were strong predictors of subsequent perceptual memory within the visual cortices. Notably, they also supported conceptual and general memory in more frontal brain regions. Semantic representations, on the other hand, predicted perceptual memory in the visual cortex, conceptual memory in the perirhinal and inferior prefrontal cortex, and general memory in the angular gyrus. These findings suggest that the way visual and semantic information contributes to later memory is intricate, depending on the specific type of information, the kind of memory being tested, and where that information is stored in the brain (74).

How brains organise knowledge about individuals and places. Humans build up incredibly detailed semantic knowledge about the world around them. This includes information about individuals, such as their jobs and personalities, and places, such as their cultural or historical importance. While certain brain regions show a preference for images of individuals and places, less is understood about whether these same areas also specifically store the semantic knowledge associated with them. To investigate this, a previous study developed a machine learning model to gauge semantic similarity using information from Wikipedia, then validated this model by comparing its similarity ratings with those provided by human participants (75). Using the computational model, researchers discovered that the brain represents semantic knowledge about individuals and places in distinct networks: An anterior temporal network for people and a posterior medial network for places. Of note, the hippocampus, a crucial memory hub, was found to represent semantic knowledge for both people and places (75).

Episodic memory. Episodic memory represents all the previous events of the life of an individual that occurred at a particular place and specific time. These types of memories are often associated with an individual emotion. For instance, remembering the first day of school, a memorable vacation, or a significant life event, such as winning a championship in any game or in event are to be included in episodic memories, and these memories and moments from life can be recalled (76,77). A previous study revealed that a specific circuit, comprising the medial prefrontal cortex, lateral entorhinal cortex and hippocampus, encodes episodic-like memories by integrating event, place and time information (78). This process appears to involve top-down regulation from the medial prefrontal cortex to the hippocampus. This circuit is distinct from those processing individual components of memory (object, time and place).

Episodic memory is vulnerable to decline in both ageing and neurodegenerative conditions (79,80). Research indicates that episodic memory performance typically undergoes a

consistent, linear decrease beginning at ~50 to 60 years of age (80). Ageing is associated with declines in processing speed, working memory, and episodic memory (81). With age, the human brain shrinks slightly, and white matter integrity diminishes, and these age-related changes also cause the loss of episodic memory, especially related to the hippocampus. The hippocampus, which is vital for encoding, consolidating, and retrieving episodic memories, undergoes substantial volume loss, particularly after age 50, alongside reduced synaptic plasticity and other neurobiological alterations (82). Furthermore, the hippocampus appears to become functionally disconnected from other brain regions, such as the posterior and anterior cingulate cortices, with age (83,84). These neural changes are linked to age-related declines in overall cognition and episodic memory. In fact, the hippocampus is often the most severely affected brain structure to show pathological changes, such as volume loss, in Alzheimer's disease (AD) and its prodromal stage (mild cognitive impairment) (85,86). Its condition is also the strongest predictor of episodic memory impairment in AD.

The prefrontal cortex is another key brain area involved in how episodic memories are formed and recalled, particularly those that require mental control, practice and the ability to block out distractions (87,88). Notably, with increasing age, the prefrontal cortex exhibits greater activity during episodic memory tasks, and this activity tends to spread out and involve both sides of the brain (89). This indicates that older adults do not exhibit the same one-sided brain activity in the frontal and prefrontal areas that younger adults typically do. This shift may be a compensation mechanism for the brain for age-related changes. In fact, older adults who perform well on memory tasks tend to use both sides of their prefrontal cortex more strongly than those who do not perform as well (87). Furthermore, with age, the prefrontal cortex region experiences a noticeable decrease in both white matter (the connections between brain cells) and grey matter (the brain cells themselves) (90,91).

Episodic memory often declines as a natural part of ageing, and this decline is also a key feature of age-related memory disorders. A number of brain areas are known to be involved in both episodic memory and age-related cognitive changes. The recent study by Almeida *et al* (92) specifically demonstrated that the cerebellum plays a causal role in episodic memory during ageing. They found that healthy older adults who received a 12-day neurostimulation program targeting the right cerebellum experienced significant improvements in their episodic memory (92). These improvements were not only immediate; they also lasted for at least four months after the stimulation period ended. These findings underscore the direct involvement of the cerebellum in long-term episodic memory processes and suggest that it may be critical for regulating and maintaining overall cognitive function.

Effect of episodic memory on delay discounting. Episodic memory may slightly decrease delay discounting in certain situations; this impact is generally minor and easily disrupted (93). Delay discounting describes the process through which the perceived value of a reward diminishes with the increment in the delay to receiving it (94). This often leads individuals to opt for smaller, immediate rewards instead of pursuing larger, future. Delay discounting is typically measured by presenting participants with hypothetical choices: a smaller sum of money available now vs. a larger sum available at a later date (95). A greater tendency to select the larger, later sum indicates lower levels of delay discounting, often seen as a measure of self-control. Conversely, consistently choosing the smaller, sooner option reflects higher levels of delay discounting, which can be an indicator of impulsivity.

Episodic future thinking (EFT) stands out as a strategy that encourages individuals to choose larger, more distant rewards (96). When prompted to vividly imagine positive future events, people tend to favour a larger sum of money available later (96,97). EFT has been shown to reduce delay discounting in both adults and children (98), and it can even help decrease cigarette consumption (99) and food intake (100).

It raises a critical question of whether simply recalling past experiences or episodic memory help reduce delay discounting. A small number of studies on EFT have used episodic memory as a comparison point. These studies have consistently demonstrated that EFT is significantly more effective at decreasing delay discounting than recalling past events (99,101). However, this does not completely rule out the possibility that episodic memory could still boost self-control compared to not engaging in any episodic thinking at all. The impact of EFT on delay discounting is likely influenced by several factors, including whether the thought is focused on the future, its emotional tone (valence), and how episodic or vivid the thinking is.

Developmental changes in episodic memory. Episodic memory is the unique ability to recall personal experiences, including not only the event itself (the 'what'), but also the specific location (the 'where') and time (the 'when') it took place (102). For instance, if one thinks their last anniversary, they may remember who they celebrated with, where they had lunch, and whether you they had dessert before or after opening presents.

