Review Article

Advancement in Yoga and Physical Therapy

''The Science Behind Raj Yoga Meditation-A Narrative Review"

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Abstract

On invitation to write an article on Yoga here we decided to write on science of Raj yoga meditation (RM). Normally besides the classic asana-based meditations there are 3 kinds of meditations i) Contemplation/ Concentration based ii) Mindful based iii) Love kind of meditation. Despite so much scepticism the western world found results with meditation and according to them Mindful based meditation is the true one as they get results of increased Concentration, attention with them. Nevertheless, here we have focused on description of science behind RM. Here thus we have detailed a narrative review. The authors performed an extensive search on various platforms like PubMed; Google scholar; Web of Science; Embassy; Cochrane review library utilizing the MeSH terms; "Raj yoga meditation (RM)"; "Cognitive brain"; "attention systems ''emotional brain'' anandamide; coronary artery disease (CAD); drug addiction; obesity; neuropsychiatric disorders to assess the efficacy of RM in treatment of various diseases" from 2000 till date in 2023. We have detailed anandamide as it is the basic hormone liberated subsequent to bliss and helps in treatment of substance use disorders(SUD) .Further the basis of different neurotransmitters like serotonin, vasopressin ,GABA etc are detailed for laymen along with professionals to get insight into RM as well as its benefits which can aid in positive thinking and power of belief that I can do it can help one attain the impossible as I am possible. So much so that if 2 group of patients are told one that you will develop stomach cancer after 5 yrs, the one told of cancer actually develops it while say 2 patients knee surgery for pain both given anaesthesia one operated other also feels relief of pain despite no surgery with the power of mind, hence the role of positive thinking has kept a lot of cancer patients alive despite given a prognosis of 6mths life.

Keywords: Raj yoga Meditation, Anandamide; Neuropsychiatric Disorders; Coronary Artery Disease Substance Use Disorders

1. Introduction

Here on being invited to write an article on yoga we decided to describe raj yoga meditation in detail to start with we know WHO definition of health is a state of complete physical, mental and social wellbeing and not merely the absence of disease or infirmity. We all realize that the worldwide incidence of depression is increasing. Furthermore, with so much unhappiness the immune system is further compromised which explains why the incidence of autoimmune diseases is escalating. Moreover, a lot of diseases whose ethology is not known is termed as psychosomatic. Hence here our aim is to describe the science correlated with raj yoga meditation to understand how we souls can reach back in a happy state.

To get insight it is significant to know what the central nervous system [CNS]is comprised of. Of this the brain is a physical organ that can be visualized whose anatomy and physiology can be clearly identified whereas what is the aatma/soul/consciousness is something which despite all their efforts scientists have been unable to capture even in patients on their death bed. The supreme soul has explained the inherent qualities of the same and his own qualities which till date no human beinghas not been able to understand.

Initially we will start detailing different systems of CNS as we had earlier detailed in our article where we compared obesity as parallel to a state of brain as seen in drug addiction.

The initial question arises

Who am I

Basically, we are not the body given the name, profession, religion but the soul/aatma/Chetna or consciousness that can't be visualized whereas the brain body are physical and can be seen.

2. Anatomy of various systems

For understanding it is significance to give a description of the insight of the Attention systems, Role of Cognitive Control Systems Brain reward systems, Role of emotion and memory as we contrasted in a model of obesity in lines with drug abuse and addiction as proposed by Volkow ND.

2.1 Role of cognitive control systems

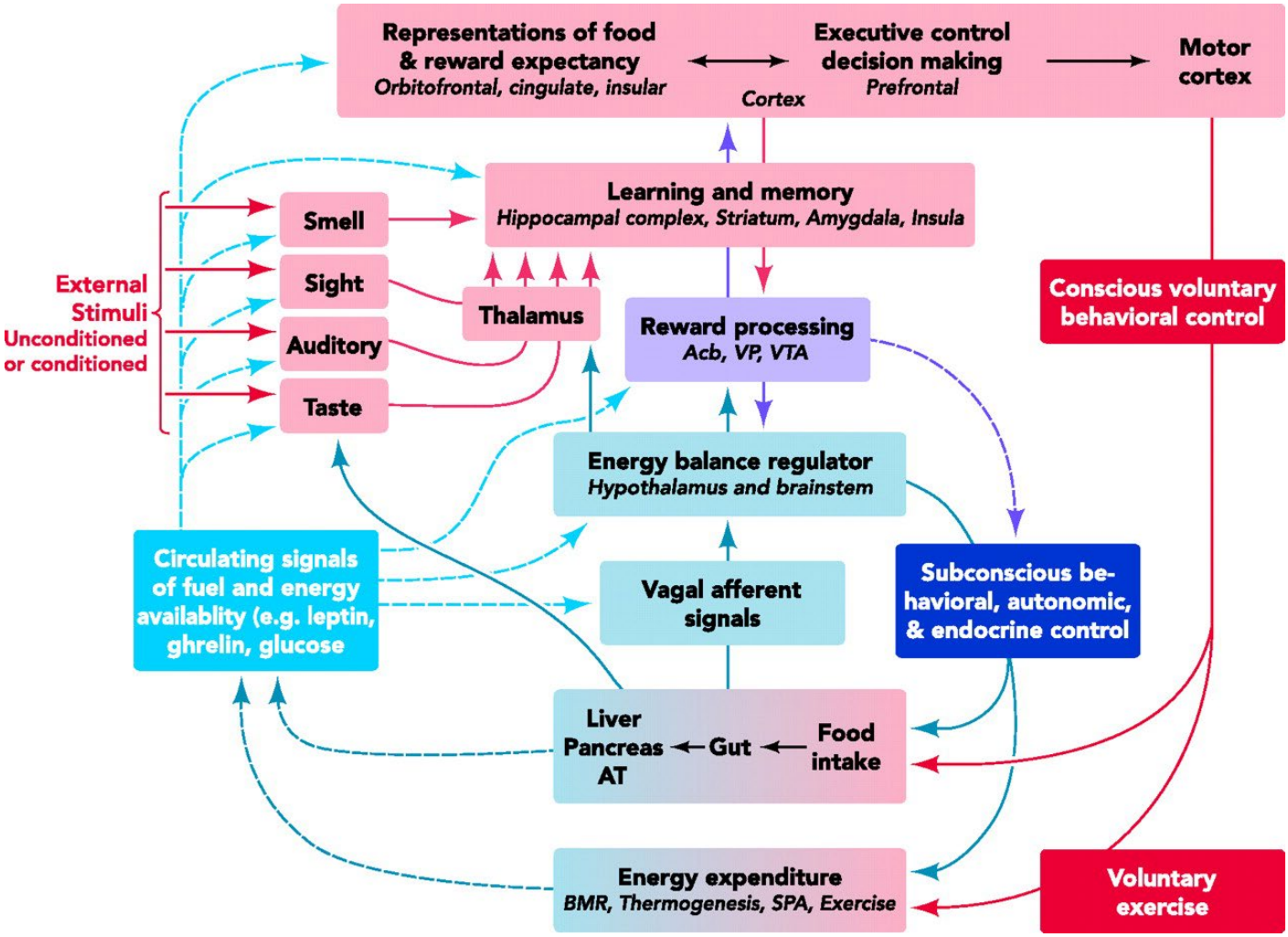
Cognitive Control have executive functions inclusive of hampering of the prepotent reactions. Cognitive regulation aids a person to refuse a piece of cake if not feeling hungry. Prefrontal Cortex (PFC) makes most part of Cognitive Control networks particularly the cingulate cortex, inferior frontal cortex, pre-supplementary motor area along with dorsolateral PFC [DLPFC] [1]. Dysfunctional hampering regulation has been illustrated in obese humans. It has been posited that dysfunctional cognitive regulation control might influence the increased reward reactions to food cues; thereby overeating [2,3]. Lesser metabolism has been observed in obese as determined by Positron Emission Tomography [PET] imaging or diminished activity in PFC that associates with dopamine receptor accessibility as well as and BMI [4,5]. Thus, abnormalities in cognitive regulation have been observed both in general as well as food particular tasks in obese people though it is not clear if poor cognitive or hampering regulation occurs secondary to obesity or results in obesity (Figure 1).

Legend for fig 1

Courtesy ref no 8-A theory of how cognitive control may interact with reward and food consumption is that in typical cases, heightened cognitive control may decrease the reward system's activation to food cues and thus decrease food consumption (a). This may be altered in obesity, where cognitive control is impaired, and the reward system may be heightened, leading to increased food consumption (b).

2.2Attention systems

Brain network of attention systems are inclusive of parietal as well as and visual cortices, along with certain regions of frontal cortex [6,7]. Escalated activation of occipital cortex has been illustrated for high calorie/HFD images (Figure 2)



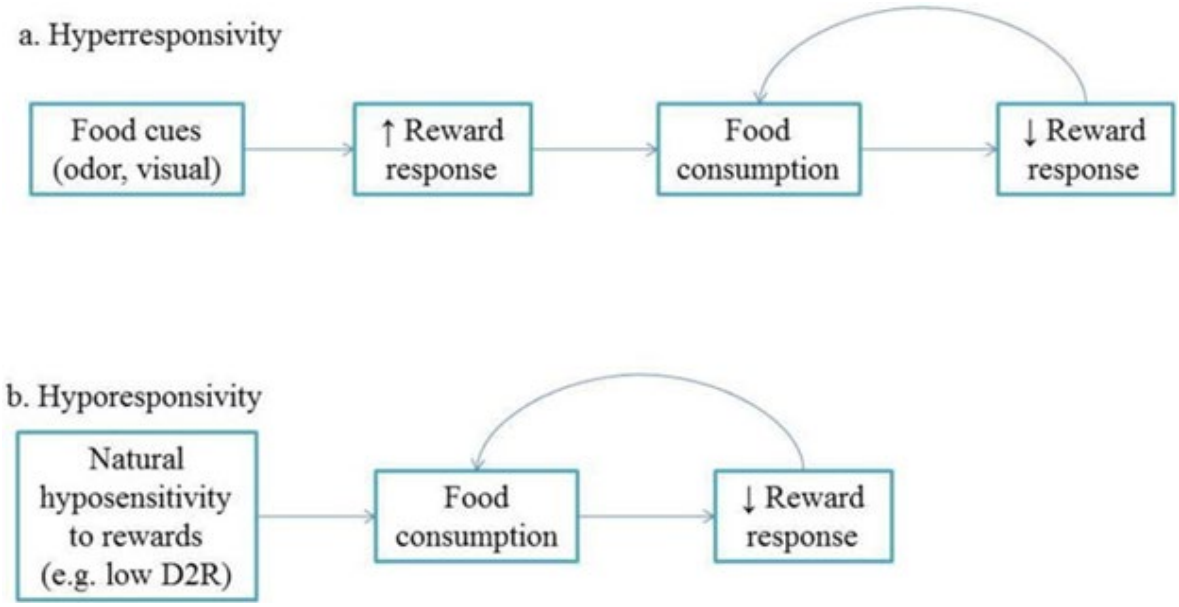
Legend for Fig2

Courtesy with permission from Berthoud HR ref no- 52. Major Systems and pathways responsible for the neural integration of internal and external information in the control of appetite and energy expenditure. Blue areas and pathways are mainly involved in metabolic and energy balance regulation. Red areas and pathways are mainly involved in communication with the external world through cognitive and emotional processes such as learnng and memory, reward, mood, stress, choice and decision making.

2.3 Role of reward systems

Major hypothesis with regards to how these get altered in obesity is: i) Hypersensitivity to reward-as summarized by PET studies have illustrated lower accessibility of dopamine D2receptors in striatum in obese in contrast to normal weight rats as well as akin observations in humans also. Thus, implication is lower dopaminergic signal might => people to seek highly rewarding foods that in turn => obesity. An under responsive reward circuit as well as and habitual food consumption of high fat/calorie foods has been contrasted to drugs of addiction as reviewed earlier [8,9-14].

