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# Stress-Induced Cognitive Impairments: A Narrative Systematic Review

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## Abstract

Stress is multifactorial, acting via the hypothalamo-pituitary-adrenal axis; with its mediator's receptors present in the brain's cognition control area. We identified the causative stressors, ameliorating interventions and patho-physiological mechanisms of stress-induced cognitive-impairments in Medline via a systematic review of PubMed™ using search keywords: 'stress*', 'cogniti*', 'executive function', 'memory', 'learning' and 'impairment', limited in time to 2009 and later, yielding 2795 papers on March 23rd, 2017, reduced to 197 after removing duplicates and reviewing the titles for appropriateness, followed by review of the articles using a-priori criteria. Perceived stress showed no gender disparity, although financial stress was significant in women like work-stress in men. Marriage conferred more stress on women. Acute and/or chronic stress affects learning, memory, attention and executive function cognitive domains. Stressors causing cognitive-impairments include prenatal stress, maternal deprivation, childhood maltreatment, anaesthesia, chronic immobilization, chronic social defeat, illness, noise, sleep deprivation and trauma. Exercise, environmental enrichment and NSAIDS reduce stress-induced cognitive-impairment. Implicated patho-physiological mechanisms include cyclo-oxygenase-2, cholinergic muscarinic and μ-opioid receptors. Newer pathophysiologic mechanisms support future research.

**Keywords:** Stress; Cognition; Cognitive impairments; Memory impairment; Learning impairment

## Introduction

Stress is multifactorial and has been described as being of physiological, physical and psychological origin [1-3]. The development of the stress response occurs ultimately via the hypothalamo-pituitary-adrenal axis common pathway [1] as described by Hans Selye's general adaptation syndrome. The stress response evokes the elaboration of its adreno-cortical (the glucocorticoids; cortisol in man and corticosterone in rodents) and adreno-medullary (especially noradrenaline) mediators [4-6]. Stress hormone receptors have been isolated in the brain's cognition control areas, like the type I glucocorticoid receptors in the limbic system, type II glucocorticoid receptors in the subcortical and cortical-prefrontal cortex, adrenergic receptors in the amygdala [4,5,7,8]; emphasizing the capacity of stress to affect cognition. These receptors presence have been presented in animal models [9-11]. Irrespective of the stressor, the stress response is evoked [1] via the same old cascade. Stress's at least putative causality of cognitive impairment is all the more cardinal as cognitive impairment has been described as being intermediary between stress and neurodegeneration [12-14].

## Research questions and Objectives

This study set out to search the literature for evidence of previous answers to the questions: what stressors cause cognitive impairments, what interventions can reverse the stress- induced cognitive impairments and by what mechanisms do stress-induced cognitive impairments occur?

**The following study objectives were deduced:**

a. To determine the stressors implicated in cognitive impairments,

b. To establish the putative experimental ameliorating interventions for stress-induced cognitive impairments,

c. To elucidate the underlying novel pathophysiological mechanisms of stress induced cognitive impairments.

## Materials and Methods

A systematic database review of the National Institute of Health's Medline via PubMed™ was done using the keywords with wildcards and truncators: 'stress*', 'cogniti*', 'executive function', 'memory', 'learning' and 'impairment'; to call papers on the research theme. These were combined using 'OR' (for synonyms) and 'AND' ( to pull the keyword groups together), then limited in time to 2009 and later, yielding 2795 results as of March 23rd, 2017, reduced to 197 after removing duplicates and reviewing the titles for appropriateness, followed by a thorough review of the articles according to a-priori criteria. The details of the search yields, evidence synthesis techniques and challenges

are summarily presented in a PRISMA flow diagram in **Figure 1**. To improve the sensitivity of the search, restrictions on study types were relaxed as against the pre-search protocol to limit results to only clinical trials, cohort studies and human studies; there were significant paucity of human studies which necessitated the decision. Most of the studies were heterogeneous and not of sufficiently high quality to proceed to the quantitative synthesis and only a narrative review following the qualitative synthesis was acceptable.

