Advances in Applied Physiology
2020; 5(2): 12-18
http://www.sciencepublishinggroup.com/j/aap
doi: 10.11648/j.aap.20200502.11
ISSN: 2471-9692 (Print); ISSN: 2471-9714 (Online)

Review Article

Beneficial Effects of Yoga on Memory and Cognition Associated to Stress

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To cite this article:
Sudeep Mitra, Mousumi Mitra, Mantu Saha, Dilip Kumar Nandi. Beneficial Effects of Yoga on Memory and Cognition Associated to Stress. *Advances in Applied Physiology*. Vol. 5, No. 2, 2020, pp. 12-18. doi: 10.11648/j.aap.20200502.11

Received: May 30, 2020; **Accepted**: June 22, 2020; **Published**: July 13, 2020

Abstract: Stress generally occurs due to the organism's non-specific response against some kind of demand imposed on them. The compensatory responses to these stresses are known as stress response. The stressful stimuli can advance the physiological and psychological effects on the body which includes executive functions of the brain. The prefrontal cortex shows an important connection between the circuits that are linked with emotions, memory and planning. Due to this activation of stressful condition or situation damage occurs in the prefrontal cortex because of the catecholaminergic nature of its innervating afferents in the other parts of the brain. Along with hippocampus, the amygdala and prefrontal cortex define the aspects of memory and visual processing within brain. The decrease in PNS and GABAergic activity that causes stress related disorders can be corrected by yoga practice resulting in amelioration of disease system in the brain such as reduction in anxiety and improvement in cognitive function. Depression, Post Traumatic Stress Disorder (PTSD) that are elevated by stress shows low heart rate variability (HRV) and GABAergic activity which can be improved in response to yoga based interventions. Yoga practices show increase in the proportion of gray matter and escalation in activation of amygdala and frontal cortex of the brain. It was concluded that breathing, meditation and posture based yoga increased overall brain function and activity.

Keywords: Yoga, Stress, Prefrontal Cortex, Cognition, Hippocampus, Amygdala

1. Introduction

Any change in the body's reaction due to its adjustment with the environment or surrounding and also due to one's own thought according to its needs caused by extrinsic or intrinsic stimulus is known as stress. The automatic response which we accomplish from these stresses is termed as stress responses [1]. Stress reaction can be of two types- negative and positive. Distress is a condition caused due to continuous stress effect which is an example of negative stress reaction [2]. Positive stress reaction can be helpful in improving achievement, motivation, adaptation etc. Even stress in its low levels can be useful, helpful and healthy [3]. Subjects who are exposed to stress are provoked for physical, mental, emotional, social, physiological, biochemical, cognitive diseases.

"According to American Psychiatric Association stress is described as a sense of being overwhelmed, worry, destruction, press, exhaustion and lethargy. Therefore, stress can influence people in every age, sex, race and situation and can result in both physical and physiological health" [4].

Stress can be classified into three different forms according to the:

1) Nature of stressor that consist of two classes i.e. Physiological stress and Psychological stress [5-6].
2) Stress influence on individual which comprises of two main branches i.e. Positive Eustress and Negative Distress [7-8].
3) Exposure to the duration of stressor which is bifurcated into Acute Stress (short term) and chronic stress (long term) [9].

Physiological stress symptoms include disrupted digestion including diarrhea, constipation and nausea, chest pain and rapid heartbeat, insomnia, low energy, headaches and twitching or shaking.

Psychological stress comprises of cognitive symptoms such as impaired concentration, trouble remembering homework, chronic worrying, reduced judgement, impaired speech and unwanted thoughts. It also includes depression and anxiety, anger, irritability or restlessness, making of bad decision. Psychological stress also comprises emotional symptoms such as impatience, feeling of sadness, restlessness, irritability and loss of interest.

Positive stress reaction can be helpful in improving achievement, motivation, adaptation etc. Distress is a condition caused due to continuous stress effect which is an example of negative stress reaction. Eustress further shows the following characteristics within an individual. It motivates & focus energy, feels excitement, improves performance, positive feeling of contentment etc.

Acute stress disorders may include flashbacks, nightmares or intrusive memories, avoidance of reminders of the event, difficulty remembering it, dissociation, an inability to experience positive emotions, anxiety, sleep disturbances, irritability and difficult concentrating.

Chronic stress disorders sign and symptoms can include: irritability, fatigue, headaches, difficulty sleeping and concentration, changes in appetite, rapid disorganized thoughts etc.

