

THE IMPACT OF ACUTE AND CHRONIC STRESS ON MEMORY ENCODING AND RETRIEVAL IN STUDENTS

¹. Dr.N. Padma Ramachandran, ². Abhishek P.R, ³. Dr.Archana P.R,

¹*MD,PGDFM,DFID(CMC Vellore) Professor and HOD of Physiology, PIMS, Walayar, India, padmaramc@yahoo.co.in

²JCET Student, Palakkad, India, prabhishek71@gmail.com

³Nila Dental Studio, Chennai, India, archusrihere@gmail.com

***Corresponding Author:**

Abstract

Stress is a very important factor in the development of cognitive functioning, especially the memory encoding and retrieval processes, which are vital to academic achievements. The review will assess the effects of acute and chronic stress on memory processes in students via the synthesis of neurobiological, psychological, and educational facts. Stress may occur as a result of inherent mental or external environmental problems that stimulate the sympathetic-adrenal-medullary axis and the hypothalamic-pituitary-adrenal axis. Although acute stress can be temporarily alert-enhancing and promote memory encoding, chronic stress, which is characterized by sustained cortisol elevation, affects neural plasticity in an adverse way, impairs the functioning of the hippocampal and prefrontal areas, and impairs the process of consolidation and retrieval. Literature by students has shown that academic pressure, test anxiety, and prolonged workload usually lead to retrieval failure, cognitive exhaustion, and poor academic outcomes. The thematic analysis also promotes the differences between individuals concerning age, coping capacity, sociocultural factors, and the possible positive impact of mild stress (eustress). With the help of questionnaires, medical histories, and standardized memory tests, there is evidence that stress-relief approaches include mindfulness, cognitive-behavioral methods, sufficient sleep, and structured relaxation methods, which can restore cognitive abilities. On the whole, the results highlight the necessity of integrated stress-management systems to facilitate the well-being of students and achieve the maximum learning success.

Keywords: Stress; Acute stress; Chronic stress; Memory encoding; Memory retrieval; Cortisol; Cognitive functioning; Students; Academic performance; Stress management

1. Introduction

Stress is a universal psychophysiological reaction that occurs when individuals feel that they have environmental demands beyond their adaptability scope. Stress has recently gained real popularity among students, as one of the key determinants of cognitive performance, academic achievement, emotional health, and mental well-being in the long term. Academic life puts students in a complicated set of stressors, including exams, deadlines, competition, social demands, financial demands, and personal doubts, all of which influence the way they encode, store, and re-access information in the process of learning activities. Since memory has been the key factor in academic operation, the positive impacts of stress on memory encoding and recalling are a critical consideration for enhancing learning performance, mental health, and avoiding cognitive decline in the long run. Stress can be classified into acute and chronic, and the two types have greatly dissimilar impacts on cognitive functioning. Acute stress is temporal, and in most cases, it comes as a result of immediate difficulties like a pending exam or a sudden intellectual task. It has been found that mild to moderate acute stress may also raise alertness, enhance attentional attention, and in certain instances lead to memory encoding as a result of brief activation of the sympathetic nervous system and temporary discharge of stress hormones like adrenaline and cortisol (Joels et al., 2006). On the contrary, chronic stress is the result of sustained or recurrent or severe exposure to challenging conditions habit which is common with students under undying academic stress. Long periods of stress are also detected by increased cortisol levels, impair neural plasticity, impair hippocampal functioning, and disrupt prefrontal cortex processes that are critical to working memory and retrieval accuracy (Lupien et al., 2009). Cognitive neuroscience has shown that the encoding and retrieval levels of memory are largely dependent on the integrity of the interrelations among the interconnected neural structures, especially the hippocampus, amygdala, and prefrontal cortex. The systems are structurally and functionally impacted by stress. Indicatively, chronic stress has been linked to hippocampal atrophy, less synaptic connectivity, and impaired neurogenesis, which result in impaired new memory formation (McEwen and Morrison, 2013). On the other hand, the amygdala, which is activated by emotional arousal, is able to augment the consolidation of emotionally aroused memories in acute stress. This duality has served to emphasize the intricacy of stress and memory interactions and why varied forms of stress have different effects on students. In education, the impairment of memory retrieval is particularly vulnerable to the influence of stress. High anxiety in an exam, say, can inhibit access to already acquired knowledge even in cases where the students have sufficient knowledge. Research indicates that test anxiety interferes with executive attention and the working memory resources, reducing the retrieval performance among high-pressure tasks (Owens et al., 2014). Such retrieval failures tend to sustain negative academic cycles, which further increase stress and add to cognitive decline. Hence, it is essential to learn how stress modulates the memory processes so as to come up with effective educational and psychological interventions. In addition, it has been shown that the relationship between stress and memory at different developmental stages is different. The stress might be stronger in younger students due to the mechanism of cognitive control development, and adolescents are subject to social and academic pressure that increases stress sensitivity. In the meantime, university students tend to deal with multifaceted environmental, mental, and lifestyle-related stressors. The studies have shown that stress reactions vary with age, gender, coping mechanisms, and socioeconomic status, and there is a need to comprehend stress within a multidimensional context (Romeo, 2010). Although there is a substantial amount of research on the topic of stress and cognition, limited studies review the topic of stress and its impact on memory encoding and retrieval in student populations as a whole and as an interaction. The available literature points out positive and negative results, yet the results are scattered across neurobiological, psychological, and educational areas. Thus, the purpose of the review is to summarize existing evidence on the basis of the physiology of stress, cognitive mechanisms, and practical implications in academics. It also incorporates the results of the studies of behavioral, neuroendocrine, and neuroimaging to explain how various types of stress determine memory processes.

