

Effect of Meditation on Stress-Induced Changes in Cognitive Functions

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Abstract

Objectives: The objective of this study was to study the effects of meditation on stress-induced changes in cognitive functions.

Methods: The study was conducted on 32 healthy adult male student volunteers who had never practiced meditation before the study. The study consisted of practicing 20 minutes of guided meditation and administration of psychologic stress to the subjects. The psychologic stress was administered to the subjects by asking them to play a (preselected) stressful computer game. The subjects were asked to meditate either before or after the administration of psychologic stress. For the control group measurements, the subjects were asked to wait quietly for an equivalent period of meditation time.

Outcome measures: The outcome measures were galvanic skin response (GSR), heart rate (HR), electromyography (EMG), sympathetic reactivity (QTc/QS2 ratio), cortisol, and acute psychologic stress scores. The central nervous system functions were assessed using Wechsler memory scale and visual-choice reaction time (VCRT). These parameters were measured both at the beginning and at the end of the intervention, using a pre-post experimental test design.

Results: Computer game stress was associated with a significant increase in physiologic (GSR, EMG, HR, QTc/QS2) and psychologic (acute stress questionnaire scores) markers of stress. Meditation was associated with relaxation (significant decrease in GSR, EMG, QTc/QS2, and acute stress questionnaire scores). Meditation, if practiced before the stressful event, reduced the adverse effects of stress. Memory quotient significantly increased, whereas cortisol level decreased after both stress and meditation. VCRT showed no significant change.

Conclusions: Practice of meditation produced a relaxation response even in the young adult subjects who had never practiced meditation before. The practice of meditation reduced the physiologic stress responses without taking away the beneficial effect of stress, namely, improved memory scores.

Introduction

MENTAL STRESS HAS BEEN REPORTED to impair cognitive functions.¹ This may be relevant to the performance of students, office workers, and many others for whom optimal alertness, concentration, and memory are significant variables affecting their performance. The adverse effects of stress on cognitive performance may be mediated by the sympatho-adrenal system and the hypothalamo-hypophysial-adrenal axis.^{2–5}

Increase in the concentration of glucocorticoids has been reported to be associated with deficits in both memory and attention,^{6,7} giving rise to the speculation that the hippocampus-based cognitive functions may be particularly vulnerable to the deleterious effects of glucocorticoids.^{8,9} Poor

cognitive performance has also been reported in hypercortisolemia,¹⁰ dexamethasone treatment,¹¹ and a high oral dose of cortisol (to approximate major stress).¹²

Very low and very high levels of stresses impair performance, whereas moderate stress is known to improve performance. These effects may be mediated by differential binding of cortisol to high-affinity mineralocorticoid receptors (MR) or lower-affinity glucocorticoid receptors (GR).^{13,14} Co-localization of these receptors on hippocampal neurons may mediate differential effects of cortisol that are site specific, long lasting, and context dependent.

The relaxing effect of meditation¹⁵ suggests that stress-induced impairment in cognitive function may be influenced favorably by meditation. The present study was designed to explore the effects of stress on cognitive functions and to

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study the effects of meditation on the aforesaid interaction. Literature search has not revealed any previous comparable studies.

Materials and Methods

The study was conducted on 32 young adult male volunteers (age 23–30 years; age mean \pm standard deviation: 27.3 ± 1.8). Subjects with a history of practicing and/or learning meditation or any other relaxation technique, indulging in substance abuse, smokers, alcoholics, or those known to be having any disease or undergoing treatment for any medical condition were excluded. Only male subjects were taken because females have a different level of stress and stress reactivity during different phases of the menstrual cycle. All the experiments were done in the afternoon to minimize the effects of diurnal variation in cortisol levels on the results of the study.

Ethical clearance for the study was obtained from the Ethics Committee for Human Subjects of All India Institute of Medical Sciences. The subjects were called for the study on 2 separate days. On the first visit, each subject was briefed about the study using a subject information sheet that contained information about the objectives, methods, and parametric evaluation of the study. Subjects were also informed about the study methodology and thereafter an informed written consent for participating in the study was obtained from them.