Now the different approaches to study stress includes three impactful approaches which include response based, stimulus based and cognitive transactional based process perspectives. Living begins ability to response stress according to the demands of the surrounding environment is generally response based perspective. This pattern of stress in case of both animals and humans shows a three stage pattern. This pattern is known is General Adaptation Syndrome (GAS). It shows alarm reaction i.e. fight and flight response, resistance stage i.e. bearing chronic stress and active use of body resources and last one is exhaustion stage i.e. damage of tissues and diseases tends to occur. Hans Selye is referred as the representative of this approach [10-11].

Secondly the stimulus based approach is determined from the studies of Holmes and Rahe [12]. It depends on the amount and severity of the stressors and evaluate their power to deplete individuals, it has been revealed that the average amount of needed effort to overcome some event might be a suitable index of its severity [13-14].

Cognitive transactional based process perspective tends to show a relationship between individual and surrounding environment. Here the individuals tend to show his/her demands from the surrounding environment and obtain all benefits and resources from it. Thus, it leads to destruction of the surrounding environment and threating of their own life for future perspective [15].

Now, a complementary and alternative medicine therapy which is widely used to manage illness is yoga. In MBSR (Mindfulness Based Stress Reduction Program) yoga acts as its main component which includes breathing techniques, physical postures and meditations. These results were used to get fruitful result in elimination of stress and its negative effects. To maintain a good coordination between mind and all aspects of body yoga plays a dominant role in this stressful condition. Therefore, there are several studies or experiments that shows the positive effects of yoga on stress causing disorders.

The present study aims at to determine the various aspects of stress on brain effecting memory and cognition with the positive effects of yoga on it.

2. Effects of Stress on Brain

Autonomic nervous system plays a pivotal role in the maintenance of stress for living beings to adapt within the surrounding environment, sympathetic nervous system activates or regulates many physiological functions within the body [16]. The intensively regulated pathway of stress response is done by hypothalamus as it secretes corticotrophin releasing hormone to stimulate pituitary gland which ejects adrenocorticotropic hormone to blood stream which in turn maintains the intensively regulated stress response [17]. This in turn imparts the adrenal to release cortisol (glucocorticoid) and hence a negative feedback loop occurs on the Hypothalamus Pituitary adrenal axis (HPA). Corticotrophin hormone acts centrally to mediate fear related behaviors [18] and triggers other neurochemical responses to stress such as the noradrenergic system via the brain locus coeruleus [19].

2.1. Stress in Relation to Memory

The effect of stress causes the alteration within the hippocampus, which function as an important role in determination of memory. As a result, alteration in the hippocampus structure tends to be paired with deficits in the memory function [20-21]. It is caused due to a certain rise in the level of glucocorticoid and glutamate, with the depletion in the levels of brain derived nerve growth factor and inhibition of neurogenesis [22-25]. It has been seen through various studies that changes in hippocampus i.e. structural and functional changes do occur due to stress [26]. These structural changes include atrophy and neurogenesis disorders along with decreasing of dendritic branches due to increase in levels of plasma cortisol [27-28]. Analysis from hippocampus dependent loading data shows the negative effects of stress on learning. The subjects don't tend to be adapted to a new environment after being exposed to it [29]. An important process i.e. long term potentiation in memory formation is altered due to adrenal steroids [30]. Prefrontal Cortex (PFC) which regulates planning, attention, problem solving tends to be destructed primarily due to stress response. The hippocampus, pre frontal cortex and amygdala are important parts of brain that define the aspects of memory and visual processing in brain.

Traumatic stress leads to mental disorders i.e. POST TRAUMATIC STRESS DISORDER (PTSD). In this case the amygdala helps to show fear response [31-32] and due to

increase in stress response dendritic arborization occurs within it [33-34]. Lesions in the medial prefrontal dopaminergic system shows no ejection of peripheral cortisol due to stress along with failure to elevate sympathetic response to stress [35-36]. Thus, dysfunction of normal emotion and an inability to relate in social situation occur. Dendritic branching in prefrontal dopaminergic system reduces due to stress and also has inhibitory inputs.

2.2. Stress in Relation to Cognitive Impairment

The perception, interpretation and reception of perceived stimuli which helps in attention, learning, decision making is known to be cognition. Reduction in cognition takes place due to the effects of stress, which may increase again due to the changes in behavioral process leading to decrease in stress [37]. There is a complicated relationship impact of stress on memories which depends on the amount or duration of stress and the relevance of stressful event to be formed memories. The acute stress helps in the formation of those memories which doesn't involve working memories such as ongoing memories and procedural knowledge [38-39].

As it has been previously determined that glucocorticosteriods are released due to activation of stress, and now as it buck lipophilic properties it can easily diffuse through the blood brain barrier and may show effects on processing and cognition [40]. Chronic stress can cause further severe complications such as pathophysiological changes occur within the brain and so causes changes in behavioral, cognitive, mood disorders [41].

