

EDWARD  
ABBOTT  
PORTFOLIO

Edward Abbott

[ilmareillustration.business@gmail.com](mailto:ilmareillustration.business@gmail.com)

@ilmare.illustrations on IG

Ilmareillustrations on Artstation



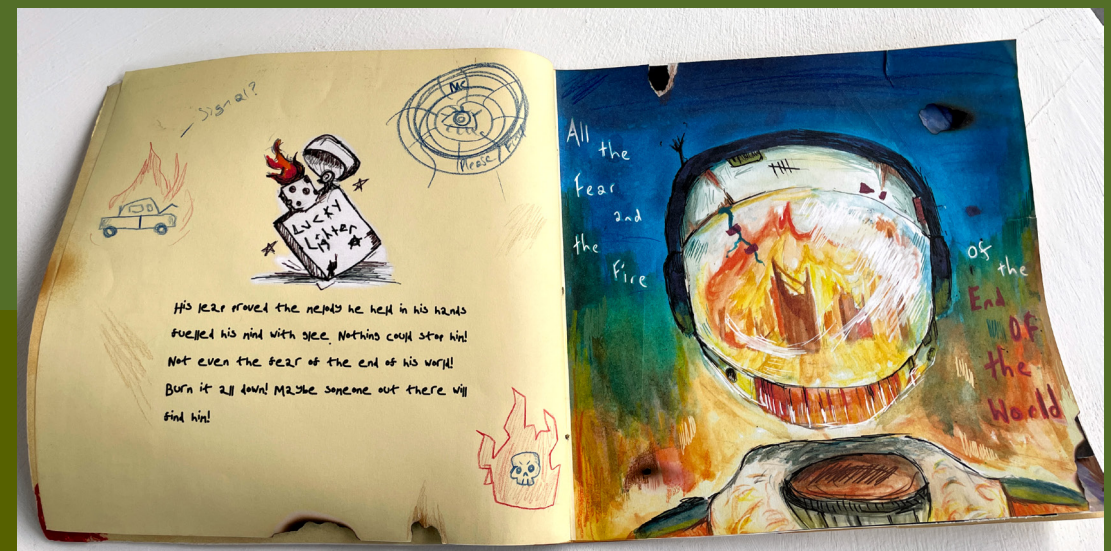
## Edward Abbott

Hey there! I'm a Cheshire based illustrator who aspires to make peoples wildest imaginations come true in my wild and free flowing art. From spray paints to watercolour, I can do it all, a true jack of all trades.

I love learning new skills and getting hands on with my work, favouring tradtional work anyone can cherish. I have an interest in comic arts, concept art in general, album covers and book illustration

# Apocalyptic Albums

Apocalyptic albums is a self authored brief that celebrates the emotions music can bring. It celebrates how music, moves, touches and drives people around the world to create and connect with humanity. The concept album uses real world music pieces to tell a narrative about a Space Ranger who has crash landed on a dead earth and is now finding their way through the lost world with music as their guide. The concept album celebrates connection and self discovery through music.