Other theory: ii) Hyperresponsivity to food cues => individuals to seek greater along with greater amounts. Escalated ingestion of these highly rewarding foods => larger disconnect amongst reward exposure to food cues along with reactions to ingestion of foods which => them to eat greater food quantities to achieve expected reward. This has been by activation of nucleus accumbent, midbrain and Orbitofrontal Cortex [OFC] in visual foods cues and to achieve the anticipated reward expectation of milk shake [15]. (Figure 3).



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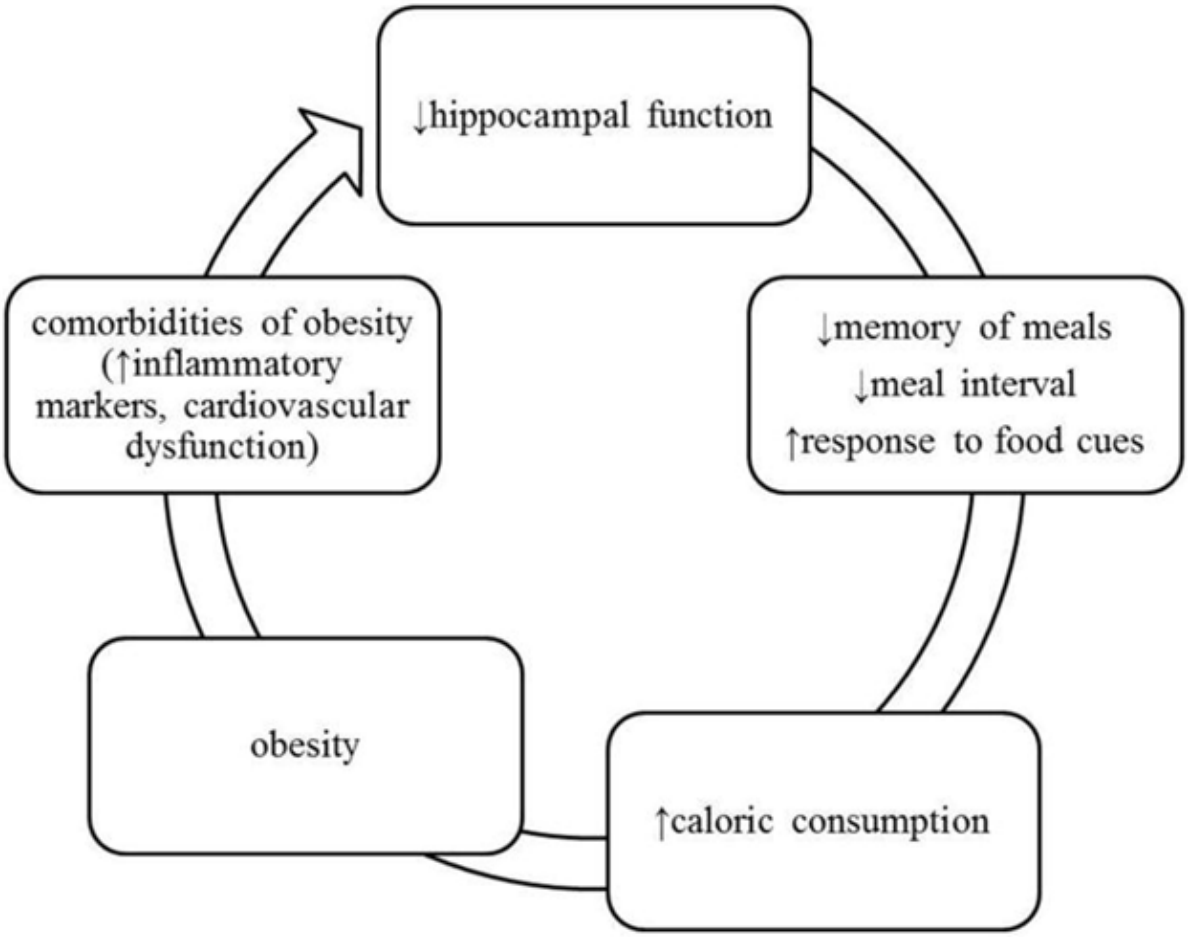
Courtesy ref no 8-Theories of how reward responsivity is affected in obesity: hyperresponsivity [a] and hyperresponsivity [b]. The first theory suggests that obese individuals have a heightened reward response to food cues but after increased food consumption, this leads to a decreased response to reward to actual food consumption [but not food cues], and this disconnect leads to greater food intake over time. The second theory posits that individuals with a natural hyposensitivity for rewards consume more food because they require more food consumption and higher calorie or high fat foods to achieve the same level of reward.

2.4 Role of emotion and memory

The amygdala is the primary one that controls appetite in reaction to emotions. Amygdala activates to food cues [16,17] and this reaction escalates in childhood, adolescent as well as adult obesity [18-21]. Activation of amygdala further anticipates ingestion of high fat or high calorie foods [22]. Participants with reaction of amygdala to food cues when not hungry had chances of gaining weight [23]. Greater quantities of leptin in adolescents associated with activation of amygdale to high calorie foods [21]. Stress relieving actions of sucrose gets modulated through amygdale circuit communicating with hypothalamus-pituitary-Adrenal [H-P-A] axis [24].

Memory is basically controlled by hippocampus and Para hippocampal formation that might impact eating. Diminished functioning of hippocampus => escalated food consumption as well as poor diet quality [25,26]. Despite canonically timing of food gets regulated by circadian rhythms along with \suprachiasmatic nucleus, proof confirmed this gets over ridden by memory as well as experiences (Figure 4) [26]. Hippocampus gets inputs with regards to food cues from numerous other areas inclusive of insula, OFC, as well as arcuate nucleus of hypothalamus. Moreover, hippocampus gets regulated by peripheral signals like leptin in addition to ghrelin to control food ingestion. Obesity, possibly could comprise hippocampus functions via Blood Brain Barrier [BBB], although hippocampus is protected by BBB, cytokines associated with inflammation might not possess the capacity of reaching hippocampus, yet there is support that inflammation in CNS might be carried out by microglia like in hypothalamus [27,28]. In obesity, microglial effects have been illustrated to results in dysfunction of hippocampus are via CNS inflammatory events. These action on hippocampus have been observed more in rodents compared to human brain with more studies required in humans.



Legend for fig4

Courtesy ref no-8-Memory influences eating behaviours in a cyclical manner. Decreased hippocampal activity leads to decreased memory of meals and increased response to food cues. This leads to increased caloric consumption and obesity, which in turn leads to increased inflammation and cardiometabolic dysfunction which in turn decreases hippocampal function.

Basically, prefrontal cortex [PFC] is the so called logical or Intellectual Brain meant for accurate decision taking- Emotional Brain mainly Limbic system as explained earlier inclusive of amygdala are responsible for emotions-normally love suppresses what is correct/right, dopamine -pleasure or hedonic, vasopressin-physical love-attraction, bonding, serotonin for happiness-- here in laymen term will concentrate on PFC &amygdala. Amygdala is an almond shaped area of Limbic system. Amygdala does not allow the PFC to think positively however this can be attained with raj yoga meditation-Basic Principles- neurons which work/fire together get wired ultimately, electrical flow from 1 – other neuron &so on ultimately autopilot mode -we automatically perform thus habit generation Belief System one can leave habits or alter habits by giving positive thoughts like drug habit repeated say cigarette/tea/high fat diet-her thoughts/ affirmations work but unstable mind-In 1948Donald Hap gave the concept of neuroplasticity. Brain neurons can't be altered once destroyed-only can alter circuit -say short circuit occurs in electric wires -works on akin principles. Plasticity moulds neurons- neuronal connections >100 million neurons habits in some circuits-replace the thought of tea/liquor say harmful as well as replace new thoughts so new connections formed.

Biochemical events in synapses as well as other neuronal compartments underlie neuroplasticity [functional as well as structural changes in the brain which aids in adapting to the environment, learning, memory, in addition to rehabilitation subsequent to brain damage]. This basic molecular level of brain plasticity covers multiple particular proteins [enzymes, receptors, structural proteins, etc.] which take part in numerous coordinated and crosstalk with signal along with metabolic events, their modulation generating a molecular basis for brain plasticity [rev in 29].

Brain-mind work like a library with Brain being a storehouse -whatever hear, see read generate experience- mind is a reader, picks up thoughts from the stored information as well as produces thoughts.

Belief System -whatever negative content is acquired from surroundings- thoughts mirror neurons principles- thoughts constitute inner world. This inner world needs daily cleaning.

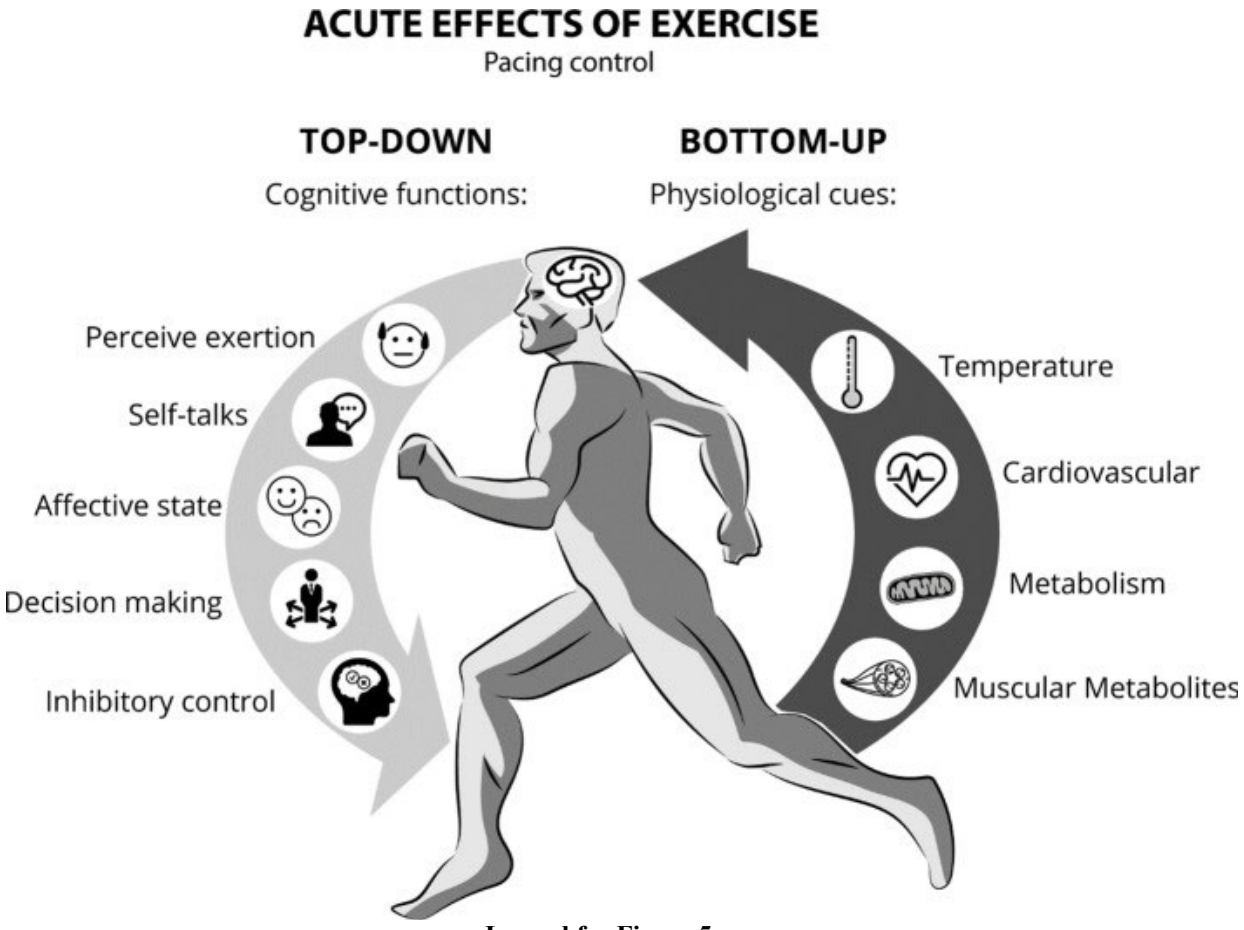
The negativity can be eliminated by cognitive affirmations -this approach repeated by constant cognitive affirmations till wiring not changed How imagination works was validated by Napoleon making an imaginary 9-member superior brains constitution and he would discuss and gradually over 6 months it gave them a positive outcome.