In case of biochemical estimations, increase in the level of IL-6 and plasma cortisol occurs and decrease in the level of cyclic adenosine monophosphate (C-amp) responsive elements binding protein and brain derived neurotrophic factor is found [42]. This biochemical outcome is seen in people who tend to be affected with stress. Another important relationship between stress and mood based cognitive disorders is that both can be estimated from increased level of interleukins and TNF-α [43].

Further acute effects of stress are induced in a short term manner due to the beta adrenergic effects whereas chronic effects which occur for long terms due to changes in gene expression mediated by steroids i.e. adrenal steroids [44]. These steroids can lead to destruction of neurons. Hippocampus related cognition disorders like decrease in genesis of neurons in the dentate gyrus area of hippocampus region occurs due to stress action [45].

At the time of stress, the most vital region within the brain is hippocampus because cognitive processes like last memories can have severe effects on facilitation, inhibition and even generating distinct response to stress. Damage and atrophy occurs in the hippocampus due to stress [46]. As previously stated prefrontal cortex activities are destructed generally temporarily due to stress action. [47]. Locus coerules, is a key player for the production of norepinephrine neurotransmitter within pons. During fight or flight response, norepinephrine helps in messaging in SNS (Sympathetic Nervous System). The SNS provide neural extension to all parts of the brain and spinal cord [48]. Within pons, raphe nucleus undergoes the transmission of serotonin neurotransmitter. When stress is combined with depression or anxiety then the mood is regulated by this neurotransmitter [49].

Alternative effects of stress on neural activity and spines within the amygdala and hippocampus have been frequently observed. It leads to cognitive changes. In the hippocampus region and BLA (Basolateral Amygdala) it has been noticed due to stress glutamergic signaling occurs. Due to this increase in amygdala activity which takes place in BLA [50], leads to expression of Brain-derived neurtotrophic factor (BDNF) along with dendritic outgrowth and spine density increases [51] whereas in case of hippocampus BDNF expression decreases along with the hippocampal pyramidal cells receiving aversive sensory information from the entorhinal cortex are also inhibited due to cholinergic input mediated activation of CA1 dendrite targeting interneurons in fear of learning [52]. Stress causes the loss of spines and debranching of dendrites within the medial prefrontal cortex neurons. Chronic stress especially on the distal apical dendritic branches occurs due to the loss of axo-spinous synapses which tends to be over 30%. Treatment with corticosterone for 3weeks has shown reversal of dendrites in the medial prefrontal cortex. Substance P, which is an 11amino acid member of the tachykinin family binds to the receptor of neurokinin-1 during stress and thus produces anxiogenic effects [53-54].

Both, the memory formation and upgradation are damaged due to stress and thus it is the central action of stress on cognition. This is mainly in terms of contextual unrelated memories and complex decision making activities. Higher cognitive processes are likely to become impaired due to stress like goal directed behavior, self-control and working memory. Attention, attention shifting and top down control are some of the appropriate sequences of action for goal directed behavior. It is a process which upholds the important information in mind as in working memory. Stress impairs medial prefrontal cortex dependent cognition with the degree of impairment being correlated to the extent of dendritic shrinkage.

Peptide hormone named ghrelin which is produced by stomach is a stress mediator. After post translational acylation, ghrelin can pass through the blood brain barrier and binds to growth hormone secretagogue 1a in the BLA thereby enhancing fear learning, independent of HPA activation. Neurogenesis is enhanced by ghrelin whereas spatial learning and memory is impaired which is seen in adult mice. Early life stresses can lead to the development of IMD (Irritable bowel syndrome) within a person which is a chronic function disorder [55-57].

3. Ameliorative Effects of Yoga on Stress

3.1. Neurophysiological Effects of Yoga Breathing

Neurophysiological model to analyze the positive effects of yoga breathing was described by Brown and Gerbarg. In this model, Brown and Gerbarg showed or demonstrated that stretch receptors which are present in the alveoli as well as in

the baroreceptors, chemoreceptors and sensors tend to pass relevant information through vagal afferents and brainstem relay stations by the use of respiratory structures. The information which is carried by them is about the state, functioning and activity of respiratory system to the other Central Nervous System (CNS) structure. This leads to the influence of perception, cognition, emotion regulation, somatic expression and behavior. Therefore, this model determines the changes in rate, depth or pattern of breathing information collected through the signals of vagal afferents. These changes gain the highest priority and have rapid widespread effects on brain function [58-60]. Coherent breathing and resonant breathing using a fixed rate of three and a half of six breadths per minute, increases Peripheral Nervous System (PNS) and Heart Rate Variability (HRV) activity. A form of resistance breathing which is Ujjayi (ocean breadth) increases PNS activity. There has been evident research on the increase vagal tone and physiologic relaxation while chanting "OM" which involves slow breathing and airway resistance [61]. These techniques decrease abnormalities in the Autonomic Nervous System (ANS) providing mood improvement, decreased anxiety and improved health. This voluntarily controlled breathing patterns can affect the ANS.