To understand how this ability develops in early to mid-childhood, a previous study explored the effectiveness of four different lab-based tasks in characterising changes in episodic memory in 200 typically developing children, aged 4 to 8 years (103). Utilising longitudinal data and a structural equation modelling framework, the findings of that study indicated that multiple episodic memory tests can effectively measure a consistent underlying latent construct of episodic memory throughout this developmental period. Furthermore, this ability consistently improves between the ages of 4 and 8 years (103). These results underscore that episodic memory, as a measurable construct, increases at a similar rate during early to mid-childhood. That study also highlighted the advantages of employing diverse laboratory tasks to track these developmental changes in episodic memory (103).

Correlation of declarative memory and children's math skills. Significant advancements have been made in understanding the brain processes behind math learning. A prevailing hypothesis suggests that declarative and procedural memory, as fundamental learning and memory systems, play critical roles in the acquisition of mathematical skills.

To investigate this, a previous longitudinal study examined whether declarative and procedural memory predict the

math proficiency of children during their elementary school years (104). The researchers of that study followed 109 children from second to fourth grade, testing them each year. The results revealed the following: i) Within each grade, stronger declarative memory skills were linked to improved math performance, but procedural memory skills were not. ii) The declarative memory of a child in an earlier grade predicted their math skills in later grades. For example, good declarative memory in the second grade was connected to improved math skills in the fourth grade (104). Sensitivity analyses confirmed the robustness of these results, apart from when accounting for stable individual differences through random intercepts in the longitudinal prediction of later math skills. These findings emphasise the foundational contribution of early, general learning and memory mechanisms to children's mathematical development (104).

How sex and education affect declarative memory in older adults. Declarative memory, which aids in the learning and recalling of everyday facts and events, typically weakens with age. However, some research suggests that the sex of an individual and the amount of education they received early in life may help lessen these memory declines.

Previously, researchers conducted a study with 704 older adults in Taiwan, ranging from 58 to 98 years of age, with varying levels of education (0 to 17 years) (105). They investigated their non-verbal declarative memory by testing their ability to recognise drawings of both familiar (such as a hairbrush) and unfamiliar made-up objects after seeing them briefly. The findings of that study revealed that age negatively affected declarative memory, although this impact varied based on the person's sex and the type of object. Memory decline was steeper for men than for women, revealing that women had an advantage in remembering real objects beginning at ~70 years of age. However, the remembering of made-up objects exhibited a slower decline with no difference between the sexes (105). Education was linked to improved memory, although its benefits also depended on sex and object type. Education helped women more than men, and it was more beneficial for remembering real objects than made-up ones. For men, each year of education brought memory gains twice as large as the losses experienced each year due to ageing. For women, these gains were 5-fold greater. These results suggest that in older adults, non-verbal memory declines with age, but improves with more education. However, both of these associations are influenced by the sex of an individual and whether the information being learned relates to existing knowledge or is entirely new. That study highlights the lasting benefits of education, particularly for women (105).

Movement behaviours and their associations with declarative memory and hippocampal development in early childhood. A previous study investigated whether 24-h movement behaviours, specifically sedentary time, physical activity and sleep, were linked to declarative memory and the size of the hippocampus (a brain area crucial for memory) in young children (105). They examined these behaviours both individually and as a whole. That study collected observational data from pre-schoolers at two points: First, with 35 children averaging 3.9 years of age and then 6 months later, with 28 of those children, who were then ~4.5 years of age. They measured their movement using activity trackers (106). The main parameters investigated were their declarative memory scores and the volumes of different parts of their hippocampus. Using various statistical models, they analysed the movement behaviours. When they examine each behaviour on its own, they found that longer sleep durations were linked to a larger total hippocampal volume, and moderate to vigorous physical activity was associated with a larger right hippocampus (106). When they considered all movement behaviours together, children who met recommended sleep guidelines tended to have larger hippocampal volumes overall, including in the right and left hemispheres, and specifically in the body and tail regions of the hippocampus. In the group of pre-school children, the researcher observed a clear positive connection between sleep duration and hippocampal volume, regardless of the child's age (106). To better understand how these 24- behaviours relate to brain health in very young children, future studies are required to include more participants and also consider the specific types and settings of movement.

8. Non-declarative memory

Non-declarative memory, also known as implicit memory, is a type of long-term memory that is not conscious. This memory allows the performance of skills and routines automatically without having to think about them (107). For example, riding a car, bike, or cycle, DNA isolation from an onion, or typing are examples of non-declarative memory. Non-declarative memory is acquired and retrieved without conscious effort. It is built up through repetition and practice, rather than through conscious recall, while declarative memory (or explicit memory) involves the conscious recollection of facts, events, and concepts. It can be divided into various types, including procedural memory, emotional memory, priming, and classical conditioning. Each memory is managed by various brain areas and associated pathways (107,108). Non-declarative memory, also known as implicit memory, supports the involuntary abilities and learned reactions, operating without conscious recall (62).

Procedural memory. This is based on motor skills such as riding a motor car, bicycle, typing, RNA isolation from blood, and playing a musical instrument. After learning, the person can perform the task without conscious effort. Research indicates that the basal ganglia plays a role in procedural memory (109). Navigational memory is also associated with the basal ganglia, although the basal ganglia are not only responsible for procedural memory; other parts of the brain also play a crucial role in navigation. The basal ganglia consist of numerous subcortical nuclei present in the cerebrum, the substantia nigra, and the subthalamic nucleus of the brain. The primary components of the basal ganglia, found in the cerebrum, include the caudate nucleus, putamen, globus pallidus and the accumbens area (110,111). Additionally, the substantia nigra (located in the midbrain) and the subthalamic nucleus (in the diencephalon) are also included in the basal ganglia (112).

The basal ganglia play a crucial role in processing and refining information from the cerebral cortex, contributing to motor, cognitive and limbic functions (113,114). The basal

ganglia serve as a critical neural centre for movement; they are responsible for activating the motor unit of muscle for the movement (115,116). The basal ganglia are also responsible for habit formation, and the caudate nucleus (a specific nuclei of the basal ganglia) especially facilitates successful outcomes by promoting effective behavioural patterns and selecting logical subgoals based on a continuous evaluation of results (117).

Apart from the motor control function, the basal ganglia also play a critical role in various cognitive processes, such as decision-making, shifting attention, updating information and adapting behaviour (118). The reward processing function, which influences behaviour, choices and motivation is also associated with the basal ganglia. Furthermore, the basal ganglia are involved in processing emotions, regulating affective states, and integrating emotional information into decision-making.