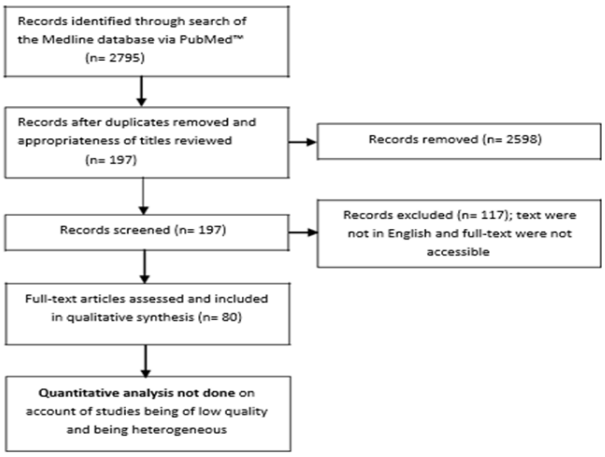


**Figure 1:** PRISMA flow diagram describing the search yields and rationale for synthesis Methods.

## Narrative Synthesis

### Demographic dynamics of stress

**The gender disparities of human stress:** The self-reported reactions to stress varied between the sexes on the physical and mental domains, this was irrespective of the reported similar average stress levels, some of these reactions (symptoms) include: irritability/anger, fatigue, loss of interest (apathy), feeling like crying, feeling nervous, depressed or sad and headaches amongst others [15]. It was reported that women seemed to have more stressors associated with money and the economy, unlike men who reported work stress as being their main stressor [15]. Females reported the use of reading as a stress managing tool more than men; they also tend towards the use of strategies requiring spending time with other people (such as sharing time with family and friends, attending religious activities), more than men would [15].

Katz and colleagues [16] working in the United States reported their work in 2016, using 507 elderly subjects, free of dementia at baseline review in 1993 and followed them up annually from 2005 using the 13-item PSS scale to assess for the level of stress and further accessed for the presence of amnesic MCI. They reported that women were more likely to be stressed than men. Kluen and colleagues working in Hamburg, Germany reported in 2017 their double-blind, placebo-controlled trial, using healthy men and women to access the capacity of the major stress hormones (glucocorticoids and noradrenaline) to significantly disrupt memory generalization. They reported the existence of sex-specific memory generalization deficits following noradrenergic stimulation, as induced by the α2-adrenoceptor antagonist, yohimbine; here, women unlike their male counterparts expressed generalization memory deficit. Pascuan and colleagues in 2017 published their works on the progeny of pregnant mice exposed to restraint stress for two hours daily in the last week of pregnancy as against a control group of pregnant mice that were left undisturbed. The female adult mice progeny showed impairments in spatial memory, upregulation of hippocampal glucocorticoid receptors as well as alteration of hippocampal T-Helper cells 1 to T-helper cells 2 ratio; none of these findings were noticed in any male progeny. Hence prenatal exposure to stress exhibits spatial memory impairing effects [9].

**Marital status disparities in human stress levels:** Married women have reported to have higher levels of stress than single women; the married women also accept more often than their single contemporaries, that their stress levels have increased over time; the single women are however more likely to feel that their actions aimed at managing stress is enough, when compared to married women [15].

### Cognitive domains affected by stress

The major cognitive domains affected include Learning, Memory, attention, executive functions, etc [17].

### Stress and its causation of cognitive impairment

Acute [18-21] and chronic [22-30] stress have been linked with the causation of cognitive impairments. Katz and colleagues showed that high levels of perceived chronic stress were linked with new onset amnesic MCI, with a 30% increased risk of MCI for every 5 point increase in the PSS scores in their study population of septuagenarians.

Mild cognitive impairment is perceived as an intermediate stage on the progression of neurodegeneration, e.g., Alzheimer's disease. It is a common ailment in the elderly, characterized by diminution in memory, attention and overall cognitive decline more than is expected for age and educational status. It permits the individual to execute their daily activities. It is an intermediate state, culminating in dementias at a 10-15% annual rate [31]. Many stressors that have been linked with the capacity to induce stress-induced cognitive impairments are reviewed below:

Chronic immobilisation stress was reported by the work of Shilpa and colleagues from India in 2017, where male wistar rats that were subjected to immobilization for 2 hours daily over a 10 day period displayed cognitive impairments. Restraint stress [32] has been shown to elicit stress and it has been evidenced by increased serum corticosterone levels in rats; it has also been shown to be associated with stress-induced cognitive impairments [33-35]. Water immersion restraint stress in adult mice, have also been linked with capacity to induce stress-induced decline in cognition, as reported by Kusinawa and colleagues in Nagoya, Japan [36].