After obtaining the consent for participation in the study, the subjects were familiarized with the modalities and systematic procedure for conduct of the study. To familiarize the subjects with computer games, all of the subjects were made to play a few computer games, while their electromyography (EMG), electrocardiography (ECG), galvanic skin response (GSR), and phonocardiography recordings were taken. Out of the set of computer games played by each subject, the computer game that gave the subject a feeling of maximum stress resulting in an increase in GSR levels as well as heart rate was identified as the stressor for that particular subject.¹⁶ This was repeated for all the subjects and stressor games identified for each subject individually.

On the second visit, subjects were divided randomly using computer-generated random numbers into four groups of 8 subjects each (Box 1). Intervention Groups 1 and 3 were administered a session of meditation, whereas the Control Groups 2 and 4 had a period of waiting for 20 minutes (used as a control for meditation). Meditation or Waiting was administered either before or after the subject had played an identified stressor: a stressful computer game. Test parameters (GSR, heart rate [HR], EMG, sympathetic reactivity (QTc/QS2 ratio), cortisol and acute psychologic stress scores) were recorded both in the baseline and the intervention (Meditation or Waiting) case administered before or after the psychologic stress was provided, depending upon the group (Box 1).

Computer game as a stressor

Computer games usually give immense pleasure after a win. However, in the initial stages of the game when a player suffers repeated defeats or constraints, the game becomes very stressful.¹⁶ The games selected for the study required the subject to play with a very few number of keys, so that he became easily familiar with the playing of the game and

BOX 1. OVERVIEW OF STUDY DESIGN

Group 1 Stress preceded by meditation	Group 2 Stress preceded by period of waiting	Group 3 Stress followed by meditation	Group 4 Stress followed by period of waiting
Blood sample for cortisol			
Acute Stress Questionnaires			
Assessment of Cognitive Functions and Visual Choice Reaction Time			
	Period of Waiting	Stressful event	Stressful event
Recording of stress parameters	Stressful event	Stressful event	Period of Waiting
Blood sample for cortisol			
Acute Stress Questionnaire			
Assessment of Cognitive Functions and Visual Choice Reaction Time			

rapidly reached a stage where he is able to prevent repeated defeats or losses. However, the game that a given subject is not able to master gives him stress and was selected as the stressor for that subject. Stressor games were identified individually for all the subjects of the study.

Measurement of stress

Recordings were done using Biopac Student Lab PRO[®] Software version 3.6.7 and MP30 Hardware from BIOPAC Systems, Inc. The following parameters were recorded:

1. ECG (0.5–35 Hz) was continuously recorded using Lead-2, sample rate at 200/sec, gain \times 2000. The HR was calculated from the R-waves using mean value of beats per minute for artifact-free 300-second graph for each stage of recording.
2. Phonocardiography (20–100 Hz) was done at sampling rate 200/second and gain \times 2000. This was done along with ECG to measure QTc/QS2 ratio.
3. QTc/QS2 as the ratio of electrical systole to the total electromechanical systole was used as an index of sympathetic discharge to the heart¹⁷ as determined from 10 cardiac cycles¹⁸ using a software. QTc was corrected QT, measured from the beginning of ventricular depolarization up to the end of repolarization. QS2 was measured from Q wave to second heart sound.
4. EMG (30–500 Hz) was recorded from the temporalis and the masseter muscles, at sampling rate 200 Hz, gain \times 2500 with Ag-AgCl surface electrodes with 10-mm contact area. The raw data were first rectified and then integrated.
5. GSR (0–35 Hz): Two (2) Ag/AgCl electrodes were tied around the index and middle finger of the left hand to record the GSR, which is a relatively reliable index for

sweat gland activity and changes in activation level of the sympathetic nervous system.

6. Serum cortisol was estimated using commercial enzyme-linked immunosorbent assay kits (DRG International Inc., USA). The interassay and the intra-assay coefficients of variation were less than 5.4% and 2.8%, respectively. An acute stress questionnaire was used to assess mental and emotional stress.¹⁹ The assessment of cognitive function was done using the Wechsler Memory Scale. The cognitive functions tested were orientation, mental control, logical memory, attention, concentration, visual reproduction, and associate learning. The scores were corrected for age and the Memory Quotient (MQ) was calculated using the table of Mental Equivalents. The visual-choice reaction time (VCRT) of the subject was measured using Donders Reaction Time from computerized software Psych/Lab™ Software.