Research indicates that the brain actively engages conscious, explicit (or declarative) processes during the development of procedural memories (119). It suggests that conscious attention and cognitive effort play an important role in skill-associated functions. Research suggests that the brain depends on declarative memory during the early-stage encoding of a new motor or cognitive procedure. The procedural learning is not entirely unconscious, but can be achieved by practice or repetition. It also involves intellectual ability (107), which helps in understanding and strategizing during the learning phase. Episodic memory (120), allowing learners to recall specific events or steps, especially when first acquiring the skill. Executive functions (121), which include abilities such as planning, problem-solving and working memory, all critical for navigating the complexities of new tasks.

Role of the hippocampus and basal ganglia in navigation. The nature of human navigational memory remains one of the most persistent and fiercely contested puzzles in cognitive science. It raises the fundamental question of whether the brain builds a literal, unified map of the world (cognitive map), or whether it merely integrates (patch) together a series of directions and landmarks (graph or route). The debate is primarily between two research groups of thought regarding how spatial information is stored in the hippocampus and entorhinal cortex. The cognitive map theory, proposed by Tolman (40) in 1948, suggests that navigation is the result of the creation of an internal map inside the brain (cognitive map) that allows organisms (rats or humans) to understand and manipulate spatial relationships (40). Moving beyond simple turn-by-turn sequences (egocentric navigation), the study by Tolman (40) with rodents suggested that animals could locate objects by referencing multiple landmarks relative to one another (allocentric navigation). The second theory proposed by Wang and Hayden (122) states that organisms do not have a unified map. Instead, they store connected nodes (landmark A → right turn → landmark B) and navigate by integrating these local memories (122). Wang and Hayden (122) also hypothesised a dual-process model for curiosity-driven learning involving the orbitofrontal cortex and the dorsal anterior cingulate cortex. Under this framework, the orbitofrontal cortex is responsible for evaluating the inherent worth of new data and integrating it into an internal cognitive map. Moreover, the dorsal anterior cingulate cortex monitors external constraints and the accessibility of information, drawing upon the orbitofrontal cortex's cognitive map to direct goal-oriented actions (122).

The 2014 Nobel Prize in Physiology or Medicine was awarded to Dr John M. O'Keefe, Dr May-Britt Moser and Dr Edvard I. Moser (123) They were recognized for their ground-breaking discovery of the nerve cells in the brain that make sense of place and navigation possible. They demonstrated how complex cognitive functions and behaviours are represented by specific cells found in a specific region of the brain. Dr John O'Keefe discovered place cells in the hippocampal region of the brain. These cells function as a type of GPS and provide the location of an animal by signalling mechanisms, enabling the brain to form spatial memories (124,125). Later, Dr May-Britt Moser and Dr Edvard I. Moser discovered grid cells in the medial entorhinal cortex, near the hippocampus. These grid cells generate a specific internal coordinate system that is important for the navigation process. These two types (hippocampal place cells and entorhinal grid cells) form an interconnected network that is required for making spatial maps and navigation (125). The work of these three scientists has fundamentally changed the understanding of how brains perform complex cognitive tasks and create spatial memory.

An animal needs to build a cognitive map (a mental representation of its environment) to navigate successfully (126). Several brain regions and different types of cells work together to form this internal navigation system. The hippocampus is a crucial region of the brain to be considered for navigation in rat models (127). Two key cells are place cells in the hippocampus and grid cells in the medial entorhinal cortex (125,128). Hippocampal place cells fire specifically when an animal is in a particular spot, creating a place field that signals its location (129). This function depends on both sensory information, such as what the animal sees, and spatial cues from the medial entorhinal cortex that are independent of its senses (124).

The medial entorhinal cortex uses grid cells to create a sensory-independent spatial map (130,131). Head direction (HD) cells signal the direction the head is facing, while grid cells fire in multiple locations, creating a repeating, hexagonal pattern (132). These grid cells generate a unique hexagonal firing pattern as an animal moves through an open space, essentially forming a coordinate system in the brain (129) (Fig. 4).

The activity of grid cells is influenced by the speed and direction of the animal. This suggests that they integrate information about self-motion (known as idiothetic cues) to calculate distance and direction, which is crucial for a process called path integration (132,133). In a previous study, to determine whether place cells help correct errors in a process called path integration, researchers decoded the location of an animal using grid cell activity (134). They achieved this with and without input from place cells. They discovered that when place fields emerged during exploration, the accumulated error in the animal's position estimates decreased (134). Place fields that were closer to the current location of the animal were more effective at reducing this error than those that were further away. That study suggested that place cells function as spatial anchors and provide precise information about the location of an animal that helps grid cells maintain accurate path integration (134).