The purpose of this comprehensive review is threefold:

1. to delineate the physiological and neural mechanisms underlying the effects of acute and chronic stress on memory encoding and retrieval;
2. to examine the cognitive and academic consequences for students across educational levels; and
3. to evaluate stress-management interventions that restore or protect cognitive functioning.

This review offers a broad concept of the mechanisms through which stress influences learning and memory in groups of students by synthesizing the results of various methodologies, such as psychophysiological data, standardized memory tests, and the study of education.

2. Background and Theoretical Framework

The complex of acute and chronic stress effects on memory activity should be studied in a multidisciplinary manner through the prism of physiological, neurobiological, and cognitive mechanisms. Stress is essentially a biological response that prepares the body to address the perceived threat via a highly controlled neuroendocrine process. Although this system is adaptive in the short term, it may be maladaptive when sustained activation causes cognitive disruptions that are especially important in the case of students whose academic achievements rely on the ability to encode and retrieve information through memory.

2.1 Physiology of the Stress Response

Stress-related reactions mainly involve two interdependent systems in the body, including the sympathetic-adrenal-medullary (SAM) and the hypothalamic-pituitary-adrenal (HPA) axes. The SAM system is activated, leading to the secretion of catecholamines like adrenaline and noradrenaline that cause increased arousal, alertness, and mobilization of energy. This quick reaction system improves the capacity to cope with urgent problems, but when overstimulated, it

distorts cognitive control (Ulrich-Lai and Herman, 2009). The slower hormonal stress response is controlled by the HPA axis through the release of cortisol by the adrenal cortex. The role of cortisol in the functioning of the body is very important both in metabolism and the immune system, as well as the mental ability. Cortisol can boost some of the memory formation processes, especially in situations that involve emotional arousal and occur under acute stress. But chronic stimulation leads to a persistent increase in cortisol levels, which have been linked to hippocampal atrophy, impaired synaptic plasticity, and impaired working memory and executive functioning (Sapolsky, 2015). These physiological processes are the basis of the research on the effects of stress on cognition.

2.2 Neurobiology of Memory Systems

Memory is not unitary; it entails a variety of systems that encode, consolidate, store, and recall the information. The processes depend on the interaction of the activation of multiple brain systems, such as the hippocampus, prefrontal cortex (PFC), and amygdala. The hippocampus is vital in the formation of new declarative memories and consolidation of information into long-term storage, which is found in the medial temporal lobe. The executive functions that the PFC controls include working memory, planning, and retrieval strategies. The amygdala, in the meantime, regulates emotional memory and the hippocampus to reinforce events of emotional significance (Squire and Wixted, 2011).

All these regions are impacted by stress in different ways. Stress of an acute nature may increase the amygdala activation, which may, on the one hand, promote the encoding of emotionally charged information, but, at the same time, lead to the inhibition of the PFC functioning, thus making it more difficult to code the information neutral in a productive way. In its turn, chronic stress has been found to decrease the volume of the hippocampus and the dendritic branching, which impede the capacity to create new and recollect the old memories (Lupien et al., 2018). Such neurobiological variations support the significance of the stress factor to be considered as a multidimensional one affecting memory via different mechanisms.

2.3 Interaction Between Stress and Memory Systems

The correlation between memory and stress is non-linear and contingent. Yerkes Dobson law, which is one of the fundamental psychological theories, states that performance rises with physiological arousal up to an optimal level, where beyond this level physiological arousal causes performance to decrease (Diamond et al., 2007). It is possible to explain this model by the fact that mild acute stress can enhance memory encoding, whereas severe acute stress or acute stress lasting some time can lead to negative effects. Acute stress affects memory due to the release of catecholamines and temporary cortisol bursts that can be useful in memorizing emotionally related information. These same routes are, however, capable of interfering with retrieval processes. Cortisol disrupts the ability of the PFC to form a working memory and blocks hippocampal retrieval brain circuits, hence the reason why students who make exam mistakes tend to forget previously learned information (Schwabe and Wolf, 2010). Stress that is chronic stress has even more dire and long-term effects. Chronic high levels disrupt long-term potentiation (LTP), a cellular process required to consolidate memories. In the long run, this may cause loss of synapses and structural impairment in hippocampal circuits. Amygdala-PFC connectivity is also dysregulated by chronic stress, which results in increased emotional reaction and diminished cognitive adaptability (Kim et al., 2015). These results add to the idea that acute stress is not always harmful, but in fact, chronic stress can be very dangerous to the functioning of the memory.

2.4 Relevance to Student Populations

Acute and chronic stressors common to students include, but are not limited to, the pressure of exams, academic demands and pressures, social expectations, family issues, and monetary pressures. These stressors can cause immediate arousal of the body and also long-term stimulation of the HPA axis. When teaching, as a psychologist or a policymaker, it is important to understand the theoretical background of stress and memory in order to help students be healthier and improve their performance in school. Figure 1 is a conceptual model that demonstrates the three main pathways of stress effect on memory processes: physiological arousal, neuroplasticity, and executive regulation.

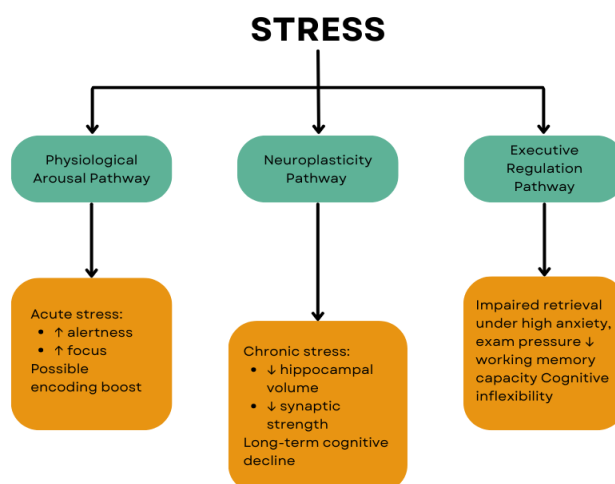


Figure 1. Conceptual Model of Stress Pathways Affecting Memory

3. Materials and Methods

It was a review with a narrative and integrative approach to the synthesis of the existing evidence on the impact of acute and chronic stress on memory encoding and retrieval in students. The search involved searching through the key academic databases, such as PubMed, Scopus, Web of Science, PsycINFO, and Google Scholar. The keywords like “acute stress,” “chronic stress,” “memory encoding,” “memory retrieval,” “students,” “cortisol,” and “cognitive performance” were searched in different combinations to bring relevant literature. The search was limited to peer-reviewed articles that were published mainly in the last two decades that to include modern neurobiological and psychological knowledge.