Chronic social defeat stress [37] as occurs following exposure to defeat from agonistic confrontations with other animals or situations /circumstances presented to the test subjects; as observed by Duque and colleagues, 2017, who exposed male mice to agonistic confrontation for 10 minutes daily for twenty days, reported amongst other findings, decline in recognition and emotional memory [37]. Childhood maltreatment [38]: Chronic stress from childhood maltreatment disrupts brain structure and function. Maltreated children perform badly on working memory tasks than do the non-maltreated. These were established following a study on 3-9 year olds; 136 non-maltreated and 223 maltreated [39].

Psychological stress: Psychological stress, induced by the Trier Social Stress Test (TSST), causes impaired memory; this formed

the basis for the work in Isreal by Guez and colleagues, where 48 healthy subjects (24 each were randomly assigned to the TSST and control groups). They were subjected to item-association memory tasks prior to and after the manipulations (stress/ non-stress). They showed that memory for both words and pictures were impaired following TSST unlike the controls. Banks and colleagues reported their work using 60 participants that did working memory tasks before and after they were exposed to psychological stress (control writing task) as a means to examine the burden of stress-related working memory impairment; they showed that psychological stress may not always induce stress-related working memory impairment [40].

Anesthesia: Spatial memory and learning deficits noted in rats repeatedly exposed to sevoflurane-induced stress. Noisy conditions: Noisy environment is shown to cause cognitive decline. Wright and colleagues reported their study using 54 healthy participants (24 of whom were men) who were put through a group of cognitive assessments (the following were measured, psychomotor speed, executive functions, attention, verbal learning, working memory and verbal memory) under noisy conditions. It was shown that in the noisy conditions, psychomotor speed was significantly slower, working memory was reduced and decision making (executive function) was done more cautiously; the findings also showed no gender disparity [41]. It had been observed also that the rats subjected to noise stress showed lower memory performance and higher oxidative stress when compared to the nonstressed rats [42]. Chronic exposure to early post-natal scream sound stress has also been reported to mediate learning and memory deficits in adult mice [43]. Illness / treatment related stress [44] were reported by Fayette and colleagues in 2017, who established that patients receiving cancer chemotherapy for various malignancies exhibited decline in multiple cognitive domains; albeit the pathophysiology behind the cognition impairing effects were not clear but thought to be related with increased oxidative stress levels, neurotoxicity from chemotherapy, anaemia, inflammatory processes, reduced brain connectivity, etc. HIV-infected women have been reported to be predisposed to learning and memory deficits, probably due to their high perceived stress. Using the perceived stress scale-10 to assess stress, 36 HIV-infected women from the Chicago Consortium of the Women's Interagency HIV Study (WIHS), they were subjected to verbal memory tasks to assess cognitive functions; they showed that their subjects had less efficient strategic retrieval and verbal memory deficits [45,46].

Early life stress has been linked with development of cognitive impairment in adulthood in laboratory animals. This is shown with pre-natal exposure to restraint stress as well as maternal deprivation stress as follows. Prenatal exposure to stress was implicated in the causation of stress-induced cognition decline by the works of Pascuan, where they exposed pregnant mice to restraint stress for 2 hours daily in the last week of pregnancy. The adult female mice that were prenatally exposed to stress, unlike their male counterparts, exhibited impaired spatial memory..

Maternal deprivation in rats has been linked with susceptibility to cognitive impairment (learning and memory) in these rats in adulthood, as described by the works of Alzoubi and colleagues in Brazil; the mechanisms have been linked with reduction in the hippocampal Brain derived neurotrophic factors. Wearick-Silva and colleagues reported impairment in spontaneous recognition in mice that were separated from their mothers within the first two weeks of their lives [47]. Sleep deprivation [48] has been linked to induction of stress, via its ability to increase oxidative stress, mostly in the hippocampus. Alzoubi and colleagues showed that 8 weeks exposure of mice to sleep deprivation impaired spatial learning and memory, as were accessed using the radial arm water maze; they also deposited that chronic sleep deprivation was capable of impairing both short and long term memory.