By utilization of Gamma neuron camera -same area in Brain gets activated when one monkey eats banana while other monkey watches eating him i.e. imagination-same events generated in neurons -hence same principle works in Rajyoga meditation Einstein, Vivekananda -smart man, followed by Nikola Tesla-who invented magnetic resonance imaging - revealed secrets of universe - insight from energy, frequency, vibrations -wire -current passes -in body current is energy-once this energy of the consciousness is present we are alive -on death this energy- disappears Universe comes from Latin word -unit means 1, verse means turn into 1,

3. Brain reward systems in addiction

This neural network is comprised of various intercommunicated Brain regions inclusive of ventral tegmental area, nucleus accumbent, amygdala, hippocampus prefrontal cortex [PFC] [30]. The PFC portrays an amalgamated neural system in humans. There requirement is for normal executive working inclusive of decision taking as well as inhibitory regulation in addition to advantageous socioeconomic working [31]. Studies utilizing positron emission tomography [PET] along with functional magnetic resonance imaging[Firm] have illustrated that subjects presenting with substance use disorders[SUD] possess reduction in activity in the PFC[32].This situations apparently is associated with reduction in the quantities of dopamine receptor in addition to aberrant firing of dopaminergic neurons[33].Such alterations in the dopamine system in addition to PFC activity facilitate compulsive substance consumption as well as behaviour regarding looking for substances along with elimination of regulation over substance ingestion [34]. Akin to that incomplete PFC generation as well as the resultant reduction in the capacity of regulating impetuous choice have been pointed to reason out the specific susceptibility of adolescents towards substance abuse [34], emphasizing the significance of avoidance of utilization of addictive psychoactive substances at this key time of Brain formation. Thereby the present convalescence approaches have highlighted the significance of Interdisciplinary treatment strategies involving targeting the regeneration of normal PFC working whereas combination of medicines, social care in addition to behavioural therapy with cooperation of Psychologists, Psychiatrists, social workers as well as family [35].

Although successful management of SUD is the field of psychiatrists with them displaying the role of aerobic exercises for SUD having an integration of mind-body crosstalk with bidirectional modes [top down [cognitive] as well as bottom up processing factors [physiological reactions] [36]] (see fig5). Here our emphasis is to highlight how spirituality might aid in getting over it.



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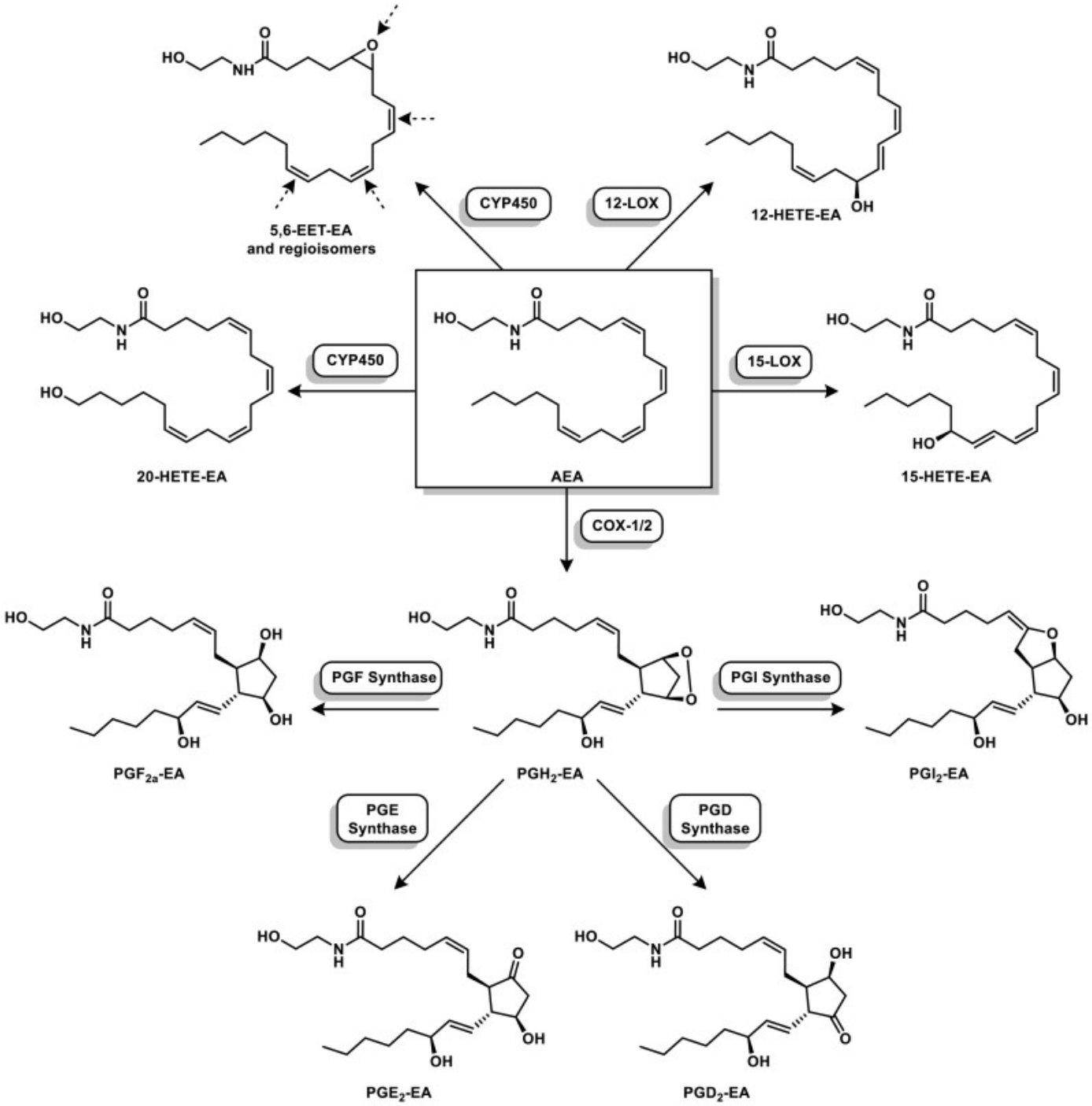
Courtesy ref no-36-Pace control during continuous exercise while integrating top-down [cognitive functions] and bottom-up processing factors [physiological responses].

Here we deal with the science of raj yoga meditation, which deals with generation of a relationship with the supreme soul. Basically, we all are souls which possesses the 7 properties of love, purity, Gyan or knowledge, Ananda or bliss, sukh orhappiness, peace or shanti power of which the supreme soul is an ocean. Energy flows from a higher energy frequency state to one of lower energy state. We human souls have got so much depleted that over time cycle of 5000 yrs when souls were intrinsically full of all these 7 properties that humans as well as animals lived together in harmony and peace. Presently in kalyug most of these properties are depleted. For generation of any relationship one has to understand that he is like a point of light as well as we can get all the love, purity, knowledge, peace, power or bliss from him. He is the one who introduces himself etc. Coming to the science of spirituality bliss gets generated by the liberation of hormone anandamide. In USA lot of trials are going on with regards to artificial production of anandamide for treating SUI but nothing better than natural anandamide or endocannabinoid for getting rid of it.

3.2 Anandamide biochemistry &physiology

N- arachidonoyl ethanolamine [AEA] or anandamide has been studied maximum exhaustively of the full family members of N-acyl ethanolamine [NAE]. In maximum tissues AEA quantities are 10 to 100-fold lesser than- palmitoyl ethanolamine [PEA], N-stearoyl ethanolamine [SEA] and N-oleoyl ethanolamine [OEA], [37,38]. Nevertheless, unlike other NAEs, AEA can activate the cannabinoid [CB1]-receptor [39]. The CB1 receptor has one of the maximum enrichments of G- protein coupled receptors [GPCRs] in the mammalian brain, which gets activated by (-)-Δ [9]-tetrahydrocannabinol [THC], the psychoactive constituent of cannabis. Subsequently, AEA and 2-arachidonoylglycerol [2- AG], a second endogenous CB1 receptor agonist, are known as endogenous cannabinoids or endocannabinoids are generated in addition to released. Both AEA along with 2-AG are partial agonists for the CB1 receptor with sub micromolar potency [40]. Absolute quantities of AEA are usually lesser in contrast to 2-AG in brain, although greater variation have been found on the method of assessment [41]. In bulk extracts of whole or area- particular rat brain lysates, AEA quantities were canonically 100- to 1000-fold lesser in contrast to 2-AG, however this variation was just2- to 8-fold on determination via in vivo micro dialysis [41]. It was posited that this results in by the greater quick post-mortem escalation of 2-AG in contrast to AEA [41]. AEA is believed to be regarded as a tonic neuromodulator – i.e., it persistently signals in the basal state – which is liberated by neurons upon Ca2+-stimulation and is rapidly degraded by Fatty acid amide hydrolase (FAAH) [42,43-45]. Despite AEA was initially detailed in the form of retrograde neurotransmitter, N-acyl phosphatidylethanolamine - phospholipase D [NAPE -PLD] has presynaptic placement whereas FAAH has postsynaptic placement, implying that AEA might be working in the form of an anterograde signalling lipid [46]. AEA further possesses the capacity of working in the form of an intracellular messenger, generated upon an influx of Ca2+-ions through activation of the GQ-pathway [47]. The word 'Ananda' –alias bliss in Sanskrit – was correctly coined, in view of escalated AEA signalling generate analgesic, anxiolytic along with anti-depressant action via CB1 receptor signalling in the brain [48-50]. On the other hand, acute as well as recurrent stress exposure in rats resulted in a diminished AEA quantities in the amygdala, modulating by enhanced FAAH activity [51]. Stressed rats illustrated an inverse association amongst amygdale AEA along with plasma stress hormone quantities [corticosterone] [52]. Decreased brain AEA signalling upon recurrent stress escalated liberation of corticosterone [53]. Compared to that repeated stress increased amygdale 2-AG quantities, which ameliorated hypothalamic-pituitary-adrenal [HPA] axis activation. AEA and 2-AG are thereby proposed to be the effectors of HPA-axis signalling in the brain, whereas possessing functionally unique part [53, 54]. Apart from its roles in modulating fear and stress behaviour, AEA was revealed to facilitate neuroprotection, memory generation along with food consumption via brain CB1 receptor activation [53-58]. Pharmacological studies in mice pointed that exogenous AEA cannabimimetic generates reactions, which are fast in onset, but shorter as well as possess lesser robust in contrast to THC, presumably in view of its rapid catabolism [59]. In agreement, Fatty acid amide hydrolase [FAAH]- knockout [KO] mice were extra sensitive to AEA treatment [60]. Exogenous AEA delivery in rats produced a central CB1-receptor- based orexigenic [appetite-stimulating] action akin to THC [61]. AEA has further been correlated to CB1 receptor signalling in the periphery, for example in adipocytes, the female reproductive system as well as skin tissue where it is implicated in energy expenditure, implantation along with epidermal differentiation, respectively [62,63]. Intriguingly, peripheral CB1 receptor activation is involved in food ingestion in addition to intestinal AEA quantities were observed to be substantially highly escalated in starved mice [64]. The analgesic effects of AEA were also observed in the periphery, where peripheral blockade of FAAH produced antinociception via a CB1 receptor-dependent mechanism [65]. Noticeably the antinociceptive action of AEA escalated synergistically when combined with PEA in a mouse model of peripheral pain [66]. AEA has been detailed in the form of partial agonist with sub micromolar robustness for the CB2 receptor, which is primarily expressed in the immune system as well as is implicated in the inflammatory reaction [40, 67-69]. Despite 2-AG has been pointed to be the true endogenous CB2 receptor ligand in view of its usually greater quantities compared to AEA and its capacity of working in the form of a full agonist [40,70], further AEA was revealed to modulate inflammation via activation of the CB2 receptor by diminishing pro-inflammatory cytokines in cells [62, 71]. Apart from the cannabinoid receptors, AEA possesses the capacity of activating the transient receptor potential vanilloid 1 [TRPV1] ion channel [72]. AEA has thus been named an end vanilloid [73,74,]. TRPV1, also termed - the capsaicin receptor, is a significant player in pain perception in addition to has placement at peripheral sensory neurons [75]. Accruing proof has shown that TRPV1 is expressed in the CNS also [76,77]. The activation of TPRV1 by AEA causes a cellular influx of Ca2+-ions as well as has been correlated with locomotor depression, hyperalgesia

under inflammatory conditions, vasodilation along with hypothermia [47, 75]. [see Fig6] [78].