Inclusion criteria used were that the study had to test stress and memory correlation in children, adolescents, or adult students, or had to explicitly apply underlying mechanisms to academic situations. Articles were eliminated when they focused on the neurological diseases, traumatic brain injuries, or psychiatric disorders, the ones not connected with stress. The English-language publications that had available full texts were taken into account. All studies were checked in terms of their purposes, sampling, stress system, and memory tests. The stress paradigms, which were both laboratory assessed, like the Trier Social Stress Test, and the naturally occurring academic stressors, were also encompassed.

The thematic analysis of data in the case of selected studies was performed. Results were summarized into conceptual domains such as the syndrome of physiological responses to stress, acute stress effects, chronic stress consequences, and implications for memory encoding and retrieval. Such thematic synthesis made it possible to reveal convergent trends and gaps in the literature. The evidence used in the review was sourced from behavioral tests, neuropsychological investigations, cortisol levels, and neuroimaging examinations to have a multidimensional approach. The general objective of this methodology was to come up with a cohesive perception of the effects of stress in the context of memory in the student populations and a platform on which one could further analyze and discuss the situation.

4. Thematic Review and Analysis

4.1 Theme 1: Acute Stress and Memory Encoding

Acute stress is a temporary physiological, psychological reaction to an urgent demand or threat. In school, the same may happen during tests, unexpected tests, or any time in a stressful classroom situation. Acute stress stimulates the sympathetic-adrenal-medullary (SAM) system and results in the immediate release of catecholamines, elevating the physiological arousal of the body and enhancing the attentional systems. Such fast neurobiological adaptations can provisionally enhance encoding mechanisms, especially in cases where the arousal levels are moderate, in that they increase alertness and task participation. It has been shown that acute stress may also enable the encoding of emotionally salient or highly relevant academic information because of increased vigilance and communication between the amygdala and the hippocampus (Roosendaal and McGaugh, 2011).

Neurobiological functioning is also affected by a short-term cortisol increase in acute stress. Although an excessive amount of cortisol may impair the process of cognitive activity, moderate and temporary upsurges of cortisol can temporarily enhance the process of memory consolidation by regulating noradrenergic release in the amygdala and hippocampus. Research indicates that moderate levels of acute stress may improve recall of central information of an educational experience and may damage peripheral detail recall (Andreano and Cahill, 2006). One of the ways in which acute stress might enhance encoding accuracy in academic situations is by improving task focus.

Acute stress does not improve memory uniformly, though. It is also possible that high-intensity stress can interfere with encoding and shift the cognitive resources to the regulation of emotions instead of processing tasks. The state of anxiety related to exams can interfere with attentional control, decrease the size of working memory, and decrease the ability to encode newly introduced information. Experiments performed by students have shown that the performance becomes unpredictable and memory encoding is worse than in cases of optimal arousal levels (Vogel and Schwabe, 2016). Moreover, academic assessment-related anticipatory anxiety may reduce performance despite exposure to stress in a short period of time.

This duality of acute stress is supported by evidence from laboratory simulations that involved student participants. As an example, Trier Social Stress Test (TSST) induction of acute stress has been found to enhance memory of emotionally relevant stimuli and hamper the encoding performance of neutral learning tasks (Klier and Buratto, 2020). In this way, acute stress has a multifaceted effect on memory encoding, the results of which are determined by the intensity of stress, the complexity of the task, and the relevance of the information to emotions. Figure 2 juxtaposes the specific and common effects of acute and chronic stress on the memory system with reference to its cognitive impacts.