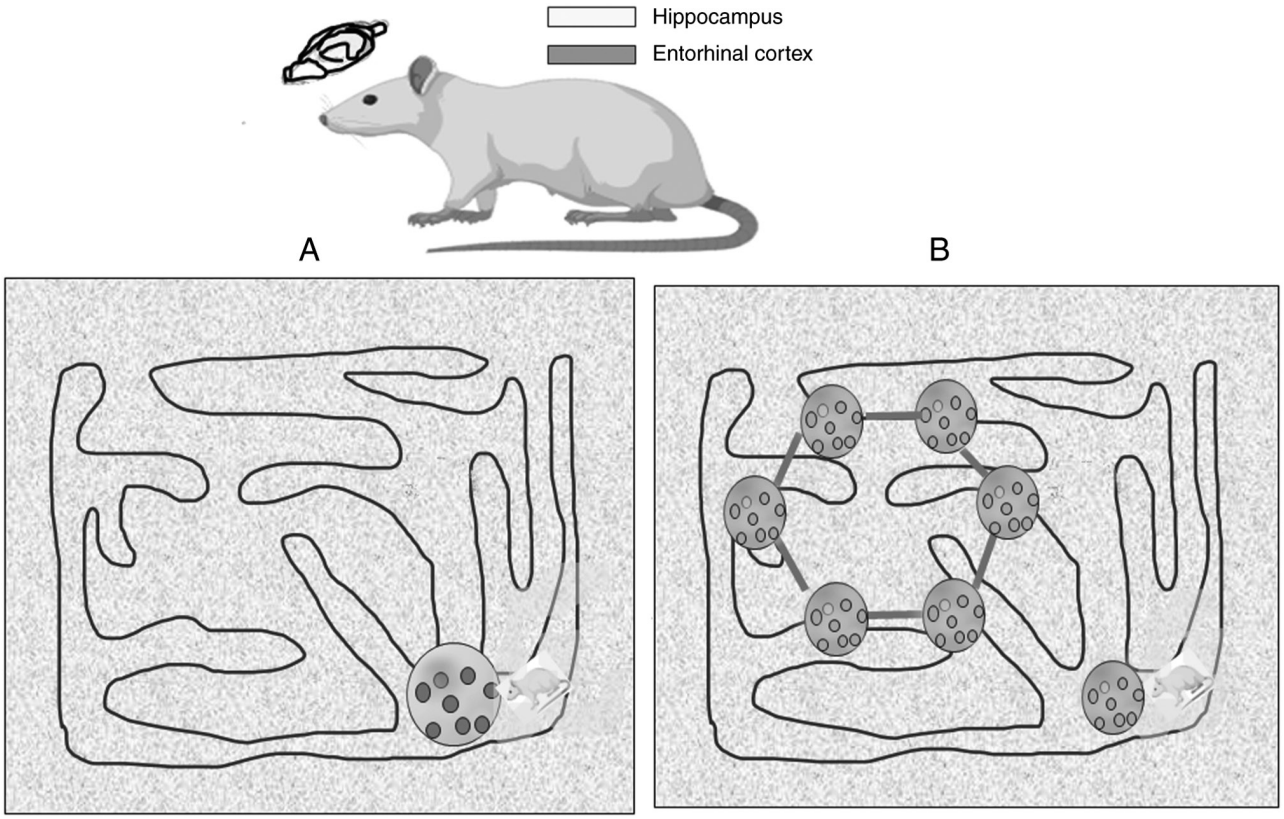

Figure 4. Schematic diagram of a rat showing place cells and grid cells. (A) The hippocampus place cells which fire when the rat reach at a particular position in the environment. Place cells serve as an internal GPS that maps the movements of a rat and enables the formation of spatial memories by firing at specific coordinates, and provide a rat with its real-time position. (B) Shows of grid cells, which are located in the entorhinal cortex, activate when the rat reaches specific spots in an environment. These firing locations form a unique hexagonal grid pattern. In this way, it generates an internal coordinate system for navigation. These two types of cells, hippocampal place cells and entorhinal grid cells, form an interconnected network for building spatial maps and navigation.

Previous research has suggested the coordination of three regions of the brain for the navigation procedure. First, the hippocampus and entorhinal cortex generate the core, map-like spatial coordinates. Second, the parahippocampal and retrosplenial cortices act as anchors, tethering these internal maps to stable physical landmarks. Finally, these spatial codes are integrated with frontal lobe processes to transform a mental map into an actionable navigation plan (135).

The role of the basal ganglia in motor behaviour has long been observed in mammals; however, the behavioural functions are not exclusively motoric in nature. The basal ganglia facilitate a form of learning in which stimulus-response (S-R) associations or habits are acquired (136). There is evidence to indicate the role of the basal ganglia, particularly the dorsal striatum, in learning and memory (109). The basal ganglia also function with other brain areas, such as the hippocampus, to support various navigation methods (137). While the hippocampus creates a mental map of one's surroundings, the basal ganglia enable more rapid, more efficient navigation based on learned patterns. The brain can then select which strategy to use, be it spatial or habit-based, depending on the situation and environment.

Navigational technology such as GPS devices and applications has become a normal part of daily life over the last 10 years. However, this technology affects the built-in navigation system of the brain, also known as spatial memory. This internal system relies on the hippocampus to function properly. A previous study examined 50 regular drivers and recorded the extent of the use of GPS throughout their lives, and tested various parts of their spatial memory (138). These tests included tasks that measured their navigation strategies, ability to create mental maps and their use of landmarks while navigating in a virtual environment. The initial results indicated that those who had used GPS more often in their lives tended to have a worse spatial memory (138). That study also followed-up 13 of these participants 3 years later and found that those who had used GPS more frequently since the first test exhibited a more significant reduction in their hippocampus-dependent spatial memory over that time. Of note, they found that those who more often used GPS, did not do so because they already felt they had a poor sense of direction. That study suggests that the heavy use of GPS itself may be causing a decline in spatial memory, rather than being a symptom of it (138). These results are particularly important as society continues to increasingly depend on GPS technology.

Priming. This phenomenon demonstrates how prior exposure to a stimulus can unconsciously influence the response to a later related stimulus. Priming may be perceptual or conceptual. Perceptual priming occurs when our prior encounter with an item makes it easier to process or identify that same item again. When an individual sees a specific image, the next time this is seen, even briefly, it will be recognized faster as the brain has been primed. In conceptual (or semantic) priming,

exposure to an item improves the ability to recall or process a related item. For instance, if one hears the word doctor, one can more quickly recognize or think of the word nurse, as the two concepts are linked in one's mind. The neocortex is involved in both the encoding of memories and their subsequent consolidation, through the interaction with the hippocampus (139).

Classical conditioning. This is a type of associative learning involves the process through which a neutral stimulus becomes linked to a specific response after being repeatedly paired with a meaningful one. A classic example is Pavlov's dogs, which learned to salivate at the sound of a bell as they associated it with food (140).

Experiences often create powerful associations that can unconsciously influence one's reactions. In the same manner as exams can induce anxiety for someone, being chased or threatened by a dog in childhood could create a lifelong fear of canines. These are instances where a specific stimulus becomes linked to an emotional response known as learned associations and emotional responses. One might find oneself reaching for his/her own smartphone when another phone nearby rings with a familiar tone. This is known as conditioned reactions to stimuli. These automatic reactions demonstrate how one can be conditioned to respond to certain cues. A well-known illustration of this is *John Watson's 'Little Albert' experiment* (141). In that study, a baby who initially had no fear of a white rat was conditioned to fear it. Researchers repeatedly paired the presence of the white rat with a loud, startling noise. Over time, the baby began to exhibit fear solely at the sight of the rat, demonstrating how a neutral stimulus can become associated with a powerful emotional reaction. It was an example of classical conditioning.

Emotional memory. The amygdala is responsible for emotional responses, such as pleasure, weeping and anger. It directly influences emotional learning and assists memory processes in other areas, such as the hippocampus and prefrontal cortex. The interaction of emotion and memory takes place across different stages of information processing, from the initial formation and strengthening of memories to their later recall (41).