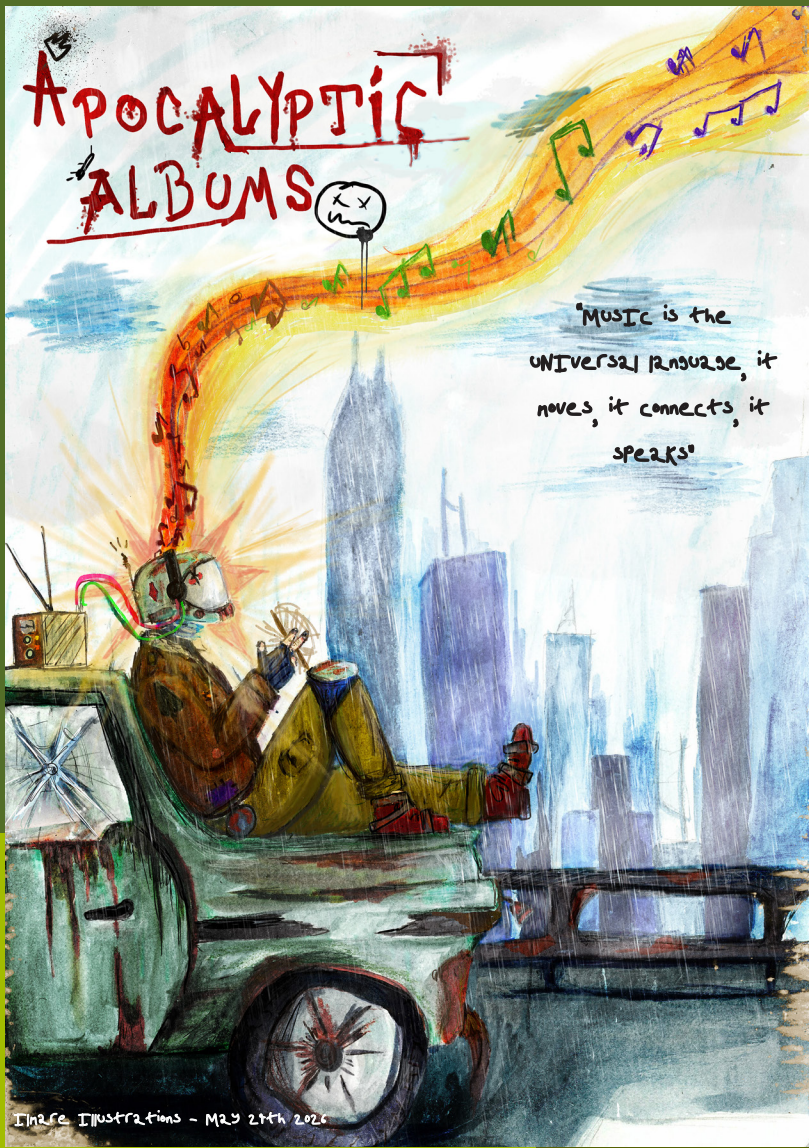
# Ilmare Helmet

This Helmet is an iconic part of the main character, used as a prop to promote album



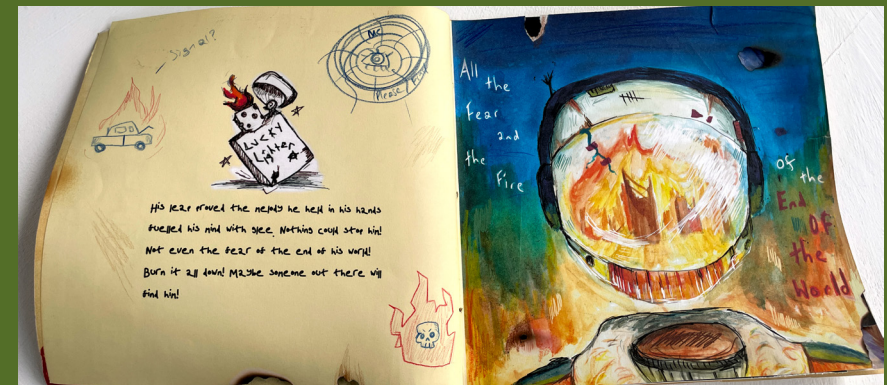
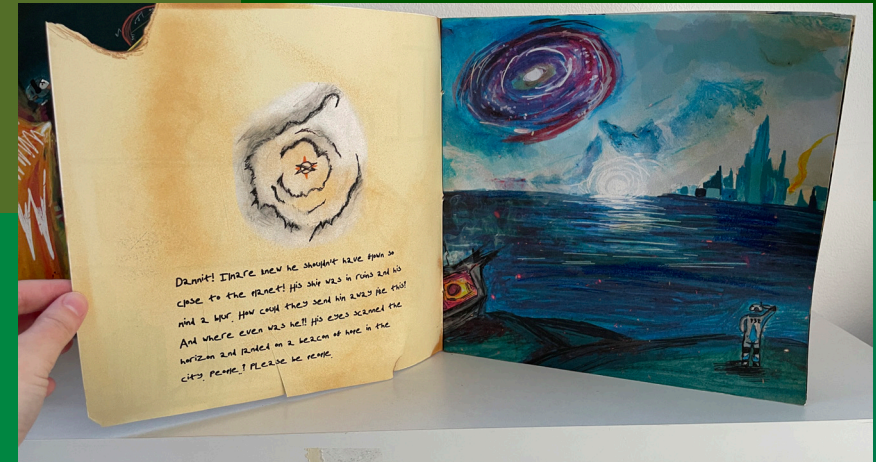
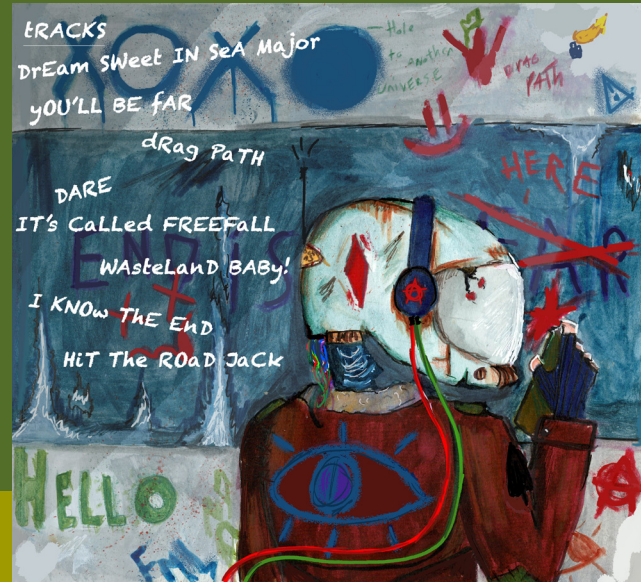
# Promotional Posters