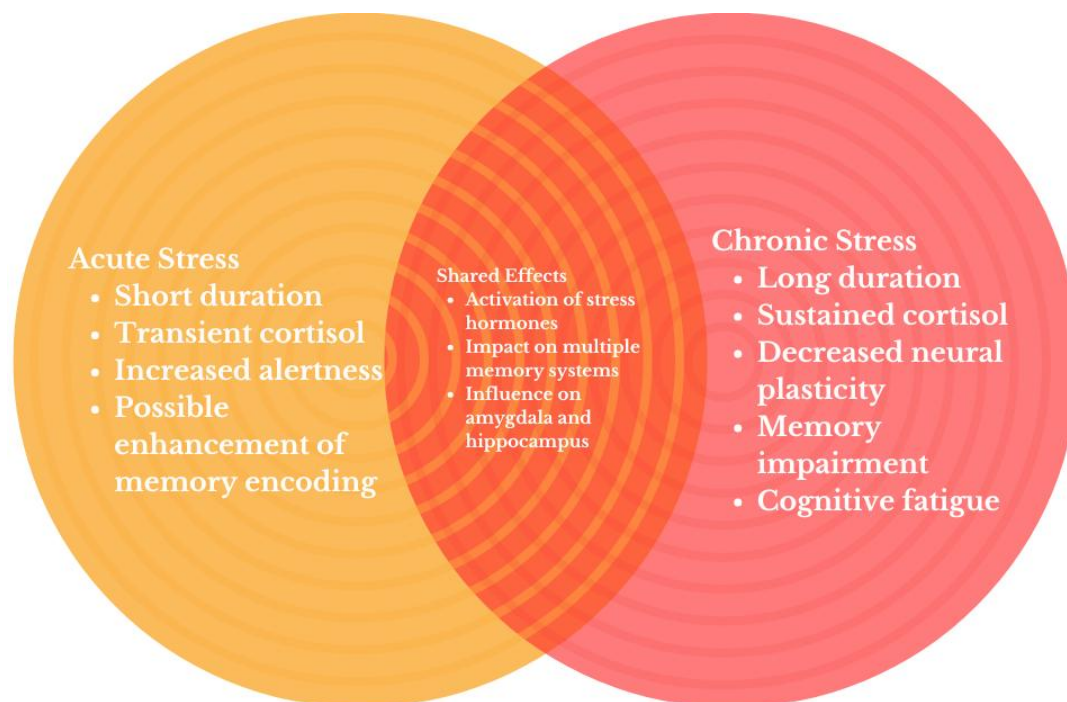


Figure 2. Comparison of Acute vs. Chronic Stress Effects on Memory

4.2 Theme 2: Chronic Stress and Long-Term Cognitive Impairment

Chronic stress is a chronic mental and physiological stress that develops as a result of stressors that lack adequate rest between occurrences. Such factors as long-term academic stress, work overload, financial constraints, family demands, or unresolved emotional anxiety tend to be the causes of chronic stress among students. In contrast to acute stress, caused by brief and adaptive stress, chronic stress causes the long-term activation of the HPA axis and the constant increase in cortisol concentration, which has a negative neurobiological and cognitive effect. Hippocampal deterioration is one of the best-reported effects of chronic cortisol exposure, and it directly affects memory consolidation and spatial learning (Pruessner et al., 2001).

The long-term effects of chronic stress interfere with synaptic plasticity and long-term potentiation (LTP), which is the key to the memory formation mechanism. With age, these alterations result in a decrease in dendritic branching of neurons in the hippocampus and a loss of neural connectivity, making it hard to effectively establish new information. Also, prolonged exposure to cortisol compromises the functioning of the prefrontal cortex, resulting in executive functioning impairments, including problem-solving, working memory, and task switching (Liston et al., 2009). These deficiencies are reflected in academic burnout, cognitive overload, and reduced learning efficiency.

Students who are exposed to chronic stress behaviorally exhibit indications of cognitive exhaustion, poor concentration, and slow processing of information. The symptoms have a tendency to cause burnout, lack of interest in academics, absenteeism, and even dropouts from academic programs. Neuropsychological studies prove that chronic stress can also endanger anxiety and depressive symptoms, which, in turn, deteriorate cognitive functions and academic success (Evans and Kim, 2007). This negative loop of stress, cognitive decline, and poor performance may be accrued throughout the academic terms and, thus, has long-term effects.

These findings are convincingly supported by evidence from neuroimaging studies. Excessive exposure to stress has been related to the shrinkage of hippocampal volume, lesser prefrontal cortical thickness, and change in amygdala responsiveness (Gianaros et al., 2007). All these biological indicators are in line with the observation in the background text that chronic stress causes long-term structural alterations in brain regions pertaining to memory. The summary of the main neurobiological variations between acute and chronic stress and their corresponding effects on the memory brain processes is summarized in Table 1.

Table 1. Neurobiological Effects of Acute vs. Chronic Stress on Memory

Component	Acute Stress	Chronic Stress
Arousal	Increased alertness	Cognitive fatigue, impaired focus
Hormones/Neurotransmitters	Catecholamines; transient cortisol	Sustained cortisol elevation
Brain Regions Affected	Amygdala; hippocampus	Hippocampus; prefrontal cortex; amygdala
Cognitive Outcomes	Possible enhancement of encoding	Impaired encoding, consolidation, and retrieval

4.3 Theme 3: Stress Interference with Retrieval Processes

The process of retrieving the stored information can be greatly impaired by stress. The impairments of retrieval are prevalent in academic appraisals, and anxiety levels and physiological arousal are high in academic examinations. Stress

during retrieval disturbs the performance of the prefrontal cortex, which is critical in accessing and organizing the stored memories. This is an inefficient part under stress, which is part of retrieval blocking, lower accuracy, and fragmentation of memory (Arnsten, 2009).

Physiologically, an increased cortisol level during the time of retrieval has a negative effect on overcoming the hippocampal pathways that form the stored memories. This is more so when it comes to complex or newly formed memories, as these demand greater neural coordination. Research has shown that stress may impair the working memory capacity, which also leads to the retrieval problems due to the lack of cognitive resources to recall (Schoofs et al., 2009). These interruptions are the reasons why learners can blank on exams after having studied well.

The study of neurocognition also underlines the effect of neural noise, which is uncontrolled neural firing patterns that grow in stress and disrupt proper memory recollection. This issue is aggravated by emotional arousal, and retrieval is highly situational and sensitive to performance pressure. It has been demonstrated through experimental research that the participants who were put under acute stress conditions demonstrated a reduced recall accuracy, slower speed of retrieval, and an increased rate of errors, particularly with academic-like memory tasks (Gagnon and Wagner, 2016). These results emphasize that retrieval is among the most susceptible phases of memory processing in times of stress and thus one of the most important issues to be addressed in terms of academic and psychological interventions.

4.4 Theme 4: Age, Individual Differences, and Types of Stressors

The effects and stress reactions to stress differ significantly in relation to age, personal characteristics, and the environment. The prefrontal cortex and hippocampus neural circuits are still developing, which makes children and adolescents very sensitive to stress. Studies indicate that emotional responsiveness and slower cognitive resilience to stress exposure occurring in adolescents, in particular, make them more susceptible to acute and chronic stress outcomes in memory (Laviola et al., 2003). The stressors in college-aged adults are various and may include academic competitiveness, issues of independence, and social pressure, among others, and may all contribute to stress responsiveness.