The biological markers of stress are cortisol and brain GABA [62-63]. The level of these markers tend to alter accordingly due to stress i.e. depression and PTSD indicates increased HPA axis activity. This occurs due to increase amount of corticotrophin releasing factor and cortisol [64]. Decrease in the amount of GABA system also occurs due to these disorders. Increase in the amount of cortisol have been reported after interventions using yoga practices. Yoga practices reduces the stress induced allostatic load in GABAergic system and HPA axis [65]. Participants who are experienced in Transcendental Meditation (TM) for 3-5 years had significantly greater decrease in cortisol level than those with 3-4 months of TM experience [66]. From mild depressive symptoms to major depressive disorder, yoga based interventions are effective in treating them. This yoga based interventions includes Sudarshan Kriya Yoga, Iyengar Yoga and Resonance breathing.

The correction of abnormalities related to ANS and Gamma-Aminobutyric acid (GABA) also decreases PTSD symptoms. A network that tend to decrease symptoms by the yoga based practices comprises of interaction of the Prefrontal Cortex (PFC), hippocampus and amygdala which is associated with inputs from ANS and GABA system. Decrease in PFC activation and increase in amygdala activation occurs with failure of PFC to inhibit the amygdala has been seen in response to stressful tests [67]. In response to emotionally laden cues, PFC activity decreases in PTSD subjects, a group known to have reduced PNS activity, as opposed to the increased PFC activation seen in subjects with high PNS activity [68].

The PFC exerts tonic inhibitory control over the amygdala via GABAergic projections. PFC tends to become hypoactive due to conditions such as uncertainty and threat. As a result, over activity of amygdala doesn't occurs with emergence of PTSD symptoms such as hypoarousal and re-expressing. This could represent a neural correlation of the failure of extinction of fear reaction over time as seen in PTSD [69]. PFC activation associated with increased PNS activity could improve inhibitory control over the amygdala via PFC GABA projections, decreasing amygdala over activity and reducing PTSD symptoms.

The Central Extended Amygdala (CEA) receives inhibitory GABAergic projections through the insular cortex. The GABAergic neurons from the CEA projects to the Para branchial Nucleus (PBN) and Dorsal Vagal Complex [70]. In between the temporal and frontal lobes of sylvian fissure the insular cortex is located. The environment and interoceptive information in the form of sensory information are conveyed by the PNS via the Nucleus tractus solitaries (NTS) to the insular cortex, where according to Craig's neuroanatomical theory, a map of the internal state of the body is maintained [71]. For energy mobilization activation of amygdala is necessary, whereas over activation of the amygdala reflects allostatic load associated with the hypervigilant condition (excess arousal) in PTSD [72]. Restoration of strong tonic GABAergic inhibition of the amygdala would result in decreased output from the CEA to the hypothalamus and brainstem nuclei, reducing symptoms of hyperarousal, over reactivity, and re-experiencing in PTSD [73]. Psychological states such as anxiety, depression, and PTSD, associated with PFC hypoactivity and lack of inhibitory control, are characterized by poor habituation to novel neutral stimuli, pre-attentive bias for threat information, deficits in working memory and executive function, and poor affective information processing and regulation [74]. The presence of GABAneurons in the thalamus, insular cortex, amygdala, and hippocampus as well as GABA projections from both the insular cortex and the PFC to the amygdala completes the pathways that would constitute an anatomical substrate for the effects of ANS balance and imbalance on emotion regulation and cognitive function.

3.2. Structural & Behavioral Changes on Memory

Evidence of amygdala with decreased cerebral blood flow and frontal lobes with increased activation have been noticed in those subjects who practiced Iyengar yoga for a period of 12weeks (60min daily) [75]. Research findings shows that after 1training session of Hatha yoga in practitioners, there is a less activation of dorsolateral prefrontal cortex while viewing negative emotional images and distractors, greater stroop task response in the ventrolateral prefrontal cortex with yoga practitioners when presented with emotionally negative disorder images [76]. Also practitioners who received various yogasanas and pranayamas training for 1hr/day, 5days/week for 3 months showed an increase in hippocampal volume [77]. Greater amount of white matter connectivity within the insular cortex is seen in those practitioners for varying types of yoga indicates greater pain tolerance [78].