Legend for Figure. 6

Courtesy ref no 78. Oxidative metabolic pathways for AEA. See text for details. Abbreviations: AEA, N-arachidonoylethanolamide [anandamide; COX, cyclooxygenase; CYP450, cytochrome P450; EET-EA, epoxyeicosatrienoyl ethanol amide; HETE-EA, hydroxyeicosatetraenoyl ethanol amide; LOX, lipoxygenase; PG-EA, prostaglandin ethanol amide.

4. Rajyoga meditation is the natural source of anandamide.

Spirituality comes from study of Spirit or soul. Normally energy flows from higher source of energy to lower state like in a battery.

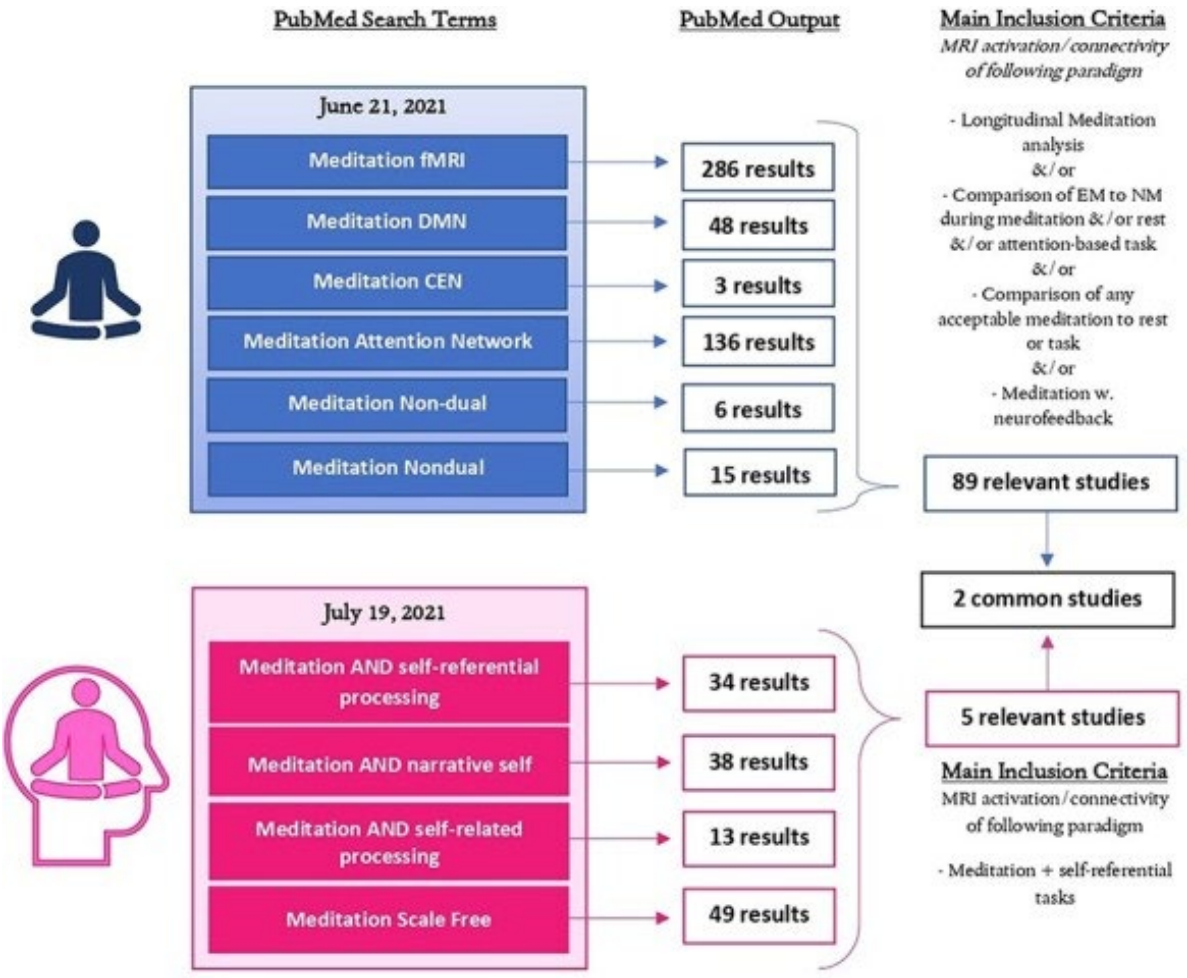
Emotions which are negative-are comprised of lower frequency vibrations Positive emotions like happiness- are comprised of higher frequency vibrations like from battery in raj yoga meditation energy flows from the ocean of love, peace, Gyan, power, sukh/happiness, happiness, bliss which is extremely charged -so we can charge our battery from this higher frequency to lower

frequency like happiness validated by ref 79]. gamma neuron camera in happiness shows aura as orange or red while darker in unhappy.

Thoughts- neurons fire -actions decided more intense if our iChat/desire v strong-firing of neurons
There are 4 kind of thoughts-i) positive [like kalyankari or helping , shubh or well meaning bhavna or feelings for others ii) negative [why x, y, z did so and so feel of revenge iii) neutral [day to day routine take bath, work etc iv) waste thoughts [no connection with present-keep thinking of past. greater such thoughts more energy depleted. Stress is given on type of thought process to be kept positive with emphasis of the supreme soul on not to accept defeat with any failures which just are tools to teach us and he cites examples of the lady who lost her leg in a train accident but with her will power she managed to climb the Himalayas .The same for any deformities he says if you don't accept defeat he walks 1000steps with us once we move one step.

In a research done in Howard-they wrote an article with title – ''A wandering mind is an unhappy mind''-on recruiting people asking how they felt at the moment their observationswere-47% times people were not focused on what they were doing-most inventions take place in lot of silence &concentration like space programmes etc.
With positive thoughts usually, one has great feelings and individual remains in good mood
Say in sangeet/music-pitch changes-voice changes but if all pitch only 1 voice comes more bland -i.e. in universe if all higher frequency vibrations souls looks same like in sangeet/music in 2007 Dr David gave the concept of rumination- negative emotions- negative influence
It has been suggested that a high tendency to ponder manifests in the form of a deficit in controlling emotion. Previous research observed that people possessing greater trend to lament over old things illustrated attention for negative stimuli in addition to escalated negative thinking, that might lead to augmentation of experiences of negative emotions. Furthermore, greater quantities of lamenting over past was correlated with lesser emotional insight., On the basis of this Lakita. [80] posited (i) high ruminators [HR] experience emotional responses of greater intensity in contrast to low ruminators [LR] for negative however not positive emotions, (ii) LR possessing greater emotional clarity in contrast to HR, as well as (iii) akin fashion of outcomes results for sulking but not for reflective reviewing. A demographic questionnaire was finished by the enrolled patients, a rumination response style questionnaire, in addition to the Beck Depression Inventory-II. They further rated emotional intensity along with isolated emotion kind for scene pictures from the CAP-D [Categorized Affective Pictures Database]. The highest [HR] and lowest [LR] quarters of ruminators were contrasted on degree of emotional intensity as well as emotional clarity. Their observations were that HR felt negative emotions with greater intensity in contrast to LR, with no variation for positive emotions. In comparison to their proposal, the two groups did not vary in their emotion understanding. This fashion of outcomes was observed for brooding but not for reflective contemplating. Their research gave insight regarding the mode behind rumination as well as emotion [80].

Love is basically a high frequency emotion, A-pleasure-say love is blind -epic suppressed-like we excuse subtle mistakes of ones we love hence judgemental brain shutdown, have attachment with them Inner peace -normally turbulence in inner world subsequent to1st reaction to stress that works on sympathetic nervous system [SNS] leads to ACTH liberation which further leads to escalated production of cortisol, adrenaline as well as noradrenaline, increased heart rate, blood pressure [BP]. Basic idea as we had been taught is fight as well as flight reaction, greater cortisol escalates sugar availability to fight any stress or enemy say lion more energy needed by limbs and shuts off digestion, Feedback from amygdala to temporal lobe to PFC which gets shut off on continued negativity while size of amygdala gets enlarged with obvious Hippocampus is affected memory loss, dementia occurs as well as gastrointestinal tract [GIT] influenced. Expert clinicians Default Mode Network [DMN]-active when not doing anything-has effect on smriti, emotions' have very low DMN activity-- Meditation might possess a considerable influence on our mental life, with expert clinicians illustrating an experience that is rid of any partition amongst a different self in addition to the environment, pointing an obvious experience of " nondual awareness." What are the neural associates of these kind of experiences as well as the way they correlate with the thought of nondual awareness itself? For getting insight regarding the actions of meditation over the brain's particular topography, Cooper teal. [81], reviewed functional magnetic resonance imaging[fMRI] brain observations from studies particular to arrangement of meditation kinds along with the degree of experience of meditators. Furthermore, they reviewed observations from studies that directly looked into the crosstalk amongst meditation in addition to the self-experience. The maximum outcomes obtained were(i) reduction posterior Default Mode Network [DMN] activity, (ii) escalated central executive networks [CEN] activity, (iii) diminished connectivity amongst posterior DMN along with posterior in addition to anterior DMN, (iv) escalated connectivity amongst anterior DMN as well as CEN along with (v) influenced connectivity amongst DMN as well as CEN [significant amongst impacted connectivity between the DMN and CEN [probably a nonlinear event]. Overall these point a considerable distributional transfer of the brain of the brain's particular topography in meditators in advanced stage -thus they posited a topography redistribution model of meditation [Tromp]. Of these a core constituent of the Troms topographic redistribution of DMN as well as Cena's associated with a reduction in the mental self-processing is associated with in addition to a synchronization with the greater nondual layers of self-processing, noticeably interoceptive as well as exteroceptive self-processing, this redistribution of the working of both brain in addition to self-processing can lead to the definitive experience of " nondual awareness. Hence the insight gained in this review yields substantial neural actions of advanced meditation, positing an outcome based unifying medal [Tromp] with the ameliorating the objective[neural] along with subjective[experimental] actions of meditation [81].



Legend for Figure. 7

Courtesy ref no 81-Flow chart of screening process for reviewed studies. Upper blue half represents the screening that was specific to meditation only, and the bottom pink half represents the screening that was specific to the interaction between meditation and the self

4.1- Brief introduction of ancient yoga history in India

200 yrs prior to Christ Oldershaw was 1st time published in India by Patanjali rishi. Despite the large number of historical in addition to the surviving extant schools of canonical Yogic practice, [inclusive of Tantric Yoga] share a common underlying framework of practice known as Ashtanga Yoga [or the eight limbs/organs of Yoga], first believed to have appeared in the Yoga Sutras of Patanjali.