Stress-memory interactions are also moderated by individual differences, including genetic predisposition, temperament, coping style, and levels of resilience. There are adaptive coping styles of some students that cushion the impacts of stress and maladaptive ones that increase cognitive performance. Indicatively, test subjects with high trait anxiety or poor emotional regulation ability tend to be more susceptible to memory discontinuity with stress (Hancock and Warm, 2003). The experiences of stress among students are further influenced by sociocultural factors such as economic status, family pressure, and cultural orientation regarding academic achievement.

The stressors may also be either intrinsic or extrinsic, which goes in line with the background text. Intrinsic stressors are caused by internal thinking pressures, i.e., perfectionism/fear of failure. Extrinsic stressors are external - academic workload, examinations, peer comparison, or environmental instability. The interaction of these forms of stressors is associated with different cognitive performances among learners. It is imperative to understand these differences so as to create specific interventions that will meet the needs of diverse students.

4.5 Theme 5: Positive Effects of Mild Stress (Eustress)

Not every stress is bad; the so-called eustress or mild, moderate stress can improve the level of cognitive performance and motivation. Eustress is associated with environmental demands that are seen to be challenging but manageable, which results in adaptive physiological arousal that stimulates learning and increases the encoding of memories. The Yerkes-Dodson curve shows that there is an optimum in both arousal levels, whereby there is increased concentration, alertness, and engagement (Teigen, 1994). Mild stresses in an academic environment, e.g., before a presentation or a deadline, may arouse focus, goal-oriented behavior, and intrinsic motivation.

On a neurobiological level, mild stress may lead to the improvement of dopaminergic activity of the prefrontal cortex, which increases working memory and enhances the effective processing of information. It has been shown that moderate arousal of the emotional state can promote synaptic plasticity with the help of limited cortisol increases in reinforcing memory formation (Howland and Wang, 2008). The beneficial effects are more likely to be experienced in students who perceive stress positively, hence the role of mindset and cognitive appraisal. Research in the educational psychological field demonstrates that the reinterpretation of stress as a motivator enhances performance and loss of negative affect (Crum et al., 2013). Eustress, therefore, is a productive version of stress that promotes learning when accompanied by sufficient coping mechanisms and relaxation.

5. Synthesis, Comparative Analysis, and Conceptual Model

The interaction between stress and memory in a group of students is complex and depends on the neural architecture, physiological dynamics, and contextual variables. The integration of evidence in the five thematic domains indicates a complex relationship in that stress offers facilitative and inhibitory influences depending on its intensity, length, and timing relative to memory processes. The acute stress seems to improve the process of encoding memory in certain circumstances, and the chronic stress always deteriorates the process of encoding, consolidation, and retrieval. It is this duality that indicates that stress should be viewed as a spectrum and not as a homogenous one.

Comparative study of acute and chronic stress indicates that the adaptive efficacy of stress is closely connected to neurobiological time. Stressful situations cause the fast release of catecholamines and a temporary rise of cortisol, which may support amygdala-hippocampal communication and the capacity to encode information that has emotional significance. This behavior can be explained by the inverted-U model of arousal and performance, which states that the moderate state of stress pathways can result in optimal performance of certain cognitive functions (Arnsten, 2015). Nonetheless, when acute stress goes beyond the optimal levels, such as with extreme exam anxiety, encoding is impaired

because working memory capacity has been lowered and the prefrontal control has been impaired. In that way, although acute stress can be beneficial to performance under controlled circumstances, its cognitive advantage can be weak and can be readily undone by increased emotional arousal.

However, the same thing does not apply to chronic stress. Sustained HPA axis activation and chronic cortisol increase impair the neural structures that are important in memory. The neuroimaging studies also found that the volume of the hippocampus and the impaired prefrontal cortex networks and hyper-responsive amygdala among participants exposed to chronic stress are always reported (Lupien et al., 2018). The changes disrupt the development of new memories and affect cognitive flexibility, as well as compromising retrieval accuracy. The students with long-term socioeconomic stress or chronic academic pressure are thus more likely to display cumulative cognitive loss, exhaustion, and academic participation. This is in line with previous findings that long-term stress will result in long-term alterations in the components of the brain that contribute to memory.

One more point that comes up as a result of cross-theme integration is that memory stages are sensitive to time. Encoding, consolidation, and retrieval have varied responses to exposure to stress. The encoding of salient information with stress before or during encoding could increase the memory of the salient information but deteriorate the neutral or peripheral information. Consolidation stress may make or break memory traces depending on the level of hormones and emotional context (Ritchey et al., 2008). Retrieval stress always has a detrimental effect and results in retrieval-blocking, heightened neural noise, and reduction of executive control. Therefore, poor performance is frequently associated with retrieval disturbances rather than memory impairments when students experience high levels of stress when undergoing examinations.

Differences in individuals also elaborate the stress-memory relationship. The existence of evidence in terms of a range of themes proves that there is a considerable variability that depends on age, temperament, coping style, and sociocultural context. Teenagers are more prone to stressors because the prefrontal cortex is still developing, and therefore, they respond more to stressors. And university students who are subjected to extrinsic stressors (financial strain, competition) might be more chronically impaired by a stress-related profile. Trait anxiety and high behavioral inhibition as temperamental characteristics make people susceptible to memory failures caused by stress, whereas resilience, optimism, and adaptive coping lead to more positive outcomes (Compas et al., 2017). Such individual-level differences imply that the interventions should not have a standard approach but instead should be customized.