Uninostril and alternate nostril yoga breathing shows higher scores on a letter cancellation task after alternate and right nostril yoga breathing. It also shows increase in spatial

memory scores in left nostril breathing [79-80]. Increase in verbal task performance is seen in unilateral forced nostril breathing practitioners [81].

4. Conclusion

Stress can be of various forms depending on its nature, influence and duration. Most forms of stress are usually harmful for human beings as they lead to various forms of diseases. Yoga plays a pivotal role in the management of stress within the brain. Hypothalamus controls the entire pathway for stress response. Now, due to the effect of stress alteration do occur within the hippocampus which includes atrophy, neurogenesis and decrease in dendritic branches. Along with hippocampus, the amygdala and prefrontal cortex define the aspects of memory and visual processing within brain. Both of this are inversely proportional to each other with the exposure of stress. In the hippocampus region and BLA (Basolateral Amygdala) it has been noticed due to stress glutamergic signaling occurs. Due to this increase in amygdala activity which takes place in BLA, leads to expression of Brain-derived neurtotrophic factor (BDNF) along with dendritic outgrowth and spine density increases whereas in case of hippocampus BDNF expression decreases along with the hippocampal pyramidal cells receiving aversive sensory information from the entorhinal cortex are also inhibited due to cholinergic input mediated activation of CA1 dendrite targeting interneurons in fear of learning.

This present review also highlights on the stressful condition in today's modern and advancing world and its management by practicing yoga. Yoga has a promising effect on brain complications related to memory and cognition. It comprises of the interaction of the prefrontal cortex, hippocampus and amygdala which is associated with the inputs from ANS and GABA system. Various forms of breathing practices increase PNS activity and improves deformities in the ANS. Yoga practices reduces the stress induced allostatic load in GABAergic system and HPA axis. Direct and indirect effects on the autonomic nervous system and GABA system can be understood through the ameliorative effects of yoga on stress. There are evidences, that increase in PNS and GABA activity is due to the interventions of Vagus Nerve Stimulation (VNS) and yoga. This may be effective in treatment resistant subjects who failed to respond to pharmacologic agents that increase activity in the GABA system. Further, it has also been seen from various studies that different forms of yoga practices could be helpful in the structural activation of brain along with its cognitive function that helps the individual in performing better task as well as increasing and improving their memory. Therefore, it can be concluded from the above study that yoga plays a pivotal role in the improvement of brain physiology affected due to stress interventions.

Funding

For this review no funding is provided by any agency.

Competing Interest

There is no competing interest to be declared.

Declarations of Interest

None.

Acknowledgements

The authors are very much thankful & grateful to the scientists of Life Sciences Research Board (LSRB), Defence Research & Development Organization (DRDO) & Defence Institute of Physiology & Allied Sciences (DIPAS) for their continuous suggestion & guidance throughout the review study.

References

[1] Habib Yaribeygi, Yunes Panahi, Hedayat Sahraei, Thomas P. Johnston, m Amirhossein Sahebkar. The Impact of stress on body function: A Review; 2017.

[2] Clinic, C. (2015, February 5). Stress. Retrieved from Cleveland Clinic: https://my.clevelandclinic.org/health/articles/stress.

[3] Tucker J, Sinclair R, Mohr C, Adler A, Thomas J, Salvi A. A temporal investigation of the direct, interactive, and reverse relations between demand and control and affective strain, Work & Stress. 2008; 22: 81-95.

[4] American Psychiatric Association. Diagnostic and statistical manual of mental disorders (5th Ed.). Washington, DC, USA: American Psychological Association; 2014.

[5] Daly A, Walsh D, Moran R. HRB Statistics Series 4. Activities of Irish Psychiatric Units and Hospitals 2006. Dublin, Ireland: Health Research Board; 2012.

[6] Keil RMK. Coping and Stress: A Conceptual Analysis. Journal of Advanced Nursing. 2004; 45 (6): 659–665.

[7] Stericker S, Show A. The use of treatment facing common mental health problems through stepped care: themed learning from a review of test sites in eth North East, Yorkshire and Humber region. UK: CSIP; 2013. 27.

[8] Selye H. Stress without distress. Philadelphia, PH, USA: J. B. Lippincott Company; 1974. p. 171

[9] Davidson KW, Mostofsky E, Whang W. Don't worry, by happy: Positive affect and reduced 10-year incident coronary heart disease. The Canadian Nova Scotia Health Survey. European Heart Journal. 2010; 31: 1065-1070.

[10] Halbern H, Gallagher M, Kenny, D. Stress assessment and development of a primary care of psychology service. New Psychologists, 2014; 23 (6): 170-8. 30.