The eight limbs of Yoga in order are, yama, niyama, asana, pranayama, pratyahara, dharana, Dhyana, and samadhi. Yama and niyama consist of five ethical principles each to guide a yoga practitioner's conduct in relation to others and themselves, respectively. In most traditions of Yoga, the teacher [or guru] kept an intricate watch on the conduct of the disciple before initiating them into the subsequent limbs. In the Ananda Margi practices as well, progress in meditation is believed to be inextricably correlated with the degree one is able to follow yama and niyama.
Yama-Its 5 limbs are i) ahinsa [nonviolence] ii) Satya /truthfulness iii) asteya(notheft) [no theft] iv) aparigraha [not possessive] as well as v) brahmcharya [celibacy all 5 some feelis akin to Jain /partly Buddhist
Niyama- Its 5 limbs are i) swach [cleanliness] ii) santushta or satisfaction iii) Tapas [self-discipline] sadhana[self-reflection] v) ishvarapranidhana [surrender to a higher power /god] [reviewed in ref 82in detail]. I am not going in detail of asana, pranayama, pratyahara, dharana, Dhyana, and Samadhi as our major aim is to describe Rajyoga Meditation. omit over the

4.2 What is raj yoga meditation (RM) and its benefits

The term Raja yoga has been detailed in numerous transcripts in addition to texts inclusive of Bhagavat Gita along with books which have been rewritten by Yoga Sutra of Patanjali, Raja Yoga by Swami Vivekananda etc. Here Rajyoga meditation [RM] points to Brahmakumaris school of thought which details the foundation principles for this type of meditation. RM is separate from other types of meditation in view of that it portrays an open-eye meditation strategy. Most of the mediation strategies are closed-eye meditation practices concentrating on their thoughts or count breathing, or chanting mantra, or remaining silent, etc. In RM Spiritual practice, a person is to make himself/herself aware in the form of being self/soul [an eternal kind of point of light] that is localized amongst the eyebrows while focusing on a meaningful external symbol [a point of light that is believed to be a symbolic portrayal of Supreme Soul/God] [83]. Furthermore, they are counselled to think positively about

the inherent qualities for example peace, love, bliss, etc. which are latent within the self- [84,85]. There is paucity of studies published on RM, specifically in the field of neurocognitive research.

More advantages of RM is that telomeres present in chromosomes become smaller with negativity which is associated with shorter life whereas long term meditators [LTM]- have longer telomeres size; thus, longer life [86] ...

4.2 Effect on Electroencephalography (EEG)

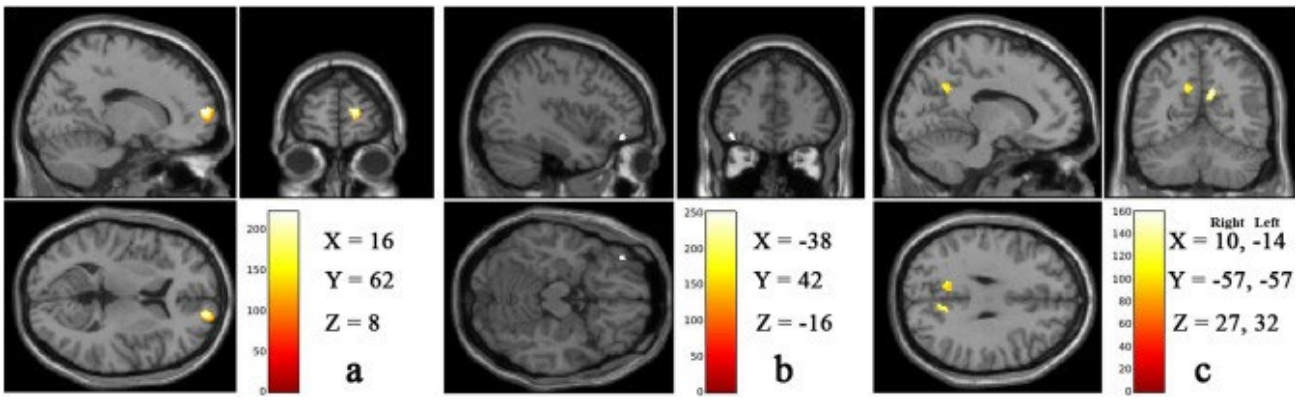
With the utilization of electroencephalography [EEG]machine Brain electrical activity can be determined with use of cap with electrodes fitted- Brain waves obtained might be i) β waves-when person normal talking /behaviour but might get exaggerated on inner world turbulence detailed in numerous transcripts On meditation mind relaxes & is in α state ii)Deeper meditation-Θ state-a state in which we sleep with dreams -this is a state when subconscious mind active as well as suggestions can be given iii)More deep -δ state-deeper sleep with nodreams-Dadi Janaki's mind labelled as the most stable mind in the world where she is in δ state while doing day-day work as well.

Newer theories new Brain cells can be created in long term meditators [LTM]-they possess thicker grey matter, modulate smriti emotions. Have increased Brain connectivity-hence anger, emotions -power there to control them with greater tolerance power.

This was corroborated by Paucity of studies were performed regarding physiological properties of raj yoga meditation with utilization of electroencephalography [EEG]. Band power in addition to cortical asymmetry had not been evaluated with raj yoga meditators. Hence. Sharma etal. [85] performed a study with the objective of assessment of action of continuous meditation practice on EEG Brain dynamic in low frequency bands of long-term raj yoga meditators. Matching of subjects was achieved in both the groups. Lesser frequency EEG bands were evaluated in resting in addition to at the time of meditation.21 male long-term meditators [LTM] as well as equal controls were recruited to take part in this study according to inclusion criteria. Semi high density was recorded prior to along with at the time of meditation in LTM group as well as control group. The major outcomes obtained from this study was spectral power of alpha in addition to theta bands along with cortical[hemispherical] asymmetry estimated with the utilization of band power. One-way ANOVA was determined for finding significant variation amongst EEG spectral properties of groups. Outcomes displayed high band power in alpha in addition to theta spectra in case of meditators. Determination of cortical asymmetry via EEG power was further observedto be great in frontal along with parietal channels. Nevertheless, no association was observed amongst the experience of meditation [in years/hrs] practice with EEG indices. Thereby they concluded that in all outcomes obtained pointed to smaller frequencies [alpha in addition to theta] aided in at the time of sustenance of meditation experience. This implied a positive influence of meditation on frontal along with parietal brain regions implicated in the events of control of selective in addition to sustenance of attention that validated the implication of emotion along with cognitive processing.

4.3 Effect on grey matter volume (GMV)

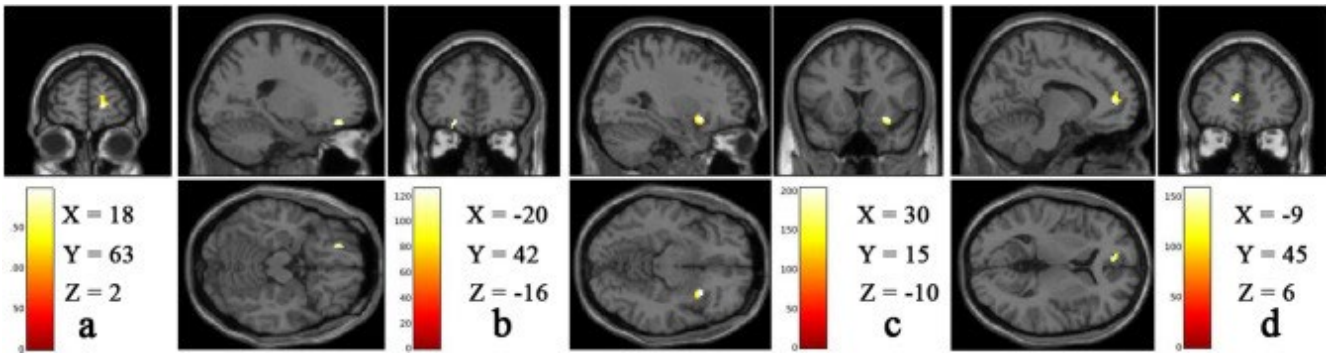
Neural plasticity [NP] gets escalated according to studies which validate that on practicing raj yoga meditation NGO elevated in reward processing areas of brain. Till date no studies, had been conducted for validating these alterations in Rajyoga meditation [RM]practitioners. Ramesh Babu et al. [87], in the present study possessed the objective of isolating grey matter volume [GMV] alterations in reward processing regions of brain as well as its correlation with happiness scores in RM practitioners in contrast to non-meditators. Structural MRI of selected enrolment of people was performed matched for age, gender in addition to handedness [n = 40/group] were assessed with the utilization of voxel- dependent morphometric approach and Oxford Happiness Questionnaire (OHQ) scores were associated. Significant escalation of OHQ happiness scores were found in RM practitioners in contrast to non-meditators. While, a tendency towards significance was the finding in RM practitioners who possessed greater experience, on contrasting OHQ scores with hours of meditation experience. Furthermore, in RM practitioners, greater GMV were found in reward processing centres; for instance, right superior frontal gyrus, left inferior orbitofrontal cortex (OFC) in addition to bilateral praecuneus. Multiple regression evaluation illustrated significant correlation amongst OHQ scores of RM practitioners along with reward processing regions right superior frontal gyrus, left middle OFC, right insula as well as left anterior cingulate cortex [87].



Legend for Figure8

Courtesy ref no 87

Representative structural MRI images, showing significant difference ($p<0.05$ FWE TFCE corrected) in (a) right superior frontal gyrus, (b) inferior orbitofrontal gyrus and (c) bilateral praecuneus in RM practitioners than NM. Colom bar represents TFCE-value. These representative structural MRI images were compiled and MNI coordinates were inserted into this compiled image using Adobe Photoshop version CS3 (https://www.adobe.com/in/).



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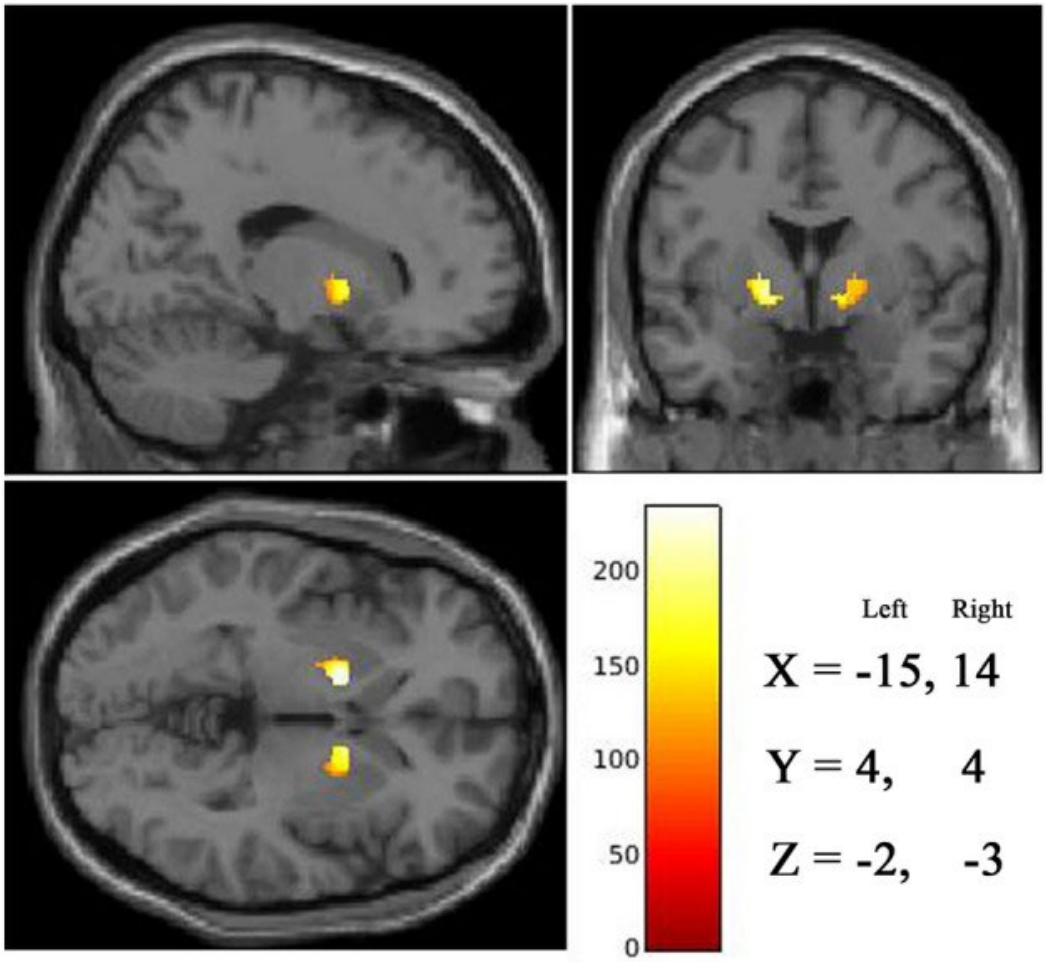
Courtesy ref no 87

Representative structural MRI image, showing significant ($p<0.05$, FWE TFCE corrected) positive correlation with GMV of (a) right superior frontal gyrus, (b) left middle orbitofrontal gyrus, (c) right insula and (d) left anterior cingulate cortex in RM practitioners. Colour bar represents TFCE value. These representative structural MRI images were compiled and MNI coordinates were inserted into this compiled image using Adobe Photoshop version CS3 (https://www.adobe.com/in/).