Bringing these strands together, a conceptual model emerges in which stress affects memory through a three-pathway mechanism:

- **Physiological arousal pathway** — acute neuroendocrine activation modulates encoding and selective attention;
- **Neuroplasticity pathway** — chronic cortisol exposure alters neural structure and disrupts synaptic functioning;
- **Executive regulation pathway** — stress-induced prefrontal cortex dysfunction impairs retrieval and working memory processes.

These pathways are dynamic and hence the numerous and even conflicting effects witnessed in the literature.

Learning about the stress-memory relationships within the framework of this combined model would help educators and mental-health specialists to support students better. To illustrate, acute stress coping during examination can enhance an individual's retrieval performance, whereas chronic stress coping during the semester can benefit an individual's cognitive health in the long term. Finally, the synthesis points to the fact that, although stress cannot be removed in academic settings, the challenge-support balance can maximize the learning process and cognitive resilience.

6. Stress Management and Intervention Strategies

Stress management is the key to protecting cognitive functioning and bolstering the academic performance of students. As acute and chronic stress may lead to memory encoding, consolidation, and retrieval dysfunctions, it is essential to apply specific interventions. Managing stress in a way that leads to minimal harmful stress, development in coping mechanisms, and resilience may have a tremendous impact on the performance and well-being of students.

Some of the most evidence-based techniques include mindfulness-based interventions (MBIs). Mindfulness improves focused attention, emotional regulation, and non-reactive awareness, thus lessening physiological stress responses. It has been found that mindfulness training reduces cortisol, increases working memory, and cognitive flexibility (Jha et al., 2010). Programs in schools and universities that involve mindfulness have been reported to reduce anxiety in examinations and enhance emotional steadiness, proving to be of educational value.

Another strategy that is effective strategy is cognitive-behavioral therapy (CBT). CBT assists students in recognizing and reorganizing their maladaptive thoughts, enhancing problem-solving, and managing their emotions. Meta-analyses reveal that CBT has a significant impact on perceived stress and academic functioning in terms of the improvement of coping skills and the decrease of negative appraisal patterns (Regehr et al., 2013). These interventions also promote confidence and performance in tests.

There are behavioral interventions like routine exercise, which are associated with the reduction of stress in the body due to biological processes. Working out lowers cortisol, enhances the release of endorphins, and stimulates neurogenesis in the hippocampus, which facilitates memory formation and cognitive health. Short-term aerobic exercise is beneficial to executive functioning and decreases stress-related cognitive impairments (Hillman et al., 2008). It is thus possible to introduce exercise opportunities in schools or campuses to offer low-cost stress interventions.

The other important factor is sleep hygiene. Sleep deprivation increases cortisol, inhibits synaptic plasticity, and memory consolidation. Academic stress often causes students to lose sleep, which exacerbates stress-related cognitive impairments. It has been demonstrated that sufficient sleep improves the work of the hippocampus, makes people less emotional, and

increases the flexibility of recall (Krause et al., 2017). Academic stress can be reduced by promoting good sleep behavior by means of education and institutional policy.

Institutional and social supports are also significant. Academic-skills workshops, peer-support groups, and counseling services assist the students to cope with the stress through the provision of emotional and practical support. Social support has been revealed to be protective against cognitive decline as well as buffer stress response, especially in the challenging academic setting (Ozbay et al., 2007). Colleges that have mental-health resources available will have a higher chance of having strong student communities.

Lastly, stress mindset intervention is a ray of hope. These interventions will make students redefine stress as a challenge and not a danger. The studies have shown that having a stress-is-enhancing attitude enhances performance under pressure and physiological stress responses (Crum et al., 2017). Including these practices in student orientations or classroom activities will potentially make resilience and academic motivation stronger.

In general, stress management among students involves a multi-dimensional process that involves the incorporation of behavioral, psychological, physiological, and institutional stress management strategies. Mindfulness helps to decrease stress reactivity, CBT will enhance coping abilities, physical activity will enhance neurobiological functioning, sleep hygiene will assist with memory consolidation, and social and mindset interventions will offer the much-needed emotional scaffold. A combination of these strategies would go a long way in minimizing the adverse impacts of stress on memory and cognitive functions. Table 2 describes the evidence-based stress-management strategies and ways that each of these aids cognitive functioning among students.

Table 2. Stress Management and Intervention Strategies

Technique	Description
Mindfulness	Enhances attention, reduces stress reactivity.
Cognitive-behavioral therapy	Reframes negative thoughts; improves coping skills
Exercise	Reduces cortisol; enhances neuroplasticity
Sleep hygiene	Supports consolidation and emotional regulation

7. Discussion

A synthesis of the results throughout this review indicates that stress has adaptive and maladaptive effects on memory among students, depending on the intensity of the stress and the duration and time it occurs. Acute stress at optimal levels may boost encoding by increasing physiological arousal and attention to the task, which is in line with the Yerkes-Dodson model. Nonetheless, beyond this optimum level of arousal, which usually occurs when exams are high stakes, there is an increase in cortisol and sympathetic stimulation at the expense of encoding and working memory. This shows that acute stress is very much situational in nature, with the influence it has being determined by the situational requirements and personal appraisal.

Conversely, long-term stress always has adverse effects on learning and memory. The long-term effects of HPA axis activation include long-term cortisol exposure, which impairs hippocampal and prefrontal systems that are involved in consolidation and executive functions. According to neuroimaging, stressed individuals who have long-term stress experience decreased volume of hippocampal and prefrontal cortical thinning (Bourne Jr & Yaroush, 2003). These neural changes present themselves in the form of cognitive exhaustion, hindered retention, and worse academic results, making chronic stress a principal impediment to the success of education in the long term.

One of the most fragile cognitive processes in the state of stress turns out to be memory retrieval. Prefrontal control is necessary to support retrieval, and this is impaired during anxiety and physiological arousal to cause neural noise and retrieval blocking. This is the reason why there is usually poor performance in examinations, even when students have properly prepared. The interventions should therefore be focused on reducing stress during evaluations.