Meditation

Initially, the subjects were taught guided meditation by a trained instructor. The subjects were given a practice session of meditation and they were instructed about the importance of relaxing completely. This was followed by subject lead meditation sessions of approximately 20 minutes each with eyes closed throughout. The instructions were received by the subject through prerecorded messages relayed to each subject through a headphone connected to a Walkman. For the first few minutes, the subject was instructed to concentrate on his breathing pattern. This was followed by an instruction that with each breath, the subject was to experience and give autosuggestion of peace, stillness, and thoughtlessness to himself. As soon as the mind reached the thoughtless or peaceful stage, he was instructed to observe and maintain the status quo.

The subjects were also given an option to replay the tape if they felt that they could not follow the instructions and hence could not relax completely.

Period of waiting

During the period of waiting, subjects in groups 2 and 4 were seated comfortably with eyes open under conditions similar to those in the meditation groups 1 and 3.

Statistical analysis was done using parametric tests with SPSS 10.0 software. Comparison of pre and post stress and meditation data was done by using paired *t* test. Comparison between postmeditation and postwaiting was done by independent samples *t* test. Differences were considered significant if the *p*-value was less than 0.05.

Results

The measurement of cortisol, MQ, and VCRT was done before and after both of the interventions were completed (Table 1). The rest of the parameters were monitored continuously.

Effect of stress

Stress was associated with a significant increase in GSR, EMG activity, HR, QTc/QS2, scores on the acute stress questionnaire, and MQ. Moreover, a significant decrease in cortisol and no significant change in VCRT was found (Table 1). When MQ was analyzed for its individual components, it

TABLE 1. EFFECT OF STRESS (*N* = 24)

Parameter	Prestress	Poststress
GSR (micromho)	1.843 ± 1.451	2.659 ± 1.968***
EMG (mV-sec)	2.975 ± 0.991	3.814 ± 2.556**
HR (beats/min)	81.2 ± 9.9	85.4 ± 9.9***
QTc/QS2	1.081 ± 0.093	1.162 ± 0.086***
Cortisol (ng/mL) (<i>n</i> = 16)	157.7 ± 53.0	125.1 ± 43.9**
Acute Stress Scores	26.1 ± 7.8	28.3 ± 8.6***
VCRT (millisec)	379.6 ± 49.0	377.4 ± 50.9
Memory Quotient	114.4 ± 18.2	124.8 ± 17.9**

Asterisks represent significance between pre versus post values: ***p* < 0.01; ****p* < 0.001.

GSR, galvanic skin response; EMG, electromyography; HR, heart rate; QTc/QS2 ratio, sympathetic reactivity; VCRT, visual-choice reaction time.

was found that stress increased the scores of logical memory, visual reproduction, and associate learning, but decreased the performance on mental control. There were no changes in the current information, orientation, attention, and concentration scores.

The effect of stress did not differ in group 2 (which had the waiting period before the stress) from that in groups 3 and 4 (which had either meditation or waiting after the stress). Therefore, the results for the effect of stress on GSR, EMG, HR, and QTc/QS2 from groups 2, 3, and 4 have been pooled. Group 1 was not included in the pool because in this group subjects meditated before stress. Hence, the isolated effect of stress alone without meditation could not be observed.

Effect of meditation

Meditation in 8 subjects from group 1 (Table 2) was associated with a significant decrease in GSR, EMG activity, and QTc/QS2 when compared with the pre meditation period and as well as compared to control (group 2). No significant changes in HR were observed.

Effect of stress when preceded by meditation

When meditation preceded stress (group 1), stress was associated with a significant increase (as compared to baseline) in GSR, EMG, HR, and MQ (Table 3). There was a significant decrease in acute stress questionnaire scores, cortisol, and VCRT. There was no significant change in QTc/QS2. When MQ was analyzed for its individual components, it was found that logical memory, visual reproduction, and associate learning showed significant increase as compared to baseline.

When a period of waiting preceded stress (group 2), stress was associated with a significant increase (as compared to group 1) in EMG and visual reproduction score and a decrease in sympathetic reactivity and mental control scores (Table 3).