Posters to promote Apocalyptic Albums



# Vinyl Sleeve, Vinyl and Lyric Book

This is the Record sleeve, the Lyric book and Vinyl. The lyric book is Ilmares journey with each song



# Book Cover and Character work

These pieces are my book choice for the Penguin Book Cover brief. I chose Lord of the Flies and created some character vignettes and chapter headers.

When a group of schoolboys are stranded on a desert island, what could go wrong? A plane crashes on a desert island. The only survivors are a group of schoolboys. By day, they discover fantastic wildlife and dazzling beaches, learning to survive; at night, they are haunted by nightmares of a primitive beast



Lord of the Flies

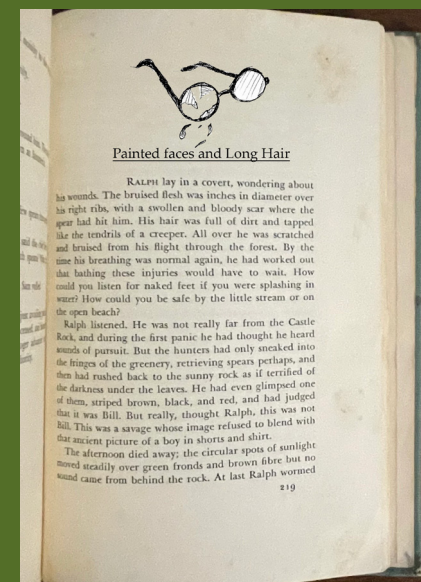
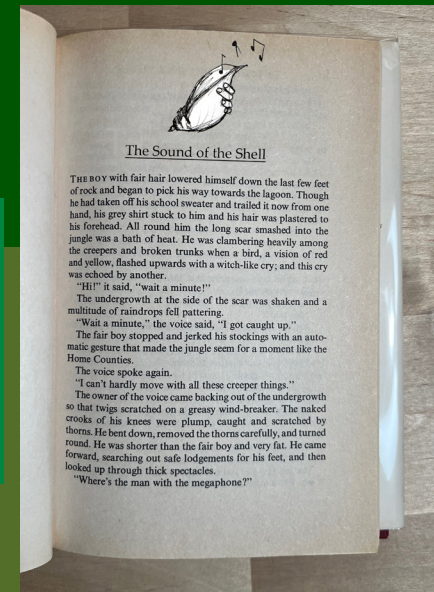
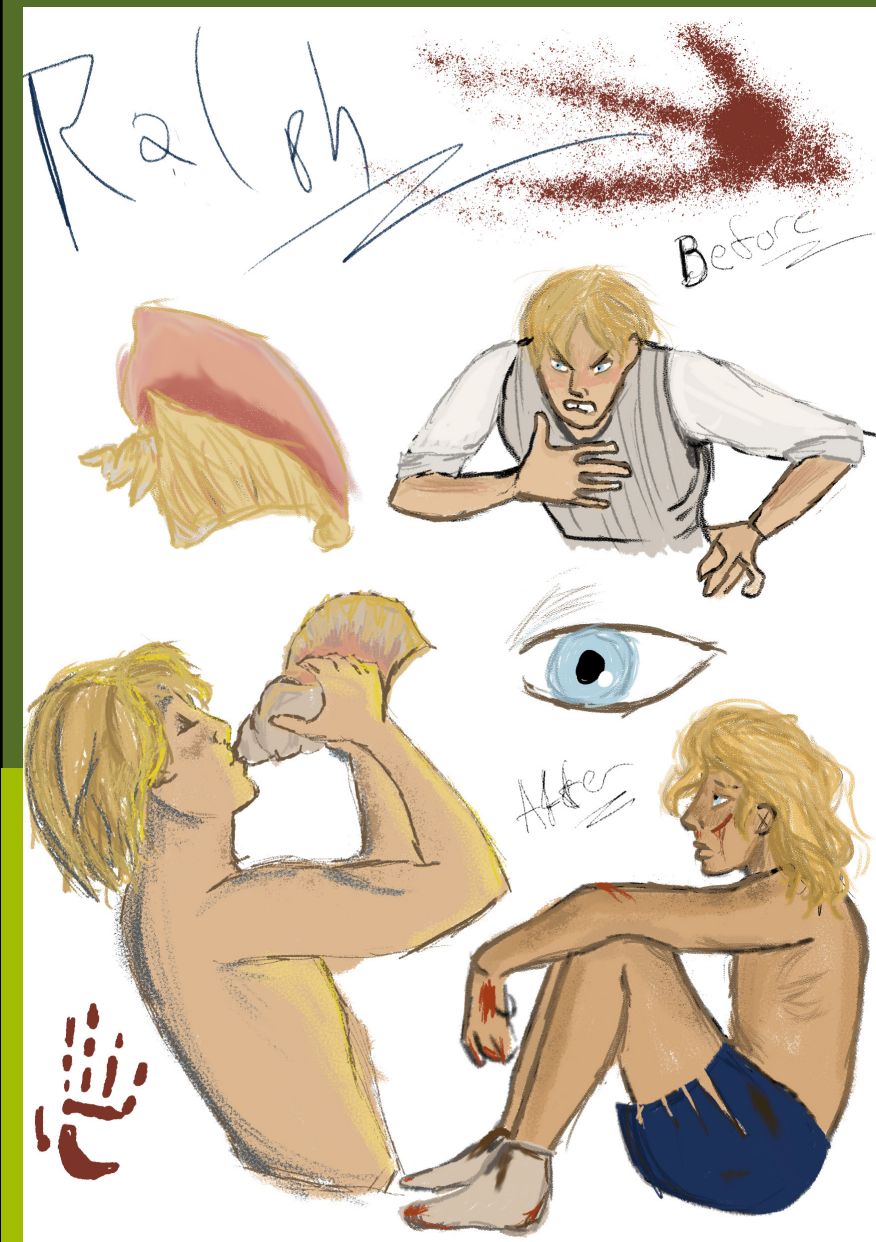
Lord of the Flies

William Golding



# Character Sheets and Chapter Headers

These are the character sheets for two of the main characters for Lord of the Flies and chapter headers in context to the book



These pieces are for a self led editorial brief on the effects of memory loss in young people. This brief mimics a double page magazine piece aimed to make young people with memory loss feel less alone.



## Memory Loss

A Scientific Review

Short-term memory is also called short-term storage, primary memory, or active memory. The term indicates different systems of memory involved in retaining pieces of information, or memory chunks, for a relatively short time, typically up to 30 seconds.

Although short-term memory is closely related to the concept of working memory, both are distinct entities. Short-term memory is a set of storage systems, whereas working memory indicates the cognitive operations and executive functions associated with the organization and manipulation of stored information. Nevertheless, the terms short-term memory and working memory are often used interchangeably. Short-term memory must also be distinguished from sensory memory, such as the acoustical echoic and iconic visual memories, which are shorter in duration, typically fractions of a second, and reflect the stimulus's original sensation or perception. In other words, sensory memory is specific to the stimulus modality of presentation. This raw sensory information undergoes processing and, upon processing to short-term, is expressed in a format different from that perceived initially. Hippocampal neurogenesis regulates the maintenance of long-term potentiation. However, the hippocampal network, including the parahippocampal gyrus, hippocampus, and neocortical areas, is not the storage site for memories but plays a crucial role in forming new memories and their subsequent reactivation. The hippocampus appears to have a limited capacity but acquires information quickly and automatically. Over time, the initially available information is permanent in other brain structures, particularly the cortex, independently from the activity of the hippocampus itself. The crucial mechanism of this transfer involves the reactivation (replay) of neural activity configurations.