Trauma [49] has been reported in many ways to induce stress. Ovariectomy in rats was reported by Cui and colleagues from their work in Shanxi, China to elevate markers of oxidative stress [50]. Whisker (vibrissal) paralysis following severance of the mandibular and buccal branches of the facial nerve in rats was observed to be associated with elevated corticosterone levels, and impaired retrieval of spatial tasks; these findings were investigated by Patorroyo and colleagues who concluded that the spatial memory impairment was attributable to the associated whisker-paralysis induced stress [51]. Surgery induced hippocampal mediated spatial learning and memory has also been reported in aged rats. Post-traumatic stress disorder (PTSD) has been linked with its capacity to induce stress and cognitive impairments [52]; Stricker and colleagues in Boston, USA reported increased rates of cognitive impairments amongst Military service members and war veterans with PTSD, in comparison with controls without PTSD [52]. Traumatic stress was induced in rats by immobilizing them on boards for 2-8 days; behavioral signs similar to PTSD were said to have been noticed, accompanied by decline in spatial memory [53].

## Agents / actions putatively involved in ameliorating stress-induced cognitive impairments

Exercise has been reported to ameliorate cognition impairing effects of stress. Cui and colleagues reported that female rats with ovariectomy-induced cognitive defects, when exposed to 8 weeks treadmill exercises of progressively increasing load showed a decline in the cognitive impairments; these benefits were attributable to the exercise being able to reduce oxidative stress. Wearick-Silva and colleagues in 2017 exposed mice to maternal separation stress within their first two weeks of life, they subsequently subjected some of the mice to a three week running protocol. Spontaneous object recognition was impaired in all mice with maternal separation; a reversal was noticed in the mice on the running protocols. Treadmill exercises offers treatment rather than prevention of stress-induced memory impairment [54]. Kim and Leem have also shown that prolonged exposure to exercise may reduce the cognitive deficits from stress [55].

Environmental enrichment was studied by Shilpa and colleagues in India, 2017. The study evaluated the effect of environmental enrichment (EE) on spatial learning and memory, anxiety, depression and other molecular markers alteration induced by chronic immobilisation stress (CIS) using male wistar rats. The rats were subjected to CIS for 2hours daily for 10 days, followed by 10 days of EE. CIS exposed rats showed weight loss, anhedonia, spatial learning and memory impairments, increased immobility and anxiety; however, EE reduced these effects, and the EE associated resolution of the stress-induced cognitive impairments were modulated by neurotrophic factors, astrocytes as well as glucocorticoid receptors in the hippocampus, frontal cortex and amygdala.

Hydrogen sulphide: in rats, the hippocampus-dependent spatial memory retrieval is impaired by acute stress. NaHS

(Sodium hydrogen sulphide) as donor of hydrogen sulfide (H2S) was observed to eliminate the impairment in retrieval of spatial memory induced by acute stress; a function thought to be exerted by the inhibition of the activation of the JNK signalling pathway by H2S. Use of NSAIDS (Non-steriodal anti-inflammatory drugs) have been reported to ameliorate the cognition impairing effects of stressors. Agents like meloxicam were reported in China by Luo and colleagues in 2017 to interfere with the cyclooxygenase 2 (COX2) enzyme in rats exposed to chronic unpredictable mild stress (CUMS), thereby reversing the learning and memory deficits induced. Indomethacin was reported from the 2017 work of Duque and colleagues in Valencia, Spain to reverse the Chronic social defeat stress-induced impairment of emotional memory in male mice, albeit there were no such benefits for recognition memory.

Nicotine: Mice exposed to stress (CUMS and isolated feeding) developed cognitive impairments, that were significantly improved by use of Nicotine. Oestrogen: The female sex hormone oestrogen has been shown to be protective against the deleterious effects of stress on cognition. Aromatase, the enzyme significantly involved in the biosynthesis of oestrogens are known to be significantly more abundant in the prefrontal cortex of females than males in animal models, thus making more oestrogen availability in the prefrontal cortex of females as against males.

## Novel pathophysiological mechanisms of stress-induced cognitive impairments Oxidative stress:

Stress generates reactive oxygen species, thought to cause changes in the brain's neuronal architecture and cognition [56]. Cyclo-oxygenase 2 (COX2) has been implicated by the work of Luo and colleagues as been putatively causative of stress-induced cognitive impairments; they investigated the role of the NSAID meloxicam in reversing the impairing effects of chronic unpredictable mild stress on rats on learning and memory. They were able to deduce that stress caused over-expression of COX2, which is thought to cause learning and memory impairments possibly via activating the hippocampal neuronal cAMP/PKA-CREB-BDNF signaling pathways. The impairing effects of stress on cognition were blunted in the rats intragastrically administered with meloxicam as well as those with COX2 interefere lentivirus. Acting probably on the COX2, Indomethacin was reported by Kluen and colleagues to reverse the stress-induced cognitive impairments in male mice that acquired cognitive impairments following chronic exposure to agonistic confrontation (chronic social defeat stress); albeit, they reversed the emotional memory impairments, unlike the recognition memory impairments.