[11] Schwarzer R, Schulz U. Stressful life events. In: Weiner IB, Editor. Handbook of psychology (Vol. 9). New Jersey, NJ, USA: John Wiley & Sons, Inc; 2003.

[12] Holmes, TH, Rahe RH. The social adjustment rating scale. Journal of Psychometric Research. 1967; 11: 213-8.

[13] Santorius N, Polansky B, Rutter CH, Holland D. Validity of two versions of the SRRS in the WHO study of mental illness. Medical Psychology. 2013; 34: 291–8. 33.

[14] Schwarzer R. Stress, resources, and proactive coping. Applied Psychology: An International Journal. 2010; 50: 400-7. 34.

[15] Briefenbergen AP, Nimitz JF, George IM. Effects of psychological stress and the workplace: A brief comment on Lazarus' outlook. In Perrewe PL, Editor. Handbook on job stress (3rd Ed.). International Journal of Society, Behaviour and Personality. 2008; 6 (7): 15-20. 35

[16] Schacter DL, Gilbert DT. Wegner DM. Psychology (2nd Ed.). New York, NY, USA: Worth Publishers; 2011. 53.

[17] Connor TM, Halloran DJ, Shanahan F. The stress response and the hypothalamic-pituitary-adrenal axis: From molecule to melancholia. QJM: monthly journal of the Association of Physicians. 2000; 95 (6): 323–333.

[18] Arborelius L, Owens MJ, Plotsky PM, Nemeroff CB. J Endocrinol. 1999; 160: 1–12. [PubMed] [Google Scholar].

[19] Melia KR, Duman RS. Proc Natl Acad Sci USA. 1991; 88: 8382–8386. [PMC free article] [PubMed] [Google Scholar].

[20] Sousa N, Lukoyanov NV, Madeira MD, Almeida OFX, Paula-Barbosa MM. Neuroscience. 2000; 97: 253–266. [PubMed] [Google Scholar].

[21] Luine V, Villages M, Martinex C, McEwen BS. Brain Res. 1994; 639: 167–170. [PubMed] [Google Scholar].

[22] Uno H, Tarara R, Else JG, Suleman MA, Sapolsky RM. J Neurosci. 1989; 9: 1705–1711. [PMC free article] [PubMed] [Google Scholar].

[23] Moghaddam B, Adams B, Verma A, Daly D. J Neurosci. 1997; 17: 2912–2127. [PMC free article] [PubMed] [Google Scholar].

[24] Nibuya M, Morinobu S, Duman RS. J Neurosci. 1995; 15: 7539–7547. [PMC free article] [PubMed] [Google Scholar].

[25] Duman RS, Heninger GR, Nestler EJ. Arch Gen Psychiatry. 1997; 54: 597–606. [PubMed] [Google Scholar].

[26] McEwen BS. Stress and hippocampal plasticity. Ann Rev Neurosci. 1999; 22: 105-22.

[27] Lupien SJ, Lepage M. Stress, memory, and the hippocampus: can't live with it, can't live without it. Behav Brain Res. 2001; 127: 137-58.

[28] Woolley CS, Gould E, McEwen BS. Exposure to excess glucocorticoids alters dendritic morphology of adult hippocampal pyramidal neurons. Brain Res. 1990; 531: 225-31.

[29] Bremner JD. Does stress damage the brain? Biol Psychiatry. 1999; 45: 797-805.

[30] Bliss TV, Lømo T. Long-lasting potentiation of synaptic transmission in the dentate area of the anaesthetized rabbit following stimulation of the perforant path. J Physiol. 1973; 232: 331-56.

[31] LeDoux JL. Ann NY Acad Sci. 1993: 149–157. [PubMed] [Google Scholar].

[32] Davis M. Annu Rev Neurosci. 1992; 15: 353–375. [PubMed] [Google Scholar].

[33] Vyas A, Mitra R, Shankaranarayana Rao BS, Chattarji S. J Neurosci. 2002; 22: 6810–6818. [PMC free article] [PubMed] [Google Scholar].

[34] Vyas A, Pillai AG, Chattarji S. Neuroscience. 2004; 128: 667–673. [PubMed] [Google Scholar].

[35] Vogt BA, Finch DM, Olson CR. Cereb Cortex. 1992; 2: 435–443. [PubMed] [Google Scholar].

[36] Devinsky O, Morrell MJ, Vogt BA. Brain. 1995; 118: 279–306. [PubMed] [Google Scholar].

[37] Scholey A, Gibbs A, Neale C, Perry N, Ossoukhova A, Bilog V, et al. Anti-stress effects of lemon balm-containing foods. Nutrients. 2014; 6: 4805-21.