A notable feature of human memory is its ability to enhance explicit, consciously recallable memories for emotional experiences. Research, drawing from neuroimaging, neuropsychological, pharmacological and neural stimulation studies, suggests that emotional stimuli activate particular cognitive and neural processes that improve explicit memory (142). Emotional arousal impacts memory through factors active during the initial encoding stage (such as attention and elaboration) and through elements that adjust memory consolidation (143,144). The role of the amygdala has been identified in strengthening explicit memory for both positive and negative emotional stimuli by modulating both encoding and consolidation (145,146).

While heightened emotional states typically lead to improved memory in healthy individuals, it remains unclear whether this effect holds true for patients with AD. A previous study aimed to compare emotional memory and emotional responses in individuals with early AD against a group of older adults without the condition (147). Participants in both groups viewed a mix of emotionally evocative (both pleasant and unpleasant) and neutral images while their cognitive and electrophysiological reactions were monitored. Memory was later assessed using both free recall and recognition tasks. The findings of that study indicated that while patients with AD displayed normal emotional reactions, their emotional memory effect was impaired; specifically, they failed to remember emotional stimuli better than neutral ones (147).

Electrical stimulation and emotional processing. The dorsolateral prefrontal cortex is generally considered to play a role in emotional processing, yet the precise contribution of each hemisphere remains a topic of ongoing debate. To investigate the unique role of the left dorsolateral prefrontal cortex in the encoding and retrieval of emotional stimuli, a previous study utilized unilateral transcranial direct current stimulation (tDCS) in healthy individuals (148). A total of 42 right-handed undergraduate students participated, receiving either anodal, cathodal, or sham stimulation to their left dorsolateral prefrontal cortex while viewing a series of neutral, pleasant and unpleasant images. Following a filler task, participants were asked to recall as many pictures as they could (148). The findings of that study indicated that participants remembered more emotional pictures (both pleasant and unpleasant) than neutral ones, irrespective of the tDCS condition. Notably, those who received anodal stimulation recalled significantly more pleasant images compared to participants in the sham and cathodal conditions. No such differences were observed for the recall of neutral or unpleasant images. These results suggest that the left prefrontal cortex contributes specifically to the encoding and retrieval of pleasant stimuli (148).

Auditory cortex involvement in emotional memory. Emotional memories are fundamental to human and animal existence, shaping future decisions and actions. Historically, brain lesion studies in animals have suggested the involvement of the auditory cortex in emotional learning primarily through processing auditory stimuli linked to emotional outcomes and relaying this data to the amygdala (149). However, electrophysiological and imaging research has revealed that emotional experiences induce specific, associative, and long-lasting changes within the auditory cortex itself. These findings indicate that the role of the auditory cortex extends beyond simple stimulus processing and transmission. Three important implications of these data have been explored: i) The potential roles of the auditory cortex in emotional learning; ii) its involvement in both early and late stages of memory trace encoding; and iii) the functional interaction between the auditory cortex and subcortical regions, particularly the amygdala, which handles affective information. It was concluded that the auditory cortex plays a more significant and prominent role in emotional learning, particularly from the early stages of memory encoding and through its connections with subcortical nuclei, than previously understood (150).

The British philosopher, Gilbert Ryle, introduced the important distinction between procedural and declarative memory in his 1949 book, *The Concept of Mind; Role of histamine in emotional memory* (151,152). As a key neuromodulator, histamine responds to various emotional states, including stress, anxiety, and reward-associated activity. We can therefore infer that histamine is capable of encoding the emotional valence (the intrinsic attractiveness/aversiveness) and salience

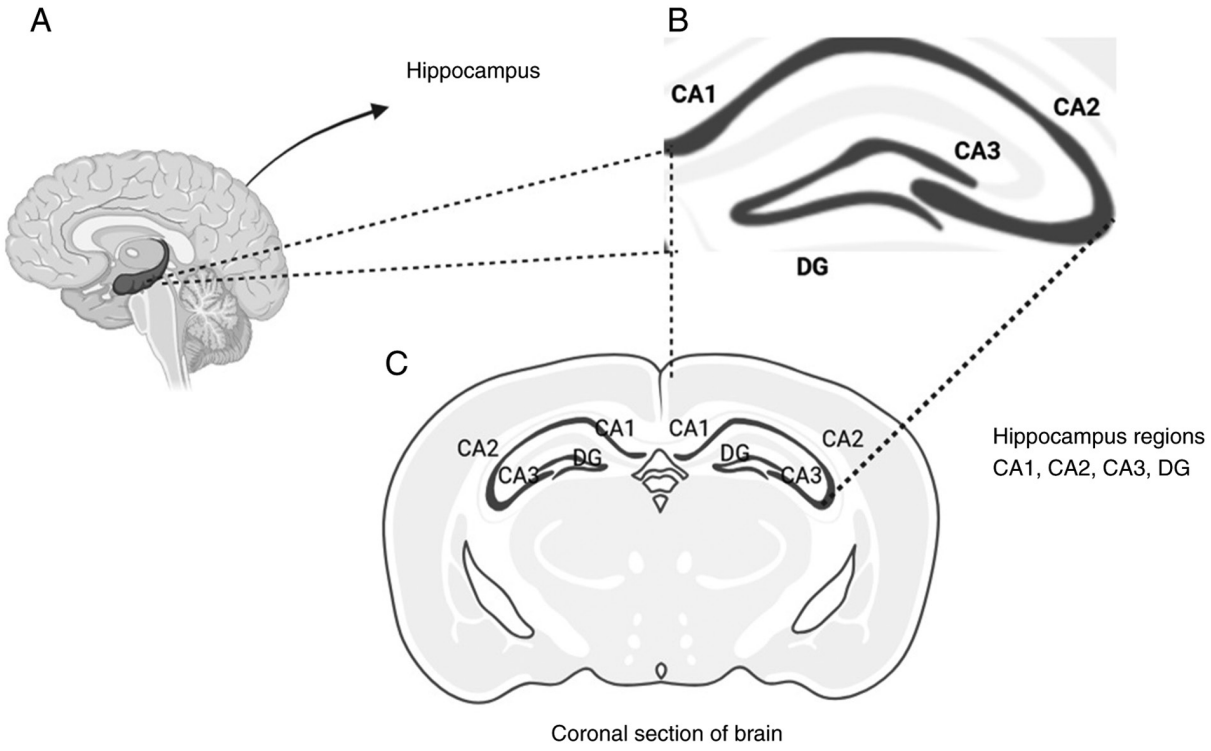

Figure 5. (A) Illustration of a location of hippocampus in the brain. (B) Different regions of hippocampus CA1, CA2, CA3 in hippocampus. (C) Coronal section of the mouse brain, and the location of the hippocampus is shown which indicates different subareas such as Cornus Ammonis (CA) and Dentate gyrus (DG). CA is subdivided into 3 region CA1, CA2, and CA3. While DG is the area where neurogenesis was shown. The image was created using BioRender (https://BioRender.com).