The effects of stress are also moderated by individual differences. Susceptibility is affected by developmental stage, temperament, coping strategies, and sociocultural background. Teenagers, whose prefrontal cortex is underdeveloped, are particularly weak, whereas students with adaptive coping mechanisms, including cognitive reappraisal or mindfulness, are more resilient (Scott Jr and House, 2005).

Notably, stress reveals that stress is not bad. Stress can be an asset in terms of motivation, involvement, and performance when the challenges are seen to be manageable. Mindset interventions may be valuable as students who think that stress enhances performance better when stressed (Jamieson et al., 2012).

Regardless of these findings, there are gaps in research. Most of the studies are based on short-term laboratory stress, which might not accurately be representative of the more complicated academic stressors. There is a need to undertake more longitudinal studies and determine the effect of the accumulation of stress throughout academic semesters. Also, much attention has been given to the hippocampus and the prefrontal cortex, yet other brain areas, such as the anterior cingulate cortex and insula, seem to play a central role in cognitive dysfunction in stress (Menon and Uddin, 2010).

Altogether, this review shows that stress, as a part of academic life, has a cognitive effect that can be changed. This knowledge of the subtle impacts of acute and chronic stress on memory can then lead educators and policymakers to create supportive environments that will minimize the negative impacts of stress and maximize the positive risks of resilience and eustress. These initiatives are necessary in improving academic achievement as well as student welfare.

8. Conclusion

This review shows that stress has a strong and multifaceted influence on memory encoding and retrieval in students. Moderate acute stress can momentarily enhance encoding through heightened physiological arousal, sharper attentional

focus, and improved amygdala–hippocampal communication. However, these benefits depend heavily on stress level and cognitive appraisal. When acute stress exceeds optimal levels—as in high-stakes examinations—it disrupts working memory, attention, and accurate encoding, demonstrating that its effects are dynamic and situation-dependent. In contrast, long-term stress consistently produces harmful cognitive outcomes. Prolonged HPA axis activation and sustained cortisol release impair neural plasticity, reduce hippocampal volume, and hinder prefrontal cortex functioning. These neurobiological changes result in long-term deficits in consolidation, retrieval, executive functioning, and overall academic performance. Students facing chronic academic demands, socioeconomic pressure, or persistent emotional strain are therefore at heightened risk of cumulative cognitive decline. A prominent theme is the particular vulnerability of memory retrieval to stress. Many students report retrieval failures, often described as “blanking out,” which arise from stress-induced prefrontal dysfunction and increased neural noise. This underscores the necessity of reducing stress in evaluative contexts and equipping students with effective coping strategies. At the same time, the presence of eustress shows that stress is not uniformly detrimental. When perceived as manageable, it can enhance motivation, engagement, and cognitive performance. These outcomes are moderated by factors such as stress mindset, coping style, developmental stage, and sociocultural context, demonstrating the need for targeted, context-specific interventions. Overall, understanding how acute and chronic stress affect memory processes provides valuable insights for educators, clinicians, and policymakers aiming to safeguard cognitive wellbeing, improve academic performance, and foster student resilience.

REFERENCES

- Joëls, M., Pu, Z., Wiegert, O., Oitzl, M. S., & Krugers, H. J. (2006). Learning under stress: How does it work? *Trends in Cognitive Sciences*, 10(4), 152–158.
- Lupien, S. J., Maheu, F., Tu, M., Fiocco, A., & Schramek, T. E. (2009). The effects of stress and stress hormones on human cognition: Implications for the field of brain and cognition. *Brain and Cognition*, 65(3), 209–237.
- McEwen, B. S., & Morrison, J. H. (2013). The brain on stress: Vulnerability and plasticity of the prefrontal cortex over the life course. *Neuron*, 79(1), 16–29.
- Owens, M., Stevenson, J., Norgate, R., & Hadwin, J. A. (2014). Processing efficiency theory in children: Working memory as a mediator between test anxiety and academic performance. *Anxiety, Stress, & Coping*, 27(4), 431–444.
- Romeo, R. D. (2010). Adolescence: A central event in shaping stress reactivity. *Developmental Psychobiology*, 52(3), 244–253. <https://doi.org/10.1002/dev.20437>
- Diamond, D. M., Campbell, A. M., Park, C. R., Halonen, J., & Zoladz, P. R. (2007). The temporal dynamics model of emotional memory processing: A synthesis on the neurobiological basis of stress-induced amnesia, flashbulb memories, and traumatic memories. *Progress in Neuro-Psychopharmacology & Biological Psychiatry*, 31(8), 1481–1511.
- Kim, J. J., Song, E. Y., & Kosten, T. A. (2015). Stress effects in the hippocampus: Synaptic plasticity and memory. *Stress*, 19(2), 1–12.
- Lupien, S. J., Juster, R. P., Raymond, C., & Marin, M. F. (2018). The effects of chronic stress on the human brain: From neurotoxicity, to vulnerability, to opportunity. *Frontiers in Neuroendocrinology*, 49, 91–105.
- Sapolsky, R. M. (2015). Stress and the brain: Individual variability and the inverted-U. *Nature Neuroscience*, 18(10), 1344–1346.
- Schwabe, L., & Wolf, O. T. (2010). Stress impairs the reconsolidation of neutral memories and enhances the reconsolidation of emotional memories. *Neurobiology of Learning and Memory*, 94(1), 54–60.
- Squire, L. R., & Wixted, J. T. (2011). The cognitive neuroscience of human memory since H.M. *Annual Review of Neuroscience*, 34, 259–288.
- Ulrich-Lai, Y. M., & Herman, J. P. (2009). Neural regulation of endocrine and autonomic stress responses. *Nature Reviews Neuroscience*, 10(6), 397–409.
- Andreano, J. M., & Cahill, L. (2006). Glucocorticoid release and memory consolidation in men and women. *Psychological Science*, 17(6), 466–470.
- Roozendaal, B., & McGaugh, J. L. (2011). Memory modulation. *Behavioral Neuroscience*, 125(6), 797–824.
- Klier, C., & Buratto, L. G. (2020). Stress and long-term memory retrieval: a systematic review. *Trends in psychiatry and psychotherapy*, 42(3), 284–291.
- Vogel, S., & Schwabe, L. (2016). Learning and memory under stress: Implications for the classroom. *npj Science of Learning*, 1, 16011.
- Evans, G. W., & Kim, P. (2007). Childhood poverty and health: Cumulative risk exposure and stress dysregulation. *Psychological Science*, 18(11), 953–957.
- Gianaros, P. J., Horenstein, J. A., Cohen, S., et al. (2007). Perigenual anterior cingulate morphology covaries with perceived social standing. *Nature Neuroscience*, 10(10), 1351–1357.
- Liston, C., McEwen, B. S., & Casey, B. J. (2009). Psychosocial stress reversibly disrupts prefrontal processing and attentional control. *Proceedings of the National Academy of Sciences*, 106(3), 912–917.
- Pruessner, J. C., Baldwin, M. W., Dedovic, K., et al. (2005). Self-esteem, locus of control, hippocampal volume, and cortisol regulation. *Archives of General Psychiatry*, 62(6), 593–600.
- Arnsten, A. F. T. (2009). Stress signalling pathways that impair prefrontal cortex structure and function. *Nature Reviews Neuroscience*, 10(6), 410–422.
- Gagnon, S. A., & Wagner, A. D. (2016). Acute stress and episodic memory retrieval: Neurobiological mechanisms and behavioral consequences. *Neuroscience & Biobehavioral Reviews*, 61, 154–166.