[38] Cahill L, Gorski L, Le K (2003). Enhanced human memory consolidation with postlearning stress: interaction with thedegree of arousal at encoding. Learn Mem 10: 270–274.

[39] Schwabe L, Wolf OT (2012). Stress modulates the engagement of multiple memory systems in classification learning. J Neurosci 32: 11042–11049.

[40] Sandi C. Stress and cognition. Wiley Interdisciplinary Reviews: Cognitive Science. 2013; 4: 245-61.

[41] Li S, Wang C, Wang W, Dong H, Hou P, Tang Y. Chronic mild stress impairs cognition in mice: from brain homeostasis to behavior. Life Sci. 2008; 82: 93442.

[42] Song L, Che W, Min-Wei W, Murakami Y, Matsumoto K. Impairment of the spatial learning and memory induced by learned helplessness and chronic mild stress. Pharmacol Biochem Behav. 2006; 83: 186-93.

[43] Solerte S, Cravello L, Ferrari E, Fioravanti M. Overproduction of IFN-γ and TNF-α from natural killer (NK) cells is associated with abnormal NK reactivity and cognitive derangement in Alzheimer's disease. Ann NY Acad Sci. 2000; 917: 331-40.

[44] McEwen BS, Sapolsky RM. Stress and cognitive function. Curr Opin Neurobiol. 1995; 5: 205-16.

[45] Gould E, Tanapat P. Stress and hippocampal neurogenesis. Biol Psychiatry. 1999; 46: 1472-9.

[46] Maras PM, Baram TZ. Sculpting the hippocampus from within: Stress, spines, and CRH. Trends in Neurosciences. 2012; 35 (5): 315–24. 57.

[47] McEwen BS, Morrison JH. The Brain on Stress: Vulnerability and Plasticity of the Prefrontal Cortex over the Life Course. Neuron. 2013; 79 (1): 16–29. 58.

[48] Phelps EA. Human emotion and memory: interactions of the amygdala and hippocampal complex. Current Opinions in Neurobiology. 2004; 14: 198-202. 59.

[49] McEwen BS. Brain on stress: How the social environment gets under the skin. Proceedings of the National Academy of Sciences. 2012; 109 (Suppl. 2): 17180–5.

[50] Padival M, Quinette D, Rosenkranz JA (2013). Effects of repeated stress on excitatory drive of basal amygdala neurons in vivo. Neuropsychopharmacology 38: 1748–1762.

[51] Mitra R, Jadhav S, McEwen BS, Vyas A, Chattarji S (2005). Stress duration modulates the spatiotemporal patterns of spine formation in the basolateral amygdala. Proc Natl Acad Sci USA 102: 9371–9376.

[52] Lovett-Barron M, Kaifosh P, Kheirbek MA, Danielson N, Zaremba JD, Reardon TR, et al. (2014). Dendritic inhibition in the hippocampus supports fear learning. Science 343: 857–863.

[53] Ebner K, Singewald GM, Whittle N, FerragutiF, Singewald N (2008). Neurokinin1 receptor antagonism promotes active stress coping via enhanced septal 5-HT transmission. Neuropsychopharmacology 33: 1929–1941. 53.

[54] Singewald N, Chicchi GG, Thurner CC, Tsao KL, Spetea M, Schmidhammer H, et al. (2008). Modulationof basal andstress-induced amygdaloid substance P release by the potent and selective NK1 receptor antagonist L-822429. J Neurochem 106: 2476–2488.

[55] Lutter M, Sakata I, Osborne-Lawrence S, Rovinsky SA, Anderson JG, Jung S, et al. (2008). The orexigenic hormone ghrelin defends against depressive symptoms of chronic stress. Nat Neurosci 11: 752–7.

[56] Meyer RM, Burgos-Robles A, Liu E, Correia SS, Goosens KA (2013). A ghrelingrowth hormone axis drives stress-induced vulnerability to enhanced fear. Mol Psychiatry. doi: 10.1038/mp.2013.135.

[57] Zhao Z, Liu H, Xiao K, Yu M, Cui L, Zhu Q, et al. (2014). Gherlin administration enhances neurogenesis but impairs spatial learning and memory in adult mice. Neuroscience 257: 175–185.

[58] Brown RP, Gerbarg PL. Sudarshan Kriya yogic breathing in the treatment of stress, anxiety, and depression: Part II-Clinical applications and guidelines. J Altern Complement Med 2005; 11: 711–7.

[59] Brown RP, Gerbarg PL. Sudarshan Kriya yogic breathing in the treatment of stress, anxiety, and depression: part I-neurophysiologic model. J Altern Complement Med 2005; 11: 189–201.