(the distinctiveness/importance) of stimuli. Consequently, histamine appears to be significant in the consolidation and retrieval of emotional memories, potentially through associative processes that connect sensory experiences with the value of unconditioned stimuli (153). Despite these insights, the specific mechanisms by which histamine contributes to emotional learning and memory are not yet fully understood. Future research utilizing cutting-edge technologies, dissecting histaminergic circuits, and employing spatiotemporally selective manipulation of histamine receptors will be crucial for addressing these gaps and for developing clinical strategies for emotional and memory disorders.

Non-associative learning. This category includes changes in the response to a single stimulus over time: Habituation is when a response to a repeated stimulus decreases. For example, if one resides close to a train track, eventually, that individual will stop noticing the sound of passing trains. Sensitization is the opposite; it is an increased response to a repeated stimulus. For example, when one is anxious, a small unexpected noise may startle someone to a greater extent than it usually would. Non-declarative memory is essential for countless daily activities, allowing for the expert navigation of one's surroundings and the mastering of intricate abilities without the need for deliberate thought.

9. Neurogenesis and memory association

The hippocampus is associated with learning and memory formation (154,155). However, researchers continue to argue over its exact functional contributions. This uncertainty arises from the involvement of hippocampal formation in various processes, including spatial navigation (developing internal cognitive maps), relational encoding (understanding the links between different pieces of information), complex associations (managing configural representations) and episodic memory (155).

Conceptual complexity arises in ongoing debate regarding how specific subfields within the hippocampus contribute to these functions. It is important to know that the dentate gyrus (DG), CA3, and CA1 are three primary regions of the hippocampus that exhibit distinct properties. Specifically, the DG is unique among them for its ability to generate new neurons throughout adulthood, a process known as adult neurogenesis (156). The DG, CA3 and CA1 region in the hippocampus is illustrated in Fig. 5.

The debate over adult hippocampal neurogenesis is one of the most confrontational topics in modern neuroscience. Rodents produce new neurons in the hippocampus throughout their lives; the process, is known as adult hippocampal neurogenesis (AHN), is a potent example of how mammalian brains remain adaptable and plastic over time. Its existence in humans remain subjects of intense disagreement among researchers (157,158). The current debate began in 2018 by two research groups that reached opposite conclusions using similar post-mortem human tissue. i) Low neurogenesis view: Sorrells *et al* (159) argued that AHN sharply decreases during childhood and becomes undetectable or extremely rare in adults. They suggested that the human hippocampus may differ fundamentally from other mammals by losing

its neurogenic capacity early in life (160). ii) The persistent neurogenesis view: By contrast, Boldrini *et al* (161) reported finding thousands of immature neurons in the DG of humans across the lifespan, from the age of 14 to 79 years. This view was later supported by Moreno-Jiménez *et al* (162) who identified tens of thousands of neuroblasts in healthy adult brains, although they noted a significant decline in patients with AD. The controversy is primarily driven by technical limitations in studying human brain tissue: i) Post-mortem delay: Markers of immature neurons, such as doublecortin (DCX), degrade rapidly after death. Critics of the no neurogenesis view argue that long delays between death and tissue fixation can make these markers vanish, leading to false negatives (157,158). ii) Marker specificity: There is debate over whether common markers such as DCX or PSA-NCAM exclusively label new neurons or if they might also label remodelling mature neurons or glial cells (163).

10. Memory modification technologies and ethical considerations

Memory modification technologies (MMTs) are tools that are being used to boost memory beyond its natural capacity. While medical treatments focus on the restoration of memory loss. Individuals have long used specific devices or technology to enhance their memory. The current understanding of neuroscience reveals that memory modulation can be achieved by either pharmacological drugs, such as glutamate receptor agonists, N-Methyl D-aspartic acid (NMDA), which can modulate memory by enhancing neuroplasticity and facilitating memory consolidation, or using neuromodulation techniques, such as deep brain stimulation (DBS) (164,165). D-cycloserine (DCS) functions as a partial agonist of the N-methyl-D-aspartate (NMDA) receptor, potentially improving the outcomes of fear extinction and exposure therapy. It achieves this either by boosting NMDA receptor activity during the extinction process or by suppressing its function during the initial consolidation of fear memories (166).

Previous research using animals demonstrated the positive result of DBS on memory enhancement (167). The hippocampus is the first region to undergo atrophy in dementia such as Alzheimer's disease (AD). The hippocampus is a primary target for MMTs. DBS is currently being refined as a clinical intervention for the early, preclinical stages of the disease (168-170). This technology is moving rapidly from laboratory research to human application, with several clinical trials already assessing its safety and efficacy. The hippocampus of the brain acts as a relay station for turning short-term perceptions into long-term memories. In AD this area is uniquely vulnerable due to deposition of neurofibrillary tangles and amyloid plaques (171). Volume loss involves the visible shrinking (atrophy) that is associated with the severity of memory loss (172).

Despite its promise, DBS faces significant hurdles. Phase II clinical trials for AD have yielded inconsistent results, while patients >65 years of age exhibited a temporary attenuation in cognitive decline, these improvements typically vanished after 12 months, with overall memory continuing to deteriorate (173). Alarmingly, research suggests that applying DBS to patients <65 years of age may inadvertently accelerate memory impairment (174). Furthermore, research has documented paradoxical memory deficits following stimulation in specific contexts. While non-invasive neuromodulation is being explored as an alternative, DBS currently retains an advantage in spatial and temporal specificity, offering more precise control over neural circuits (175,176). However, this lead is being challenged by emerging technologies like focused ultrasound, which can non-invasively stimulate or suppress deep-brain activity with high accuracy (177). Despite these technological leaps, several foundational challenges remain. i) Unknown mechanisms: The precise cellular mechanism that drive memory changes during stimulation are not yet fully understand. ii) Limited efficacy: Current outcomes are often modest and vary significantly between individuals. iii) Developmental barriers: Both invasive and non-invasive methods face rigorous regulatory and safety hurdles that slow their translation to clinical use. Nevertheless, these tools represent a frontier in cognitive science, offering the potential to enhance human memory far beyond the capabilities of traditional mnemonic strategies.