23. Schoofs, D., Wolf, O. T., & Smeets, T. (2009). Cold-pressor stress decreases performance on working memory tasks. *Psychophysiology*, 46(1), 118–126.
24. Hancock, P. A., & Warm, J. S. (2003). A dynamic model of stress and sustained attention. *Human Factors*, 45(4), 381–389.
25. Laviola, G., Macrì, S., Morley-Fletcher, S., & Adriani, W. (2003). Risk-taking behavior in adolescent mice: Psychobiological determinants and early epigenetic influence. *Neuroscience & Biobehavioral Reviews*, 27(1–2), 19–31.
26. Crum, A. J., Salovey, P., & Achor, S. (2013). Rethinking stress: The role of mindsets in determining stress responses. *Journal of Personality and Social Psychology*, 104(4), 716–733.
27. Teigen, K. H. (1994). Yerkes–Dodson: A law for all seasons. *Theory & Psychology*, 4(4), 525–547.
28. Howland, J. G., & Wang, Y. T. (2008). Synaptic plasticity in learning and memory: stress effects in the hippocampus. *Progress in brain research*, 169, 145–158.
29. Arnsten, A. F. T. (2015). Stress weakens prefrontal networks: Molecular insults to higher cognition. *Nature Neuroscience*, 18(10), 1376–1385.
30. Compas, B. E., Jaser, S. S., Bettis, A. H., et al. (2017). Coping, emotion regulation, and psychopathology in childhood and adolescence. *Journal of Child Psychology and Psychiatry*, 58(6), 711–728.
31. Ritchey, M., Dolcos, F., & Cabeza, R. (2008). Role of amygdala connectivity in the persistence of emotional memories over time. *Cognitive, Affective, & Behavioral Neuroscience*, 8(3), 329–337.
32. Bourne Jr, L. E., & Yaroush, R. A. (2003). *Stress and cognition: A cognitive psychological perspective* (No. IH-045).
33. Jamieson, J. P., Mendes, W. B., & Nock, M. K. (2012). Improving acute stress responses through cognitive reappraisal. *Journal of Experimental Psychology: General*, 141(3), 417–422.
34. Menon, V., & Uddin, L. Q. (2010). Saliency, switching, attention, and control: A network model of insula function. *Brain Structure and Function*, 214(5–6), 655–667.
35. Scott Jr, L. D., & House, L. E. (2005). Relationship of distress and perceived control to coping with perceived racial discrimination among black youth. *Journal of Black Psychology*, 31(3), 254–272.
36. Crum, A. J., Akinola, M., Martin, A., & Fath, S. (2017). The role of stress mindset in shaping cognitive, emotional, and physiological responses to challenging situations. *Anxiety, Stress, & Coping*, 30(4), 379–395.
37. Hillman, C. H., Erickson, K. I., & Kramer, A. F. (2008). Be smart, exercise your heart: Exercise effects on brain and cognition. *Nature Reviews Neuroscience*, 9(1), 58–65.
38. Jha, A. P., Stanley, E. A., Kiyonaga, A., Wong, L., & Gelfand, L. (2010). Examining the protective effects of mindfulness training on working memory capacity and affective experience. *Emotion*, 10(1), 54–64.
39. Krause, A. J., Simon, E. B., Mander, B. A., et al. (2017). The sleep-deprived human brain. *Nature Reviews Neuroscience*, 18(7), 404–418.
40. Ozbay, F., Johnson, D. C., Dimoulas, E., et al. (2007). Social support and resilience to stress. *Psychiatry*, 4(5), 35–40.
41. Regehr, C., Glancy, D., & Pitts, A. (2013). Interventions to reduce stress in university students: A review and meta-analysis. *Journal of Affective Disorders*, 148(1), 1–11.
42. Squire, L. R., & Zola-Morgan, J. (1991). The cognitive neuroscience of human memory since H.M. *Annual Review of Neuroscience*, 14, 259–288.