Different clinical conditions, including strokes, brain aneurysms, traumatic

brain injuries, primitive or metastatic neoplasms, and infectious diseases, may impair various components of short-term memory. However, the damage to short-term memory is seldom selective. For example, aneurysm rupture can lead to short-term and long-term memory loss. Apart from diseases that induce short-term memory alterations through direct neural damage, a wide range of medical conditions such as systemic infections; thyroid diseases; surgery, including neuroinflammation-mediated postoperative delirium and postoperative cognitive dysfunction; or psychiatric diseases, including depression; or psychological trauma can also impact short-term memory. In this regard, evidence shows that violence during childhood impairs cognitive processes, including memory (psychogenic amnesia). Cancer treatments, including radiation and chemotherapy, can induce short-term damage through a complex neuroinflammation mechanism.

### THE AMNESTIC SYNDROME

The amnesic syndrome is an impairment in the ability to form new memories. Regardless of its etiology, the memories of recent events are particularly vulnerable, whereas long-term memory is more resilient and protected from damage. This concept is not new; towards the end of the 19th century, the French psychologist Théodule-Armand Ribot (1839-1916) demonstrated that amnesia affects memories in reverse order of their development. [This idea underscores the importance of distinguishing between anterograde and retrograde amnesia. The former concerns the new memories, whereas the latter is well-established by neural networks. This distinction explains why some conditions involving drugs,

such as benzodiazepines, can lead to a deficit, especially within short-term memory. In turn, amnesic disorders primarily affect anterograde memories. A recent study on short-term memory binding has identified amnesic mild cognitive impairment as the most typical preclinical stage of Alzheimer disease. Neuropsychological measures are relatively sensitive and specific diagnostic tools for amnesic mild cognitive impairment, although they do not replace the utility of biomarkers.

### EVALUATION AND TREATMENT

Several strategies have been proposed to enhance memory tasks, including behavioral and non-behavioral approaches. Behavioral approaches, or cognitive training, are mostly focused on the maintenance rehearsal, which facilitates memory processing through repetitive stimuli, and the elaborative rehearsal, which entails associating new information with stored knowledge and analyzing it. Several proposed behavioral strategies include the combination of Tai Chi movements and breathing. However, further research and controlled studies are required to confirm their real benefit. Concerning pharmacological approaches, although commonly prescribed, drugs approved to manage memory symptoms of Alzheimer disease, such as cholinesterase inhibitors, have not demonstrated significant benefits in slowing down or preventing the progression of mild cognitive impairment of Alzheimer disease.

# Editorial - Spot Illustrations

These spot illustrations would go across the pages following the core piece of the article

**The Amnesic Syndrome**  
The amnesic syndrome is an impairment in the ability to form new memories. Regardless of its etiology, the memories of recent events are particularly vulnerable, whereas long-term memory is more resilient and protected from damage. This concept is not new; towards the end of the 19th century, the French psychologist Theodule-Armand Ribot (1839-1916) demonstrated that amnesia affects memories in reverse order of their development. This idea underscores the importance of distinguishing between anterograde and retrograde amnesia. The former concerns the new memories, whereas the latter is well-established by neural networks. This distinction explains why some conditions involving drugs, such as benzodiazepines, can lead to a deficit, especially within short-term memory. In turn, amnesic disorders primarily affect anterograde memories. A recent study on short-term memory binding has identified amnesic mild cognitive impairment as the most typical preclinical stage of Alzheimer disease. Neuropsychological measures

are relatively sensitive and specific diagnostic tools for amnesic mild cognitive impairment, although they do not replace the utility of biomarkers. Several strategies have been proposed to enhance memory tasks, including behavioral and non-behavioral approaches. Behavioral approaches, or cognitive training, are mostly focused on the maintenance rehearsal, which facilitates memory processing through repetitive stimuli, and the elaborative rehearsal, which entails associating new information with stored knowledge and analyzing it. Several proposed behavioral strategies include the combination of Tai Chi movements and breathing. However, further research and controlled studies are required to confirm their real benefit. Concerning pharmacological approaches, although commonly prescribed, drugs approved to manage memory symptoms of Alzheimer disease, such as cholinesterase inhibitors, have not demonstrated significant benefits in slowing down or preventing the progression of mild cognitive impairment to Alzheimer disease. Different clinical conditions, including