Modern strategies for memory improvement often target synaptic plasticity through the stimulation of neurogenesis or synaptogenesis. Other approaches focus on the modulation of neurotransmitter systems, specifically their transmission and receptor sensitivity, or the disinhibition of neural circuits essential for memory formation. The most widely accepted mechanism for these interventions is LTP. This process involves a sustained increase in synaptic strength triggered by the simultaneous activation of pre- and post-synaptic neurons. Theoretically, MMTs could be engineered to improve the efficiency of LTP, thereby sharpening overall cognitive performance.

A significant hurdle in developing these technologies is the lack of anatomical and functional specificity. As LTP occurs across virtually all excitatory pathways in the hippocampus, a generalized boost to this process could lead to unintended consequences: i) Memory overgeneralization: Instead of enhancing only useful information, MMTs may inadvertently strengthen every sensory input, rendering it difficult for the brain to distinguish important memories from trivial noise. ii) Pathological reinforcement: Global increases in plasticity can exacerbate conditions such as addiction, where drug-related cues become hyper-salient, or anxiety disorders, where traumatic associations are strengthened rather than extinguished. iii) Cognitive clutter: Enhancing synaptic excitability indiscriminately may interfere with the natural ability of the brain to prune unnecessary connections, potentially leading to cognitive interference.

The current research progress in memory-related neuroscience has transitioned from theoretical observation toward the active modulation of mnemonic traces. Recent experimental successes in animal models, specifically regarding the induction of false memories (178,179) the suppression or silencing of specific engrams (180,181), and the recalibration of emotional valences (182), signal a forthcoming experiment toward clinical human applications. However, as the scientific community pursues these cognitive optimizations through rigorous experimentation, the technical feasibility of memory manipulation must be balanced against its profound ethical ramifications (183). As MMTs, ranging from pharmacological

drugs to neural implants, move from theory to reality, they raise several critical ethical dilemmas. The core of the debate is a conflict between two major philosophical perspectives: i) Transhumanism: This argues that individuals have a moral obligation to use technology to overcome biological limitations and improve human well-being (184). ii) Bioconservatism: This argues that humans should preserve human nature and that essential cognitive changes risk destroying the important things that make life meaningful (e.g., vulnerability, effort and authentic history).

11. Exercise, health and memory

Regular physical activity can be considered a key lifestyle factor that enhances neurocognitive functions and brain plasticity. It may also help reduce the risk of cognitive decline linked to AD (185).

Research in animals consistently demonstrates that regular exercise promotes the growth of new brain cells, a process called neurogenesis, in the adult hippocampus. This, in turn, improves learning and memory (186). While neurogenesis in the adult human hippocampus has been frequently suggested (161,187), it has also faced recent questioning (159).

A growing body of evidence indicates that hippocampal synaptic plasticity (the ability of brain connections to strengthen or weaken) is at least partly influenced by BDNF (188). Physical exercise increases gene expression of BNDF (mRNA and protein levels of BDNF) in the hippocampus and other brain areas (188). Conversely, in the event that the action of BDNF in the hippocampus is blocked, the positive effects of exercise on memory are hindered (189).

The changes in BDNF levels related to exercise are typically rapid and temporary (190). Specifically, BDNF levels rapidly increase in hippocampal subfields following exercise, along with improvements in LTP and synaptic plasticity (191,192). These rapid changes may contribute to memory enhancement that lasts for a few hours.

Regular physical activity is a powerful tool for improving brain function. It boosts memory and strengthens synaptic plasticity within the hippocampus, a brain region critical for learning and memory. Exercise also increases levels of BDNF, a protein vital for neuron growth and survival.

Even short bursts of exercise, known as acute exercise, have been shown to benefit hippocampal plasticity in rodents. This is partly due to the release of endocannabinoids, particularly anandamide (AEA), and an increase in BDNF. However, it has not been clear if a single exercise session has similar effects on BDNF and AEA levels in humans, and if these changes, in turn, affect memory. To investigate this, a previous study was conducted using blood biomarkers, behavioural tests, and fMRI measurements in healthy male volunteers (193). The researchers assessed how a single exercise session impacted associative memory and the underlying brain mechanisms. Each participant underwent memory tests after three different conditions: rest, moderate-intensity exercise, or high-intensity exercise. A long-term memory retest was conducted three months later. The findings were significant: Both immediately after the exercise and at the three-month retest, memory performance was better after moderate-intensity exercise compared to rest. This improvement in memory after moderate exercise was linked to an increase in both AEA and BDNF levels. Specifically, AEA was associated with hippocampal activity during memory recall, while BDNF was found to enhance hippocampal memory representations and long-term memory performance (193). These results demonstrate that a single session of moderate-intensity exercise can indeed improve the consolidation of hippocampal memory. That study suggests that the signalling pathways involving endocannabinoids and BDNF likely play a synergistic role in modulating the underlying neural plasticity mechanisms of the brain (193).

12. Association between diet and memory

The connection between diet and memory is bidirectional. Consuming a high-calorie diet and becoming obese can harm both interoceptive processing and memory function in the hippocampus of humans and rodents. These negative changes in brain function can then contribute to even more excessive eating, creating a self-perpetuating cycle that makes it difficult to maintain a healthy weight (194).

There is a clear, bidirectional association between eating habits and memory. Research in both humans and rodents shows that a high-energy diet and obesity impair cognitive abilities, specifically memory and the function of the hippocampus (195). This cognitive decline may, in turn, lead to continued overconsumption, thereby initiating a harmful cycle that encourages unhealthy weight gain.

Research in mice has identified specific neural circuits that suppress food intake, originating from both the ventral hippocampus (196) and dorsal hippocampus (197) and projecting to different septal nuclei. Since the vHC is primarily linked to emotional and food-related memories and the dHC to episodic and spatial memories, both regions likely contribute to the memory of an eating episode. This shared memory representation may then inhibit subsequent food consumption through both common and unique mechanisms. Furthermore, memory is not contained in a single area but is a composite of information from multiple brain regions, each contributing different aspects of the same experience (198,199). While most research in rodents focuses on the hippocampus, other areas within the 'core memory network' may also connect memory and eating. Future studies should particularly investigate areas, such as the angular gyrus, that are altered in human obesity (200).