Effect of stress when followed by meditation

Stress followed by meditation (group 3) was associated with a significant decrease (as compared to baseline) in GSR, EMG, cortisol, and acute stress questionnaire scores (Table 4) and a significant increase in MQ scores. Mental control de-

TABLE 2. EFFECT OF MEDITATION (N=16)

	<i>Pre meditation</i>	<i>Post meditation</i>	<i>Pre waiting</i>	<i>Post waiting</i>
GSR (micromho)	2.035 ± 1.62	0.895 ± 1.068** www	1.793 ± 1.662	1.427 ± 1.228*
EMG (mV-sec)	2.580 ± 0.275	2.282 ± 0.211* w	2.609 ± 0.248	2.650 ± 0.427
HR (beats/min)	78.6 ± 14.6	77.5 ± 14.4	79.0 ± 12.9	78.2 ± 11.3
QTc/QS2	1.117 ± 0.11	1.086 ± 0.121* w	1.044 ± 0.098	1.062 ± 0.086

Asterisks represent significance between pre versus post values; * $p < 0.05$; ** $p < 0.01$. w represents significance between meditation and waiting; w, $p < 0.05$; www, $p < 0.001$.

GSR, galvanic skin response; EMG, electromyography; HR, heart rate; QTc/QS2 ratio, sympathetic reactivity.

creased, whereas logical memory showed a significant increase. As compared to control (group 4), EMG was significantly higher.

Discussion

Computer games acted as a stressor as indicated by physiologic changes known to be associated with stress in terms of GSR, EMG, HR, QTc/QS2, and stress questionnaire scores.¹⁶ Serum concentration of cortisol, commonly considered a biochemical marker of stress, showed a significant decrease. Although all the experiments were done in the afternoon, the diurnal and pulsatile changes in cortisol levels could have confounded the results. Skosnik et al.²⁰ also reported a decrease in cortisol level with computer game stress, which correlated positively with cognitive scores. In the present study, however, no correlation between cortisol levels and cognitive function could be found.

The significant increase in MQ after stress in the present study could represent the beneficial effect of stress on cognitive functions representing the midportion of the inverted U relationship between stress and performance. Henckens et al.²¹ demonstrated that acute stress is accompanied by a shift into a hypervigilant mode of sensory processing which may improve memory. Substantial evidence from animal

studies suggests that enhanced memory associated with emotional arousal results from an activation of β -adrenergic system during and after an emotional experience.²² Gold et al.²³ reported that stress-related release of catecholamine induced relatively modest increase in glucose concentrations, leading to enhanced learning and memory processes in rodents and humans.

The increase in logical memory, visual reproduction, and associate learning and decrease in mental control suggests that the same stressor could affect performance for different components of cognitive function. Using functional magnetic resonance imaging, Henckens et al.²¹ reported that acute stress profoundly affects the neural substrates of memory formation in a region-specific manner.

The activation level of hippocampal neurons depends on a finely tuned balance of MR- and GR-mediated actions.²⁴ The MR- and GR-mediated cellular events could be among the factors determining the function of hippocampal networks. The role of altered cortisol levels on long-term potentiation and primed burst potentiation in hippocampus, which is the synaptic model for learning and memory processes, needs to be studied in future.

Meditation alone, as studied in group 1 subjects, as well as in comparison to the waiting group, had a significant relaxing effect. These results are on the basis of only 20 minutes

TABLE 3. EFFECT OF STRESS BEFORE VERSUS AFTER MEDITATION (N=16)