Additionally, with escalating hours of RM practice, a significant positive correlation was found in bilateral ventral pallidum. These observations suggest that RM practice escalates GMV in reward processing areas correlated with happiness [86].



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Courtesy ref no 87

Representative structural MRI image, showing significant ($p < 0.05$ FWE TFCE corrected) positive association of GMV of bilateral ventral pallidum in RM practitioners with hours of meditation practice. Colour bar represents TFCE value. These representative structural MRI images were compiled and MNI coordinates were inserted into this compiled image using Adobe Photoshop version CS3 (https://www.adobe.com/in/).

4.4 Effect on coronary artery disease

Dr Satish Gupta etal [88] for the first time under IRDO study with the guidance of supreme soul in one hundred and twenty-three angiographically revealed moderate to severe coronary artery disease [CAD] patients having the delivery of healthy as well as happy lifestyle [HLS] that was constituted of lesser-fat as well as greater-fibre vegetarian diet, moderate aerobic exercises in addition to stress management via raj yoga meditation. The maximum subtle characteristics were to train in self-responsibility [meaning heal +thy] along with self-empowering via inner self-consciousness [swaths imply inner selfish- consciousness] strategy with the utilization of raj yoga meditation. Subsequent to their 7day in-house short stay patients were advised to follow up regarding revaluation in addition to further training in its advancements. On the finishing of 2 yrs a repeat angiography was advised. 360Coronary damaged areas were evaluated by 2 separate angiographers. In case of CAD patients having maximum sticking to the orders of HLS ,had a regression of percentage diameter stenosis by 18.23±12.04absolute percentage points.91% illustrated a tendency towards regression along with 51.4% damaged areas regressed by > 10 absolute percentage points .The cardiac events in CAD patients were :11 had maximum adherence along with 38 had the minimal adherence on a follow up of6.48years(risk ratio; maximum vs minimal adherenc e4.32;95%CI:1.69=11.705;P<0.002].In Toto healthy alterations in cardiovascular, metabolic along with Psychological paradigms reduce in absolute percentage diameter coronary stenosis as well as cardiac processes in patients of CAD were intricately correlated with sticking to HLS disease. Nevertheless, greater than 50% of adherence is imperative to attain a significant alteration [88].

4.4 Effect of RM on modulation of anxiety levels prior to coronary artery bypass surgery

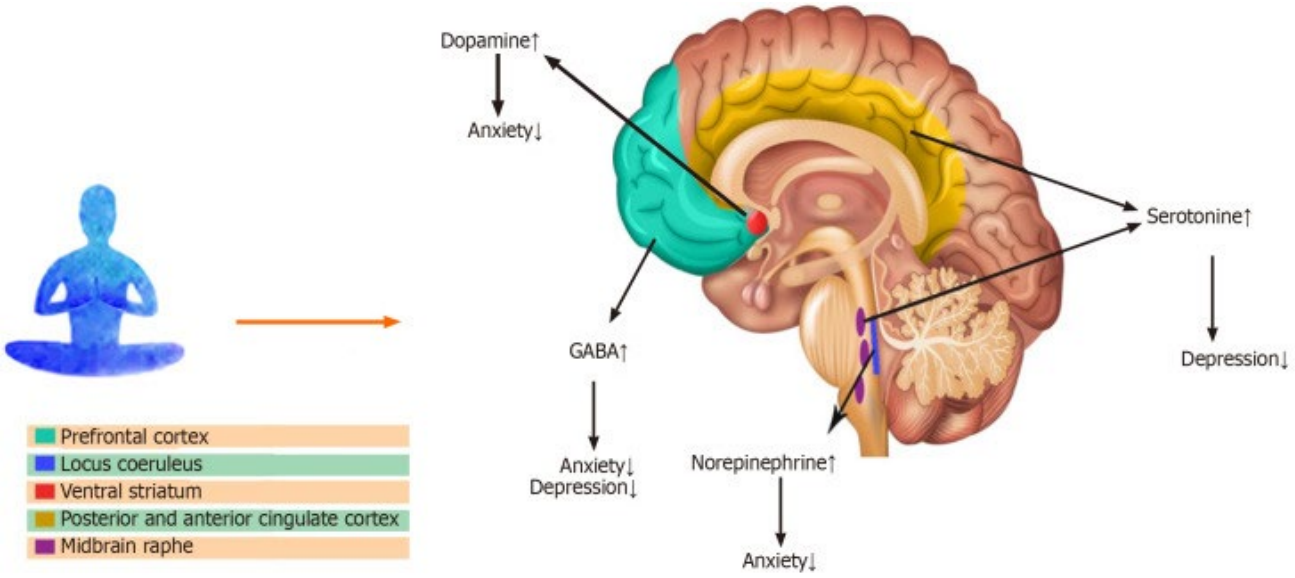
A prospective randomized control study was carried out by Kiran etal. [89] in a single tertiary care centre. One hundred and fifty patients undergoing elective coronary artery bypass surgery were recruited in the study. The patients were randomized in two groups namely, Group 1 [Rajyoga group] and Group 2 [Control Group]. Anxiety was estimated on a visual analogy scale 1–10 prior to the initiation of Rajyoga training or patient counselling [T1], on the morning of the day of surgery [T2], on the 2nd postoperative day [T3], in addition to on the 5th postoperative day [T4]. The serum cortisol level was determined in the morning of the day of surgery [T1], on the 2nd postoperative day [T2] as well as on the 5th postoperative day [T3], respectively. In the study, it was observed that the anxiety level of the patients prior to the surgery T1) as well as on the day of surgery [T2] were comparable amongst the two groups. Nevertheless, on the 2nd postoperative day [T3], the patients who underwent Rajyoga training had lower anxiety level in contrast to the control group [3.12 ± 1.45 vs. 6.12 ± 0.14, $P < 0.05$] as well as on the 5th postoperative day [T4] it was found that Rajyoga practice had led to significant reduction in anxiety level [0.69 ± 1.1 vs. 5.6 ± 1.38, $P < 0.05$]. The serum cortisol quantities were further favourably modulated by the practice of Rajyoga meditation Thus the conclusions drawn were that. Mindbody intervention was observed to be efficacious in diminishing the anxiety of the patients and modulating the quantities of cortisol in patients undergoing well acknowledged stressful surgery like coronary artery bypass surgery [89].

4.4 Effect of RM on obsessive compulsive disorder (OCD)

Patients with obsessive compulsive disorder [OCD] [diagnosed as per the Diagnostic and Statistical Manual of Mental Disorders fifth edition] were divided into two groups - (i) The meditation group [MG], that were inclusive of 28 patients along with (ii) The no meditative group [NMG], that were inclusive of 22 patients. MG practiced RM protocol for 3 months period along with the pharmacological treatment. The NMG continued on pharmacological management as usual. The symptomatology was evaluated at baseline in addition to 3 months using the Yale-Brown Obsessive-Compulsive Scale [Y-BOCS]. At 3 months, both groups illustrated improvement in symptoms. The improvement in MG was statistically significant with an alteration, of 9.0 ± 3.16 in Y-BOCS as well as a $49.76 \pm 9.52\%$ decrease in symptoms. Improvement scores of NMG were also statistically significant with an alteration, 3.13 ± 2.59 in Y-BOCS and $18.09 \pm 14.69\%$ decrease in symptoms. MG illustrated significantly greater improvement in Y-BOCS scores [49.76 ± 9.52] in contrast to NMG [18.09 ± 14.69] with the utilization of the student's paired t-test [$P < 0.001$]. Thus, this study implicated that the RM is an efficacious adjunctive therapy to decrease obsessions in addition to compulsions in patients with OCD [90].

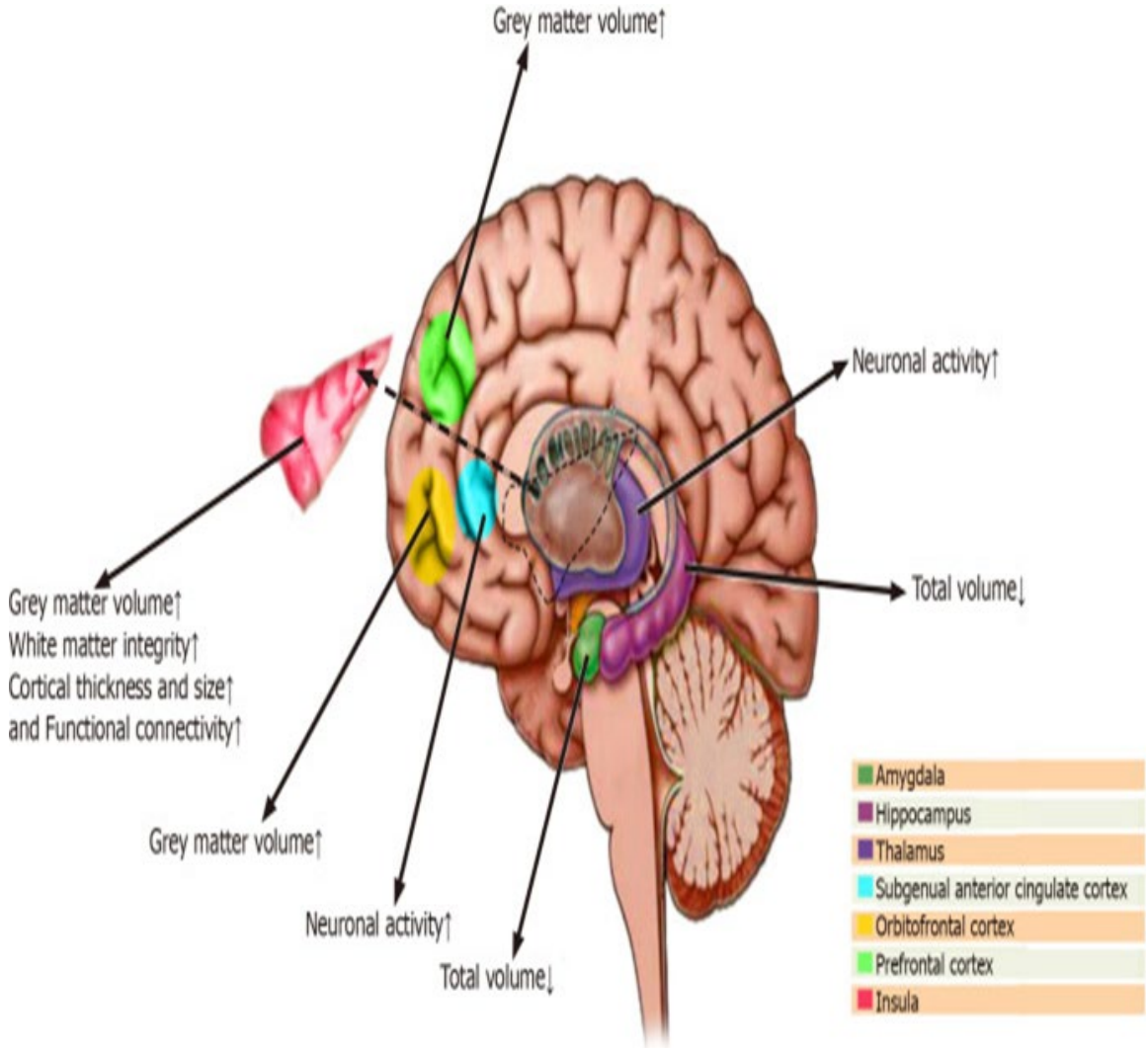
4.5 Effect of RM on treatment of neuropsychological diseases

Yoga is considered a widely-used strategy for health conservation as well as and can be utilized in the form of a treatment approach for a myriad of medical situations, inclusive of neurological in addition to psychological disorders. Hence, Neurollahimoghadam et al. [91], reviewed germane articles involving different neurological in addition to and psychological conditions and along with collected data in the context of how yoga possesses positive influence on patients with a variable variety of conditions, inclusive of its modulatory action on various brain bioelectrical activities, neurotransmitters, along with synaptic plasticity. The part of yoga practice as an element of the treatment of neuropsychological diseases was assessed dependent on these observations. These diseases comprise of psychological disorders like depression, Bipolar disorder, Schizophrenia, Anxiety, along with neurological conditions like Migraine, Parkinson's disease; Alzheimer's disease, Epilepsy as well as Multiple sclerosis [91].