[60] Brown RP, Gerbarg PL. Yoga breathing, meditation and longevity. Annals New York Academy of Science 2009; 1172: 54–62.

[61] Telles S, Nagarathna R, Nagendra HR. Autonomic changes during ‘‘OM’’ meditation. Indian J Physiol Pharmacol 1995; 39: 418–20.

[62] Streeter CC, Whitfield TH, Saper RB, Owen E, Gensler M, Turnquist N, etal. The effect of yoga and walking on brain GABA levels. San Francisco, CA: American Psychiatric Association Annual Meeting; 2009.

[63] Pike JL, Smith TL, Hauger RL, Nicassio PM, Patterson TL, McClintick J, et al. Chronic life stress alters sympathetic, neuroendocrine, and immune responsivity to an acute psychological stressor in humans. Psychosom Med 1997; 59: 447–57.

[64] Bremner JD, Licinio J, Darnell A, Krystal JH, Owens MJ, Southwick SM, et al. Elevated CSF corticotropin-releasing factor concentrations in posttraumatic stress disorder. Am J Psychiatry 1997; 154: 624–9.

[65] Jevning R, Wilson AF, Davidson JM. Adrenocortical activity during meditation. Horm Behav 1978; 10: 54–60.

[66] MacLean CR, Walton KG, Wenneberg SR, Levitsky DK, Mandarino JP, Waziri R, et al. Effects of the transcendental meditation program on adaptive mechanisms: changes in hormone levels and responses to stress after 4 months of practice. Psychoneuroendocrinology 1997; 22: 277–95.

[67] Thayer JF, Sternberg E. Beyond heart rate variability: vagal regulation of allostatic systems. Ann N Y Acad Sci 2006; 1088: 361–72.

[68] Lane RD, McRae K, Reiman EM, Chen K, Ahern GL, Thayer JF. Neural correlates of heart rate variability during emotion. Neuroimage 2009; 44: 213–22.

[69] Bremner JD, Elzinga B, Schmahl C, Vermetten E. Structural and functional plasticity of the human brain in posttraumatic stress disorder. Prog Brain Res 2008; 167: 171–86.

[70] Sun N, Yi H, Cassell MD. Evidence for a GABAergic interface between cortical afferents and brainstem projection neurons in the rat central extended amygdala. J Comp Neurol 1994; 340: 43–64.

[71] Craig AD. Interoception and Emotion. In: Lewis M, Haviland-Jones JM, Barrett LF, editors. Handbook of Emotions. Third Edition: The Guilford Press; 2008. p. 272–88.

[72] Shin LM, Orr SP, Carson MA, Rauch SL, Macklin ML, Lasko NB, et al. Regional cerebral blood flow in the amygdala and medial prefrontal cortex during traumaticimagery inmale and female Vietnam veterans with PTSD. Arch Gen Psychiatry 2004; 61: 168–76.

[73] Koob G, Volkow N. Neurocircuitry of Addition. Neuropsychopharmacology 2010; 35: 217–38.

[74] Thayer JF, Brosschot JF. Psychosomatics and psychopathology: lookingup and down from the brain. Psychoneuroendocrinology 2005; 30: 1050–8.

[75] Cohen Debbie L, Wintering N, Tolles V, Townsend RR, Farrar JT, Galantino ML. Cerebral blood flow effects of yoga training: preliminary evaluation of 4 cases. J Altern Complementary Med 2009; 15 (1): 9-14.

[76] Froeliger Brett E, Garland EL, Modlin LA, McClernon FJ. Neurocognitive correlates of the effects of yoga meditation practice on emotion and cognition: a pilot study.

[77] Hariprasad VR, Varambally S, Shivakumar V, Kalmady SV, Venkatasubramanian G, Gangadhar BN. Yoga increases the volume of the hippocampus in elderly subjects. Indian J Psychiatry 2013; 55 (3): S394.

[78] Villemure C, Ceko M, Cotton VA, Bushnell MA. Insular cortex mediates increased pain tolerance in yoga practitioners. Cereb Cortex May 21st 2013.

[79] Telles S, Joshi Meesha. Yoga breathing through a particular nostril is associated with contralateral event-related potential changes. Int J Yoga 2012; 5 (2); 102.

[80] Naveen KV, Nagarathna R, Nagendra HR, Telles S. Yoga breathing through a particular nostril increases spatial memory scores without lateralized effects. Psychol Reports 1997; 81 (2): 555-61.

[81] Jella SA, Shannahoff-khalsa David. The effects of unilateral forced nostril breathing on cognitive performance. Int J Neurosci 1993; 73 (1): 61-8.