<i>Parameter</i>	<i>Stress preceded by meditation (group 1)</i>		<i>Stress followed by meditation (group 3)</i>	
	<i>Prestress</i>	<i>Poststress</i>	<i>Prestress</i>	<i>Poststress</i>
GSR (micromho)	2.0 ± 1.6	3.3 ± 2.8*	2.5 ± 1.3	1.4 ± 1.1*** ++
EMG (mV-sec)	2.580 ± 0.275	3.398 ± 0.672*** w	3.753 ± 1.441	2.917 ± 0.680** w, +++
HR (beats/min)	78.6 ± 14.6	82.6 ± 14.7*	80.2 ± 9.8	82.3 ± 11.4
QTc/QS2	1.117 ± 0.119	1.135 ± 0.118 w	1.089 ± 0.124	1.068 ± 0.067
Cortisol (ng/mL)	160.0 ± 52.9	109.1 ± 34.0*	135.1 ± 33.2	96.4 ± 28.3***
Acute Stress Scores	31.6 ± 5.5	28.4 ± 4.2**	26.0 ± 10.7	23.8 ± 7.6*
VCRT (millisec)	379.6 ± 41.7	331.2 ± 37.6***	343.0 ± 64.5	361.6 ± 59.6 ++
Memory Quotient	112.5 ± 8.8	129.1 ± 11.3***	128.1 ± 19.5	138.3 ± 21.5*
Current information	6 ± 0	6 ± 0	6 ± 0	6 ± 0
Orientation	5 ± 0	5 ± 0	5 ± 0	5 ± 0
Mental control	7.125 ± 1.80	6.625 ± 1.22 w	8.625 ± 0.71	7.5 ± 1.32*
Logical memory	11.75 ± 3.19	14.75 ± 3.18***	12.125 ± 3.78	15.625 ± 4.31**
Attention & concentration	11.5 ± 1.42	12 ± 1.54	13.5 ± 12.12	13.625 ± 2
Visual reproduction	11.5 ± 2.20	13.125 ± 1.27** w	12.5 ± 1.73	13 ± 1.32
Associate learning	18 ± 1.99	20.75 ± 0.44**	19.625 ± 2.30	20.5 ± 1.01

Asterisks represent statistical significance between pre versus post values; * $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$; + represents statistical significance between meditation followed by stress versus stress followed by meditation; ++ $p < 0.01$; +++ $p < 0.001$; w represents significance between meditation followed by stress vs. waiting followed by stress; w, $p < 0.05$.

GSR, galvanic skin response; EMG, electromyography; HR, heart rate; QTc/QS2 ratio, sympathetic reactivity; VCRT, visual-choice reaction time.

TABLE 4. EFFECT OF STRESS BEFORE VERSUS AFTER WAITING (N=16)

Parameter	Stress preceded by waiting (Group 2)		Stress followed by waiting (Group 4)	
	Pre	Post	Pre	Post
GSR (micromho)	1.793 ± 0.662	2.948 ± 2.667***	1.272 ± 1.291	1.098 ± 1.261*
EMG (mV-sec)	2.609 ± 0.248	2.893 ± 0.377*	2.565 ± 0.225	2.610 ± 0.358
HR (beats/min)	79.0 ± 12.9	82.6 ± 11.8*	84.5 ± 6.3	87.5 ± 8.6*
QTc/QS2	1.044 ± 0.098	1.151 ± 0.115***	1.111 ± 0.025	1.140 ± 0.022***
Cortisol (ng/mL)	145.6 ± 68.1	130.4 ± 53.7	169.8 ± 32.4	119.9 ± 34.3**
Acute Stress Scores	26.8 ± 4.6	28.4 ± 5.2*	25.6 ± 7.9	26.0 ± 6.1
VCRT (millisec)	366.9 ± 52.9	363.3 ± 46.4	392.3 ± 44.5	391.6 ± 54.4
Memory Quotient	104.8 ± 20.5	115.9 ± 19.0*	124.0 ± 8.8	133.8 ± 11.9
Current information	5.625 ± 0.53	5.625 ± 0.53	5.875 ± 0.33	5.875 ± 0.33
Orientation	5 ± 0	5 ± 0	5 ± 0	5 ± 0
Mental control	8.25 ± 1	6.375 ± 0.73	8.125 ± 1.20	7.125 ± 1.41
Logical memory	8.875 ± 4.80	12.375 ± 4.02	13 ± 2.11	15.75 ± 1.32
Attention & concentration	10.875 ± 1.94	11.375 ± 2.18	12.75 ± 2.29	13.125 ± 2.44
Visual reproduction	12 ± 1.72	12.5 ± 1.66	12.5 ± 1	13.125 ± 1.05
Associate learning	14.625 ± 4.55	18.125 ± 3.78	19.125 ± 1.36	20.125 ± 1.99

Asterisks represent significance between pre versus post values: * $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$. There was no statistical significance between waiting followed by stress versus stress followed by waiting in any of the parameters.