Legend for Figure. 11.

Courtesy ref no 91
The effect of yoga intervention on various neurotransmitters in different brain regions. GABA: γ-aminobutyric acid



Legend for Figure. 12.

Courtesy ref no 91
The effect of yoga practice on the functional activities of various brain regions.

5. Conclusions

Thus, basically RM is separate from hath yoga which involves physical body exercises or asanas whereas this is meant to be the food for mind and positive thinking. We all are souls but significance is what kind of soul like say for mahatma Gandhi mahatma, souls with good virtues as devtasor deities ,all these are in plural only param alias supreme aatma -soul is referred for God which is one and only 1 .while souls doing bad karma are referred to as paapatma.Christ,Budha,Mohammed all came to establish Dharam or religion likeBudhists,For Islam there are 2founders Ibrahim & Mohammed later established Islam both, Christ comes later for Christianity, then Guru Nanak for Sikhism, Jain Dharam etc. With RM & positive thinking as illustrated numerous diseases can get cured varying from SUD, various CAD without any angiography, different Neuropsychological Diseases, cancers. A good example to be cited is a cardiologist who developed an infective brain lesion with Mycobacterium although strain was different from Tuberculosis but a v rare for which in world not many reports were there. He developed convulsions. In CDC Atlanta it was sensitive only to 1 drug of 19 drugs -he had to take daily amikacin which was v toxic and gradually sores in buttocks ,finally iv &surgery done -he thought he was rid but after 1 year another lesion appeared on the other side of brain when dress decided not to operate-finally in consultation with one of dads who told him you are a meditator so try giving in early morning charge to your brain after taking positive energy from supreme soul and subsequent to 1 year to the surprise of other doctors from the MRI the lesion was gone and now he is a v good practicing interventional cardiologist. It is important to do RM early in morning being in soul form by which a good connection with supreme soul is established.

References

1. Aron, A. R. (2011). From reactive to proactive and selective control: developing a richer model for stopping inappropriate responses. Biological psychiatry, 69(12), e55-e68.
2. Chapman, C. D., Benedict, C., Brooks, S. J., & Birger Schist, H. (2012). Lifestyle determinants of the drive to eat: a meta-analysis. The American journal of clinical nutrition, 96(3), 492-497.
3. Volkow, N. D., Wang, G. J., Fowler, J. S., & Telling, F. (2008). Overlapping neuronal circuits in addiction and obesity: evidence of systems pathology. Philosophical Transactions of the Royal Society B: Biological Sciences, 363(1507), 3191-3200.
4. Volkow, N. D., Wang, G. J., Telling, F., Fowler, J. S., Thanes, P. K., Logan, J., et al (2008). Low dopamine striatal D2 receptors are associated with prefrontal metabolism in obese subjects: possible contributing factors. Neuroimage, 42(4), 1537-1543.
5. Volkow, N. D., Wang, G. J., Telling, F., Fowler, J. S., Goldstein, R. Z., et al. (2009). Inverse association between BMI and prefrontal metabolic activity in healthy adults. Obesity, 17(1), 60-65.
6. Corbett, M., Kincade, J. M., & Shulman, G. L. (2002). Neural systems for visual orienting and their relationships to spatial working memory. Journal of cognitive neuroscience, 14(3), 508-523.
7. Corbett, M., & Shulman, G. L. (2002). Control of goal-directed and stimulus-driven attention in the brain. Nature reviews neuroscience, 3(3), 201-215.
8. Kaur, K. K., Alabamia, G., & Singh, M. (2013). Current management of obesity in an infertile female-recent advances and future prospective drugs. Journal of Pharmacy and Nutrition Sciences, 3, 000-000.
9. Kaur, K. K., Allahbadia, G. N., & Singh, M. (2019). Therapeutic Utilization of Neuro Imaging Studies in Obesity for Optimal Utilization of Drugs used in Treatment for Obesity-Lessons Learnt from Bariatric Surgery. J Ageing Restor Med (JARM), 2(2), 89-97.
10. Farr, O. M., Chiang-sham, R. L., & Manzoor's, C. S. (2016). Central nervous system regulation of eating: Insights from human brain imaging. Metabolism, 65(5), 699-713.
11. Volkow, N. D., & Wise, R. A. (2005). How can drug addiction help us understand obesity?. Nature neuroscience, 8(5), 555-560.
12. Volkow, N. D., Wang, G. J., Fowler, J. S., Tomasa, D., et al (2012). Food and drug reward: overlapping circuits in human obesity and addiction. Brain imaging in behavioural neuroscience, 1-24.
13. Volkow, N. D., Wang, G. J., Tomasz, D., & Baler, R. D. (2013). Obesity and addiction: neurobiological overlaps. Obesity reviews, 14(1), 2-18.
14. Micha elides, M., Thanes, P. K., Volkow, N. D., & et al (2012). Translational neuroimaging in drug addiction and obesity. ILAR journal, 53(1), 59-68.
15. Stace, E., Spoor, S., Boon, C., Veldhuis, M. G., & et al (2008). Relation of reward from food intake and anticipated food intake to obesity: a functional magnetic resonance imaging study. Journal of abnormal psychology, 117(4), 924..
16. Doherty, J. P., Eichmann, R., Critchley, H. D., & et al (2002). Neural responses during anticipation of a primary taste reward. Neuron, 33(5), 815-826.
17. Small, D. M., Veldhuis, M. G., Felted, J., Make, Y. E., et al (2008). Separable substrates for anticipatory and consummatory food chemo sensation. Neuron, 57(5), 786-797.
18. Steckel, L. E., Weller, R. E., Cook III, E. W., Twieg, D. B., Knowlton, R. C., et al (2008). Widespread reward-system activation in obese women in response to pictures of high-calorie foods. Neuroimage, 41(2), 636-647.
19. Holsten, L. M., Zaccone, J. R., Brooks, W. M., Butler, M. G., Thompson, T. I., et al. (2006). Neural mechanisms underlying hyperphagia in Prader-Willi syndrome. Obesity, 14(6), 1028-1037.
20. Boutella, K. N., Wiring, C. E., Bischoff-Grete, A., Melrose, A. J., et al (2015). Increased brain response to appetitive tastes in the insula and amygdala in obese compared with healthy weight children when sated. International journal of obesity, 39(4), 620-628.
21. Gasproof, A. M., Acadia, C., Sea, D., Kubit, J., Van Name, M. A., et al (2014). Leptin is associated with exaggerated brain reward and emotion responses to food images in ado-

lescent obesity. Diabetes care, 37(11), 3061-3068.
22. Mehta, S., Mel horn, S. J., Seraglio, A., Tyagi, V., Grabowski, T., et al. (2012). Regional brain response to visual food cues is a marker of satiety that predicts food choice. The American journal of clinical nutrition, 96(5), 989-999..
23. Sun, X., Kroemer, N. B., Veldhuis, M. G., Babbs, A. E., de Araujo, I. E., et al (2015). Basolateral amygdala response to food cues in the absence of hunger is associated with weight gain susceptibility. Journal of Neuroscience, 35(20), 7964-7976.
24. Ulrich-Lai, Y. M., Christiansen, A. M., Wang, X., Song, S., & Herman, J. P. (2016). Statistical modelling implicates neuroanatomical circuit mediating stress relief by 'comfort 'food. Brain Structure and Function, 221, 3141-3156.
25. Martin, A. A., & Davidson, T. L. (2014). Human cognitive function and the obesogenic environment. Physiology & behaviour, 136, 185-193.
26. Parent, M. B., Darling, J. N., & Henderson, Y. O. (2014). Remembering to eat: hippocampal regulation of meal onset. American Journal of Physiology-Regulatory, Integrative and Comparative Physiology, 306(10), R701-R713.
27. Milan ski, M., Degas peri, G., Coope, A., Morori, J., Denis, R., Cintra, D. E., et al (2009). Saturated fatty acids produce an inflammatory response predominantly through the activation of TLR4 signalling in hypothalamus: implications for the pathogenesis of obesity. Journal of Neuroscience, 29(2), 359-370.
28. Thaler, J. P., Choi, S. J., Schwartz, M. W., & Wise, B. E. (2010). Hypothalamic inflammation and energy homeostasis: resolving the paradox. Frontiers in neuroendocrinology, 31(1), 79-84..
29. Guliyev, N. V. (2017). Molecular mechanisms of neuroplasticity: an expanding universe. Biochemistry (Moscow), 82, 237-242.
30. Volkow, N. D., & Boyle, M. (2018). Neuroscience of addiction: relevance to prevention and treatment. American Journal of Psychiatry, 175(8), 729-740.
31. Leisner AI. Addiction is a Brain disease and it matters. Science1997; 278(5336);45-7.
32. Damas to AR. The somatic marker hypothesis and the possible functions of prefrontal cortex. Philon Trans R Soc Lund B Boils Sci 1997; 351 (1346): 413-20.
33. Goldstein, R. Z., & Volkow, N. D. (2011). Dysfunction of the prefrontal cortex in addiction: neuroimaging findings and clinical implications. Nature reviews neuroscience, 12(11), 652-669.
34. VOLKOW, N. D., FOWLER, J. S., WANG, G. J., TELANG, F., et al (2010). 8.3 Imaging Dopamine's Role in Drug Abuse and Addiction. Dopamine handbook, 39, 407.
35. Costa, K. G., Cabral, D. A., Howl, R., & Fontes, E. B. (2019). Rewiring the addicted brain through a psychobiological model of physical exercise. Frontiers in psychiatry, 10, 600.
36. Costa, K. G., Cabral, D. A., Howl, R., & Fontes, E. B. (2019). Rewiring the addicted brain through a psychobiological model of physical exercise. Frontiers in psychiatry, 10, 600..
37. Gouveia-Figueroa, S., & Nodding, M. L. (2015). Validation of a tandem mass spectrometry method using combined extraction of 37 oxylipins and 14 endocannabinoid-related compounds including pros amides from biological matrices. Prostaglandins & other lipid mediators, 121, 110-121.
38. Hansen, H. S., & Diep, T. A. (2009). N-acylethanolamines, anandamide and food intake. Biochemical pharmacology, 78(6), 553-560.
39. Devaney, W. A., Hans, L., Breuer, A., Pertwee, R. G., Stevenson, L. A., et al. (1992). Isolation and structure of a brain constituent that binds to the cannabinoid receptor. Science, 258(5090), 1946-1949.
40. Setout, M., Gretter, U., Fingerless, J., Grim, T. W., Fezza, F., De Petrocelli's, L., et al. (2017). Cannabinoid CB2 receptor ligand profiling reveals biased signalling and off-target activity. Nature communications, 8(1), 13958.
41. Huczynski, M. W., & Parsons, L. H. (2010). Quantification of brain endocannabinoid levels: methods, interpretations and pitfalls. British journal of pharmacology, 160(3), 423-442.
42. Katona I, Freund TF. Multiple functions of endocannabinoid signalling in the brain. Annul Rev Neurosis 2012; 35:529–58.
43. Di Matzo, V., Fontana, A., Codas, H., Schonely, S., Cimino, G., et al (1994). Formation and inactivation of endogenous cannabinoid anandamide in central neurons. Nature, 372(6507), 686-691.
44. Beltramo, M., Stella, N., Carignane, A., Lin, S. Y., Marianna's, A., et al (1997). Functional role of high-affinity anandamide transport, as revealed by selective inhibition. Science, 277(5329), 1094-1097.
45. Wilson, R. I., & Nicoll, R. A. (2001). Endogenous cannabinoids mediate retrograde signalling at hippocampal synapses. Nature, 410(6828), 588-592.
46. Nilsa, R., Duduk, B., Urban, G. M., Mackie, K., Watanabe, M., Cravat, B. F., et al (2008). Enzymatic machinery for endocannabinoid biosynthesis associated with calcium stores in glutamatergic axon terminals. Journal of Neuroscience, 28(5), 1058-1063.
47. Westshore, N., Van Der Stilt, M., Sumbawa, H., Kristel, H., Moers, A., et al. (2006). Forebrain-specific inactivation of GQ/G11 family G proteins results in age-dependent epilepsy and impaired endocannabinoid formation. Molecular and cellular biology, 26(15), 5888-5894.
48. Katharina, S., Gaetani, S., Figley, D., Valine, F., Durante, A., Tontini, .et al(2003). Modulation of anxiety through blockade of anandamide hydrolysis. Nature medicine, 9(1), 76-81.
49. Gobi, G., Bambuco, F. R., Mangier, R., Bertolt, M., Campong, P., Salinas, M., et al (2005). Antidepressant-like activity and modulation of brain monoaminergic transmission by blockade of anandamide hydrolysis. Proceedings of the National Academy of Sciences, 102(51), 18620-18625.
50. Hohmann, A. G., Suplita, R. L., Bolton, N. M., Neely, M. H., Figley, D., et al. (2005). An endocannabinoid mechanism for stress-induced analgesia. Nature, 435(7045), 1108-1112.
51. Morena, M., Patel, S., Bains, J. S., & Hill, M. N. (2016). Neurobiological interactions between stress and the endo-