Cholinergic muscarinic receptors. M opioid receptors are putatively implicated in the causation of stress-induced cognitive impairment, especially in the recognition memory domain. The works of Liu and colleagues in China, using C57 mice that were exposed to 15 minutes of forced swimming (an acute stressful event) reported diminution in recognition memory (the retrieval phase, unlike the unaffected acquisition and consolidation phases); changes that were absent in animals administered with intra-peritoneal naloxone (opioid receptor antagonists) prior to exposure to the stressor. Polymerase chain reaction studies showed increase in μ opioid receptor mRNA in the brain (hippocampus also) in the stressed mice. This creates a huge debate for opioid receptors, as being a putative means for elaboration of the stress-induced memory impairments.

Cannabinoid type-1 receptors: Systemic, hippocampal, and peripheral blockade of cannabinoid type-1 (CB1) receptors abolished the stress-induced memory impairment, especially on adrenergic and noradrenergic cells, showing a cross-link between the central and peripheral mechanisms of stress causation [57]. Angiotensin II type 1 receptor blocker (ARB): Telmisartan an ARB interrupts stress-induced cognitive decline by its AT1 receptor blockade of the HPA axis, as shown in wistar rat models [58,59]. β-adrenoceptor antagonists: These have been shown to inhibit memory reconsolidation and have been putatively thought to be beneficial in prevention of consolidation of pathogenic memories associated with Post-traumatic stress disorder [60,61]. αza-adrenoceptor agonists: Chronic stimulation of αza-adrenoceptors protect the prefrontal cortex from the detrimental effects of stress [62]. Dopamine-1 receptor: High levels of Dopamine-1 receptor (D1R) activation of cyclic adenosine mono-phosphate (cAMP) calcium –K+ channel signalling and its attendant reduction of neuronal firing in the prefrontal cortex is a putative mechanism by which stress impairs working memory [63].

Histamine-3 receptor: Modulation of histamine 3 (H3) receptors is putatively useful in treating, not preventing stress-induced cognitive impairments [64]. Vasopressin-1b receptor: Vasopressin has been noted to activate the HPA axis via its vasopressin-1b (V1b) receptors in the pituitary. Direct antagonism of V1b receptor interferes with HPA activation and thus avoids the stress induced memory deficits [65].

## Beneficial effects of stress on cognition

While as above, it has been shown that stress has deleterious effects on the cognition, it has also been observed by Kato and colleagues who showed that chronic stress and acute administration of a stress hormone can reverse the cognition (recognition) impairing effects of alcohol withdrawal(Kato et al. 2016). They worked with mice that received 3% ethanol for 7 days, with or without restraint stress (for an hour daily); some of the mice were also pre-treated with intra-peritoneally administered corticosterone at doses of 1, 3 or 10mg/kg body weight. The significant decline in cognition noticed after 48 hours of ethanol withdrawal in the ethanol plus no stress group, was reversed in the ethanol plus stress group as well as the mice administered with an intermediate dose (3mg) of corticosterone, unlike the small and large dose groups(Kato et al. 2016). It has been reported that early life exposure to stress resulted in protection of cognition from the deleterious effects of stress in old age in rat models (Jauregui-Huert 2015).

## Conclusion

Stress-induced cognitive impairments occur from multiple stressors. However, exercising, improving environmental conditions, NSAID use amongst other novel measures can eliminate the stress-induced cognitive decline. Newer pathophysiologic mechanisms definitely open doors to future research on molecular basis of stress induced cognitive impairments. In plain language; the brain's capacity to execute tasks can be marred by stress of multiple types, these bad effects can be removed by exercising, ensuring clean and better organized environment (especially our living and working space) and guided use of some pain-killers called Non-steroidal anti-inflammatory drugs. The future of research in this area is all the more opened by recent discoveries of new ways by which stress can affect the brain.

## Conflict of Interest Disclosure

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