GSR, galvanic skin response; EMG, electromyography; HR, heart rate; QTc/QS2 ratio, sympathetic reactivity; VCRT, visual-choice reaction time.

of meditation done by the subjects for the second time in their lives, the first time being during the practice session. Since (1) it was guided meditation, (2) subjects were allowed to discontinue if they reported that they could not follow meditation as suggested by the recorded tape, and (3) marked physiologic relaxation was recorded, it is assumed that it was a successful relaxation experience for the subject. This suggests the effectiveness of meditation in producing relaxation, which just sitting quietly is unable to produce. This beneficial effect of meditation can be studied in future studies on experienced as well as regular meditators.

Meditation is known to induce a set of integrated physiologic changes termed the relaxation response.¹⁵ Using magnetic resonance imaging, Lazar et al.²⁵ in 2000 have shown that the practice of meditation activates neural structures involved in attention and control of the autonomic nervous system, which may lead to improved cognitive function. Frumkin and Pagano²⁶ reported improved performance on a memory task after transcendental meditation. Similar improvements with meditation were found in the present study. Appelle and Oswald²⁷ reported a decrease in reaction time in meditators as compared to nonmeditators. In the present study, no change in reaction time was observed that could have resulted from difference in the method and duration of meditation.

Xiong and Doraiswamy²⁸ have summarized various health benefits of meditation suggesting preserved cognition and prevention of dementia. While the mechanisms remain investigational, studies show that meditation may reduce cortisol secretion, and this could have neuroprotective effects, potentially via elevating levels of brain-derived neurotrophic factor, which could play a role in brain aging and mental fitness. Use of meditation in psychologic and medical practice for stress management as well as a variety of physical and mental disorders is documented.²⁹ Physiologic benefits help yoga practitioners show greater resilience in stressful conditions.³⁰ The present study suggests that prac-

tice of meditation may benefit young adult subjects in reversing the acute effects of stress.

Experimental stress resulted in significant sympathetic activation and increased subjective scoring of stress. Meditation produced significant reduction in sympathetic responses. When both stress and meditation were given, the sequence made some difference. The effect of the variable presented later was predominant in case of physiologic responses related to sympathetic activation, but cortisol levels and acute stress scores were lower and MQ was higher than in the controls when both stress and meditation were given, regardless of the sequence. Stress alone did not affect VCRT significantly, but when stress was preceded by meditation, stress reduced VCRT significantly. From these results, it is apparent that practice of meditation after stress may ameliorate some negative effects of stress. However, this needs to be tested in a larger number of subjects regarding effects of meditation on acute as well as chronic stress.

The MQ scores increased significantly when meditation was practiced either before or after stress. Thus, meditation did not impair the beneficial effect of stress on mental performance. Hence, use of meditation in demanding situations (e.g., examinations) may reduce the physiologic and subjective manifestations of acute stress while retaining the beneficial effect of stress on memory.

Finally, the results of the present study may be specific to the type of subjects chosen, their perception of computer games in the laboratory settings, as well as their specific interest in learning and practicing meditation. Studies on the long-term effect of practicing meditation in large number of subjects needs to be done to substantiate the findings of the present study.

Conclusions

Stress was induced by playing computer games in young adult male subjects. Acute stress and meditation produced

predictable physiologic and psychologic changes. Relaxation induced by meditation had physiologic and psychologic effects essentially opposite to those of stress. Meditation had more favorable effects when it preceded the stress than when it followed the stress.

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Disclosure Statement

No competing financial interests exist.