cannabinoid system. Neuropsychopharmacology, 41(1), 80-102.
52. Hill, M. N., McLaughlin, R. J., Morris, A. C., Vial, V., Fluoresce, S. B., et al. (2009). Suppression of amygdale endocannabinoid signalling by stress contributes to activation of the hypothalamic–pituitary–adrenal axis. Neuropsychopharmacology, 34(13), 2733-2745.
53. Hill, M. N., McLaughlin, R. J., Bingham, B., Shrestha, L., Lee, T. T., et al (2010). Endogenous cannabinoid signalling is essential for stress adaptation. Proceedings of the National Academy of Sciences, 107(20), 9406-9411.
54. Lutz, B., Marsicano, G., Maldonado, R., & Hillard, C. J. (2015). The endocannabinoid system in guarding against fear, anxiety and stress. Nature Reviews Neuroscience, 16(12), 705-718.
55. Morena, M., Roosendaal, B., Terza, V., Ratana, P., Peluso, A., Hauer, D., et al (2014). Endogenous cannabinoid release within prefrontal-limbic pathways affects memory consolidation of emotional training. Proceedings of the National Academy of Sciences, 111(51), 18333-18338.
56. Marsicano, G., Goodenough, S., Monroy, K., Hermann, H., Eder, M., et al (2003). CB1 cannabinoid receptors and on-demand defences against excitotoxicity. Science, 302(5642), 84-88.
57. Jamshid, N., & Taylor, D. A. (2001). Anandamide administration into the ventromedial hypothalamus stimulates appetite in rats. British journal of pharmacology, 134(6), 1151-1154.
58. Van der Stilt, M., Veldhuis, W. B., Van Haaften, G. W., Fezza, F., Bison, T., et al. (2001). Exogenous anandamide protects rat brain against acute neuronal injury in vivo. Journal of Neuroscience, 21(22), 8765-8771.
59. Smith, P. B., Compton, D. R., Welch, S. P., Razvan, R. K., Microlam, R., et al (1994). The pharmacological activity of anandamide, a putative endogenous cannabinoid, in mice. Journal of Pharmacology and Experimental Therapeutics, 270(1), 219-227.
60. Cravatt, B. F., Demarest, K., Patricelli, M. P., Bracey, M. H., Gang, D. K., et al (2001). Supersensitivity to anandamide and enhanced endogenous cannabinoid signaling in mice lacking fatty acid amide hydrolase. Proceedings of the National Academy of Sciences, 98(16), 9371-9376.
61. Williams, C. M., & Kirkham, T. C. (1999). Anandamide induces overeating: mediation by central cannabinoid (CB1) receptors. Psychopharmacology, 143, 315-317.
62. Maccarrone, M., Bab, I., Bíró, T., Cabral, G. A., Dey, S. K., et al (2015). Endocannabinoid signaling at the periphery: 50 years after THC. Trends in pharmacological sciences, 36(5), 277-296.
63. van Eenige, R., van der Stilt, M., Remsen, P. C., & Koeman, S. (2018). Regulation of adipose tissue metabolism by the endocannabinoid system. Trends in Endocrinology & Metabolism, 29(5), 326-337.
64. Gómez, R., Navarro, M., Ferrer, B., Trigon, J. M., Bilbao, A., et al. A peripheral mechanism for CB1 cannabinoid receptor-dependent modulation of feeding. Journal of Neuroscience, 22(21), 9612-9617.
65. Clapper, J. R., Moreno-Sanz, G., Russo, R., Guitarrón, A., Macondo, F., et al (2010). Anandamide suppresses pain initiation through a peripheral endocannabinoid mechanism. Nature neuroscience, 13(10), 1265-1270.
66. Carignane, A., Rana, G. L., Giuffrida, A., & Piomelli, D. (1998). Control of pain initiation by endogenous cannabinoids. Nature, 394(6690), 277-281.
67. Munro, S., Thomas, K. L., & Abu-Saar, M. (1993). Molecular characterization of a peripheral receptor for cannabinoids. Nature, 365(6441), 61-65.
68. Pertwee, R. G., Howlett, A. C., Abood, M. E., Alexander, S. P. H., Di Marzo, V., et al (2010). International Union of Basic and Clinical Pharmacology. LXXIX. Cannabinoid receptors and their ligands: beyond CB1 and CB2. Pharmacological reviews, 62(4), 588-631.
69. Turcotte, C., Blanchet, M. R., Laviolette, M., & Flamand, N. (2016). The CB 2 receptor and its role as a regulator of inflammation. Cellular and Molecular Life Sciences, 73, 4449-4470.
70. 7Di Marzo, V., & De Petrocellis, L. (2012). Why do cannabinoid receptors have more than one endogenous ligand?. Philosophical Transactions of the Royal Society B: Biological Sciences, 367(1607), 3216-3228.
71. Turcotte, C., Chouinard, F., Lefebvre, J. S., & Flamand, N. (2015). Regulation of inflammation by cannabinoids, the endocannabinoids 2-arachidonoyl-glycerol and arachidonoyl-ethanolamide, and their metabolites. Journal of leukocyte biology, 97(6), 1049-1070.
72. Zygmunt, P. M., Petersson, J., Andersson, D. A., Chuang, H. H., Sørgård, M., et al(1999). Vanilloid receptors on sensory nerves mediate the vasodilator action of anandamide. Nature, 400(6743), 452-457.
73. Stelt MVD, Marzo VD. End vanilloids. Eur J Brioche 2004;271:1827–34.
74. Marzo, V. D., & Petrocellis, L. D. (2010). Endocannabinoids as regulators of transient receptor potential (TRP) channels: a further opportunity to develop new endocannabinoid-based therapeutic drugs. Current medicinal chemistry, 17(14), 1430-1449.
75. Tooth A, Blumberg PM, Boncza's J. Vitamins & Hormones. 81. Academic Press; 2009. p. 389–419. Ch. 15.
76. Martins, D., Tavares, I., & Mogador, C. (2014). "Hotheaded": the role OF TRPV1 in brain functions. Neuropharmacology, 85, 151-157.
77. Marron, M. C., Morabito, A., Gustier, M., Churchie, V., Luetic, A., et al. (2017). TRPV1 channels are critical brain inflammation detectors and neuropathic pain biomarkers in mice. Nature communications, 8(1), 15292.
78. Mock, E. D., Gagestein, B., & van der Stelt, M. (2022). Anandamide and other N-acylethanolamines: A class of signaling lipids with therapeutic opportunities. Progress in Lipid Research, 101194.
79. Ramesh, M., Stahlian, B., Sinus, E., & Kirani, S. R. (2013). Efficacy of raja yoga meditation on positive thinking: An index for self-satisfaction and happiness in life. Journal of clinical and diagnostic research: JCDR, 7(10), 2265.
80. Laski, L. S., Moyal, N., & Henik, A. (2021). Rumination, emotional intensity and emotional clarity. Consciousness and Cognition, 96, 103242..

81. Cooper, A. C., Ventura, B., & North off, G. (2022). Beyond the veil of duality—topographic reorganization model of meditation. Neuroscience of Consciousness, 2022(1), niac013.
82. .Katyal, S. (2022). Reducing and deducing the structures of consciousness through meditation. Frontiers in Psychology, 5410.
83. Ramsay, T., Manderson, L., & Smith, W. (2010). Changing a mountain into a mustard seed: Spiritual practices and responses to disaster among New York Brahma Kumari's. Journal of Contemporary Religion, 25(1), 89-105.
84. Telles, S., & Desi Raju, T. (1993). Automatic changes in Ramkumar's Raja yoga meditation. International Journal of Psychophysiology, 15(2), 147-152.
85. Sharma, K., Chandra, S., & Dubey, A. K. (2018). Exploration of lower frequency EEG dynamics and cortical alpha asymmetry in long-term raj yoga meditators. International journal of yoga, 11(1), 30.
86. Dissanayake, N. N., Sirisena, N. D., & Samaranayake, N. (2022). Impact of meditation-based lifestyle practices on mindfulness, wellbeing, and plasma telomerase levels: a case-control study. Frontiers in Psychology, 562.
87. Babe, M. R., Cadaver, R., Koteshwara, P., Sathian, B., & Rai, K. S. (2020). Rajyoga meditation induces grey matter volume changes in regions that process reward and happiness. Scientific reports, 10(1), 16177.
88. Gupta, S. K., Sawhney, R. C., Rai, L., Chavan, V. D., Dani, S., et al (2011). Regression of coronary atherosclerosis through healthy lifestyle in coronary artery disease patients--Mount Abu Open Heart Trial. Indian heart journal, 63(5), 461-469.
89. Kiran, U., Ladha, S., Makhija, N., Kapoor, P. M., Choudhury, M., et al (2017). The role of Rajyoga meditation for modulation of anxiety and serum cortisol in patients undergoing coronary artery bypass surgery: A prospective randomized control study. Annals of cardiac anaesthesia, 20(2), 158.
90. Mehta, K., Mehta, S., Chalana, H., Singh, H., & Thaman, R. G. (2020). Effectiveness of Rajyoga meditation as an adjunct to first-line treatment in patients with obsessive compulsive disorder. Indian Journal of Psychiatry, 62(6), 684.
91. Nourollahimoghadam, E., Gorji, S., Gorji, A., & Ghadiri, M. K. (2021). Therapeutic role of yoga in neuropsychological disorders. World journal of psychiatry, 11(10), 754.