The dried root of *Rhodiola rosea* (*R. rosea)* was previously found to improve the ability of an insect to link odours and tastes with rewards, and it also prevented the typical memory decline associated with aging in adult flies. When administered to larval *Drosophila*, dried *R. rosea* root material improved their odour-taste reward associative memory in a dose-dependent manner (201). It was also shown to prevent a similar, age-related decline in this type of appetitive memory in adult flies. The substance not only improved memory scores in larvae but also mitigated the decline of this memory as the adult flies aged (201).

A recent study investigated whether the human tendency to remember the location of high-calorie foods more accurately than low-calorie foods is consistent across different cultures. Researchers conducted an online spatial memory test with participants from the USA and Japan, and compared the results to a previous study with a Dutch population (202).

The findings revealed that individuals in all three countries displayed this high-calorie memory bias. That study concluded that the strength of this cognitive trait is similar across diverse populations and is not significantly influenced by sociodemographic factors (202).

Dietary magnesium (Mg^{2+}) can improve memory in both young and aging rats, primarily by increasing synaptic density in the hippocampus and raising the expression of the NR2B subunit of the NMDA receptor (203). A recent study in fruit flies (*Drosophila*) found that magnesium also enhanced their long-term memory. Notably, this effect in flies appears to be independent of NMDA receptors in the mushroom body (204). Instead, it relies on a conserved Mg^{2+} efflux transporter encoded by the *unextended (uex*) gene. This gene is crucial for normal memory function and contains a cyclic nucleotide-binding domain. The location of UEX is altered in flies with mutations in cAMP-related genes, which are known to cause memory deficits. Imaging suggests that UEX-dependent efflux is necessary for the rhythmic maintenance of Mg^{2+} in Kenyon cells (204). This research suggests that regulated neuronal magnesium efflux is vital for both normal memory and memory enhanced by magnesium supplementation.

13. Conclusion and future directions

The conclusion of modern memory research highlights that memory is both a biological event (involving synaptic plasticity and the hippocampus) and a cognitive feat. Memory is supported by specialized circuits; the hippocampal-cortical network directs declarative memories (facts and events), whereas the auditory cortex, basal ganglia, and cerebellum facilitate non-declarative skills and habits. Math skills are more associated with declarative memory compared to nondeclarative memory. The auditory cortex plays a more significant and prominent role in emotional learning, particularly from the early stages of memory encoding and through its connections with subcortical nuclei amygdala. The role of the histamine neurotransmitter in emotional memories has been evaluated. The intricate workings of the hippocampus and entorhinal cortex navigate the spatial dimensions of one's world. The hippocampus and entorhinal cortex generate the spatial coordinates of the map. The parahippocampal and retrosplenial cortices integrate maps to stable physical landmarks, and these spatial codes are integrated with frontal lobe processes to transform a mental map into a navigation plan.

Despite significant advances, a number of gaps and future possibilities remain. While there is a strong understanding of how the hippocampus lays down new memories, the precise mechanisms by which they are consolidated and transferred to the neocortex for long-term storage are still being elucidated. Researchers are also finding the complexity between emotion and memory, moving beyond general associations to understand how specific emotional states, such a fear, can compress or distort our perception of time and space in memory. Furthermore, the role of sleep in memory consolidation is an emerging field, with new research confirming its critical function in solidifying learned information and skills.

In the case of short-term memory, brief bouts of cardiovascular exercise (referred to as acute exercise) exert a moderate positive effect. Acute exercise and long-term exercise, both types of exercise, can benefit short-term memory. Acute exercise may be particularly effective for boosting long-term memory (205). Researchers have also explored whether any exercise-induced changes in BDNF and VEGF levels are linked to memory improvements (206). However, how acute exercise affects emotional memory is less clear. Acute exercise appears to be linked to increased memory confidence and accuracy for the most important details of emotional stimuli. Additionally, certain exercise types, such as treadmill exercise, are associated with a higher occurrence of memory intrusions. Aerobic exercise training can indeed reverse hippocampal volume loss in late adulthood, and this positive change is accompanied by improved memory function. It is a powerful reminder of how movement can directly impact brain health. Acute exercise appears to be linked to increased memory confidence and accuracy for the most important details of emotional stimuli. Additionally, certain types of exercise, such as treadmill exercise, are associated with a higher occurrence of memory intrusions (207). The hippocampus, a crucial brain region for memory, naturally shrinks (84). This decline can lead to impaired memory and a heightened risk of conditions like dementia (208).

Advanced neuroimaging techniques will continue to map the dynamic brain networks responsible for different memory types in real-time. As of early 2026, researchers are trying to analyse the intersection of neuro-engineering, AI and cognitive psychology. Researchers are finding ways to write and edit memories using high technology tools such as optometry. Using light to activate specific neurons (engrams), researchers can now activate or suppress specific memories in animal models. Future research is required to focus on translating these findings into non-invasive therapies for humans with post-traumatic stress disorder or severe phobias. A previous study revealed that slow gamma oscillations (40 hertz) are associated with the retrieval of spatial memory (209). The researchers used optogenetics in an Alzheimer model of mice to activate medial septal parvalbumin neurons and found that optogenetic stimulation restores hippocampal slow gamma oscillations amplitude, and phase-amplitude coupling in mice (210).

When a memory is recalled, it becomes labile (changeable) for a few hours. Research is investigating how to safely intervene during this window to reduce the emotional pain associated with traumatic memories. Extensive research is being conducted on how our reliance on smartphones (external memory) is physically reshaping our brains. A critical question is whether humans are losing storage capacity, but gaining retrieval efficiency. A new concept termed the 'Third Way of Memory' is emerging. Generative AI can create photos, videos, or chatbots of deceased loved ones. Researchers are studying how these 'simulated memories' affect the grieving process and our collective sense of history (211,212).

Brain-computer interfaces (BCIs) are being developed to monitor brain signals in real-time. For patients with AD, these devices could detect a 'memory lapse' and provide a tiny electrical pulse to help the brain bridge the gap. Clinical trials are exploring whether DBS techniques can enhance memory retrieval in patients with traumatic brain injuries or early-stage dementia. Emerging studies suggest our gut health directly

impacts neurogenesis in the hippocampus. Future research is looking into 'psychobiotics' dietary interventions designed specifically to boost memory and slow cognitive decline.

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Authors' contributions

RY and SK conceptualized the study and prepared the original draft of the manuscript. AAC and RM reviewed and edited the manuscript. RY and SK supervised the study. RY was involved in figure illustration. All authors have read and agreed to the published version of the manuscript. Data authentication is not applicable.

Ethics approval and consent to participate

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Competing interests

The authors declare that they have no competing interests.

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