References

- de Kloet ER. Stress in the brain. *Eur J Pharmacol* 2000; 405:187–198.
- Valentino RJ, Van Bockstaele E. Convergent regulation of locus coeruleus activity as an adaptive response to stress. *Eur J Pharmacol* 2008;583:194–203.
- Sara SJ. The locus coeruleus and noradrenergic modulation of cognition. *Nat Rev Neurosci* 2009;10:211–223.
- Lupien SJ, Lepage M. Stress, memory, and the hippocampus: Can't live with it, can't live without it. *Behav Brain Res* 2001;127:137–158.
- McEwen BS. Physiology and neurobiology of stress and adaptation: Central role of the brain. *Physiol Rev* 2007;87: 873–904.
- Oitzl M, Flutterm, M, de Kloet ER. The effect of corticosterone on reactivity to spatial novelty is mediated by central mineralocorticoid receptors. *Eur J Neurosci* 1994;6:1072–1079.
- Vedhara K, Hyde J, Gilchrist ID, Tytherleigh M. Acute stress, memory, attention and cortisol. *Psychoneuroendocrinology* 2000;25:535–549.
- McEwen BS, Sapolsky RM. Stress and cognitive function. *Curr Opin Neurobiol* 1995;5:205–216.
- Wolkowitz OM, Reus VI, Canick J, et al. Glucocorticoid medication, memory and steroid: Psychosis in medical illness. *Ann N Y Acad Sci* 1997;14:823:81–96.
- Mauri M, Sinforiani E, Bono G, et al. Memory impairment in Cushing's disease. *Acta Neurol Scand* 1993;87:52–55.
- Newcomer JW, Craft S, Hershey T, et al. Glucocorticoid induced impairment in declarative memory performance in adult humans. *J Neurosci* 1994;99:220–232.
- Newcomer JW, Selke G, Melson AK, et al. Decreased memory performance in healthy humans induced by stress-level cortisol treatment. *Arch Gen Psychiatry* 1999;56:527–533.
- Reul JM, De Kloet ER. Two receptor systems for corticosterone in rat brain: Microdistribution and differential occupation. *Endocrinology* 1985;117:2505–2511.
- de Kloet ER, Vreugdenhil E, Oitzl MS, Joëls M. Brain corticosteroid receptor balance in health and disease. *Endocr Rev* 1998;19:269–301.
- Wallace RK. Physiological effects of transcendental meditation. *Science* 1970;167:1751–1754.
- Sharma R, Khera S, Mohan A, et al. Assessment of computer game as a psychological stressor. *Ind J Physiol Pharmacol* 2006;50:367–374.
- De Caprio L, Ferro G, Cuomo S, et al. QT/QTc ratio as an index of autonomic tone changes. *Am J Cardiol* 1984;53:818–822.
- Boudoulas H, Geleris P, Lewis RP, Rittgers SE. Linear relationship between electrical systole, mechanical systole and heart rate. *Chest* 1981;80:613–617.
- Cardena E, Koopman C, Classen C, et al. Psychometric properties of the Stanford Acute Stress Reaction Questionnaire (SASRQ): A valid and reliable measure of acute stress. *J Trauma Stress* 2000;13:719–734.
- Skosnik PD, Chatterton RT Jr, Swisher T, Pask S. Modulation of attentional inhibition by norepinephrine and cortisol after psychological stress. *Int J Psychophysiol* 2000;36:59–68.
- Henckens MJAG, Hermans EJ, Pu Z, et al. Stressed memories: How acute stress affects memory formation in humans. *J Neurosci* 2009;29:10111–10119.
- Cahill L, Prins B, Weber M, McGaugh JL. Beta-adrenergic activation and memory for emotional events. *Nature* 1994; 371:702–704.
- Gold AE, MacLeod KM, Deary IJ, Frier BM. Hypoglycemia-induced cognitive dysfunction in diabetes mellitus: Effect of hypoglycemia unawareness. *Physiol Behav* 1995;58:501–511.
- Joels M, De Kloet ER. Mineralocorticoid and glucocorticoid receptors in the brain: Implications for ion permeability and transmitter systems. *Prog Neurobiol* 1994;43:1–36.
- Lazar SW, Bush G, Gollub RL, et al. Functional brain mapping of the relaxation response and meditation. *Neuroreport* 2000;15:11:1581–1585.
- Frumkin LR, Pagano RR. The effect of transcendental meditation on iconic memory. *Biofeedback Self-Regul* 1979; 4:313–322.
- Appelle S, Oswald LE. Simple reaction time as a function of alertness and prior mental activity. *Perceptual Motor Skills* 1974;38:1263–1268.
- Xiong GL, Doraiswamy M. Does Meditation enhance cognition and brain plasticity? *Ann NY Acad Sci* 2009;1172:63–69.
- Newberg AB, Iversen J. The neural basis of the complex mental task of meditation: Neurotransmitter and neurochemical considerations. *Med Hypothesis* 2003;61:282–291.
- Parshad O. Role of yoga in stress management. *West Indian Med J* 2004;53:191–194.

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