strokes, brain aneurysms, traumatic brain injuries, primitive or metastatic neoplasms, and infectious diseases, may impair various components of short-term memory. However, the damage to short-term memory is seldom selective. For example, aneurysm rupture can lead to short-term and long-term memory loss. Apart from diseases that induce short-term memory alterations through direct neural damage, a wide range of medical conditions such as systemic infections; thyroid diseases; surgery, including neuroinflammation-mediated postoperative delirium and postoperative cognitive dysfunction; or psychiatric diseases, including depression; or psychological trauma can also impact short-term memory. In this regard, evidence shows that violence during childhood impairs cognitive processes, including memory (psychogenic amnesia). Cancer treatments, including radiation and chemotherapy, can induce short-term damage through a complex neuroinflammation mechanism.<sup>6</sup>



Recent reviews reported impairments of executive function in primary progressive aphasia. Executive functions pertain to a set of cognitive processes, specifically shifting, inhibition, and updating of working memory, and are involved in the cognitive control of behavior. Results revealed that the primary progressive aphasia variant and disease duration were significant moderators of performance, whereas task modality and years of education were not. The non-fluent or agrammatic primary progressive aphasia and the logopenic primary progressive aphasia variants were similarly affected, but the semantic variant was affected to a lesser extent.<sup>[21]</sup> Another study described the semantic variant of primary progressive aphasia as a progressive loss of semantic knowledge impairing the ability to name and recognize the meaning of words, which can be improved through learning therapy.

Other conditions that impair memory tasks include alcohol and drug abuse, such as marijuana; heavy cigarette smoking; sleep deprivation; severe stress; and vitamin B12 deficiency. Prolonged alcohol intake can lead to Korsakoff syndrome, which is a complex amnesic disorder with neuropsychological sequelae caused by vitamin B1 (thiamine) deficiency. In addition to alcohol, other causes can lead to vitamin B1 deficiency (non-alcoholic Korsakoff syndrome) with related memory disorders, including dietary deficiencies, prolonged vomiting, and eating disorders. Korsakoff-like amnesic syndromes have also been observed after brain lesions involving the anteromedian thalamus and hippocampus. Recent reviews have emphasized the crucial role of the prefrontal cortex in mediating executive functions and organizing a person's thinking, decision, and behavior, particularly to stress. Stress activates the hypothalamic-pituitary-adrenal axis, releasing prefrontal neurotransmitters, with dopamine neurotransmission responding as a stress modulator. Different reviews showed that stressful events are associated with increased dopamine concentrations in the medial prefrontal cortex. This increase affects working memory by causing an inability to process information selectively and impairing cognitive function. Among other causes of memory impairment, a common adverse effect of electroconvulsive therapy is short-term memory alteration during treatment. Another notable concern is medication-induced memory loss. The list of drugs implicated includes benzodiazepines, antiepileptic drugs, opioids, and tricyclic antidepressants. Limited evidence, primarily from observational data and case reports, is available regarding statin use and memory loss. Most of these drugs, such as benzodiazepines, impair memory processing and, in turn, can present an obstacle to the consolidation of information.

In particular, memory loss without interference in daily life or independent function is the main feature of mild cognitive impairment, which represents the stage between the expected cognitive decline of normal aging and the more severe decline observed in Alzheimer disease. A recent study showed that the difference between mild cognitive impairment and visual short-term memory from normal cognitive aging is evident only where the spatial configuration of stimuli is retained at original locations. Findings also revealed a reduced ability to inhibit irrelevant items and location priming (by repetition) deficits. However, visual short-term memory for simultaneous items declines significantly in normal aging but is not influenced differently by spatial or object configuration change. Moreover, alterations in different memory domains have been shown in Parkinson disease, in those affected by Huntington disease, and in primary progressive aphasia. Several strategies have been proposed to enhance memory tasks, including behavioral and non-behavioral approaches. Behavioral approaches, or cognitive training, are mostly focused on the maintenance rehearsal, which facilitates memory processing through repetitive stimuli, and the elaborative rehearsal, which entails associating new information with stored knowledge and analyzing it. Several proposed behavioral strategies include the combination of Tai Chi movements and breathing. However, further research and controlled studies are required to confirm their real benefit. Concerning pharmacological approaches, although commonly prescribed, drugs approved to manage memory symptoms of Alzheimer disease, such as cholinesterase inhibitors, have not demonstrated significant benefits in slowing down or preventing the progression of mild cognitive impairment to Alzheimer disease.

