## NeuroRegulation



# Slouched Posture, Sleep Deprivation, and Mood Disorders: Interconnection and Modulation by Theta Brain Waves

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#### **Abstract**

Factors such as sleep, posture, and diet can impact EEG readings and have physiological and neurological effects that, when in dysfunctional ranges, may increase susceptibility to developing affective mood disorders or other psychiatric issues. Based on an observation of a neurofeedback client generating excessive amounts of theta rhythms while in a slouched posture, we discuss the role of theta rhythms in brain function and emotional regulation. Slouched posture has been strongly correlated with depressive symptoms. Although the precise nature of the relationship between slouched posture, sleep, and depressive symptoms remains unclear, the literature suggests a cyclical, reciprocal dynamic that is modulated by the involvement of theta rhythms. We recommend that neurofeedback practitioners assess their patients' posture while training, as it could affect the training's effectiveness. Sleep patterns should be assessed prior to the initiation of neurofeedback; if sleep issues remain a consistent problem, efforts to optimize the biological matrix may be indicated. Simple changes in body posture, diet monitoring, and strategies to reduce sleep deprivation may be helpful.

Keywords: body posture; REM sleep; mood disorders; theta rhythms; emotional regulation

Citation: Barr, E. A., Peper, E., & Swatzyna, R. J. (2019). Slouched posture, sleep deprivation, and mood disorders: Interconnection and modulation by theta brain waves. *NeuroRegulation*, 6(4), 181–189. https://doi.org/10.15540/nr.6.4.181

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A 19-year-old male diagnosed with major depressive disorder (MDD) and attention-deficit/hyperactivity disorder (ADHD), during a neurofeedback session, began to produce excessive amounts of theta brain waves in the frontal lobe, which he was having difficulty suppressing. Upon noticing that his posture was slouched, with his head tilted downwards, he was asked to correct his posture by sitting straight up, and the theta rhythms diminished once this was done.

This observation spurred our investigation into the possible relationship between body posture, theta rhythms, sleep, and emotional affect, particularly in the case of affective mood disorders.

#### Introduction

With the increasing incorporation of electroencephalograph (EEG) technology into psychiatric practice, more attention is being directed to the role of brain waves in the manifestation of psychiatric issues such as mood. Preliminary evidence suggests that specific EEG patterns are associated with certain affective states. Emotional affect has been described as a function of the interplay of chemical, electrophysiological, and homeostatic processes (John & Prichep, 2006), thus EEG could reasonably reflect emotional states.

An aspect of sleep that is often overlooked is its role in emotional functioning. Many conceptualize sleep as a process by which one replenishes their energy and "recharges" for the next day. This perspective may be too narrow since research indicates that the

benefits of getting a good night's rest and the consequences of sleep deprivation extend far beyond that. Further, the interplay between sleep and posture may be far more cyclical than previously thought.

Research on the relationship between posture and mood suggests a strong correlation between slouched posture and depressive symptoms (Peper & Lin, 2012; Peper, Lin, Harvey, & Perez, 2017). While slouched, it is more difficult to access positive memories than in the erect posture, and the brain is more activated in the slouched position than erect position while trying to access positive memories (Peper et al., 2017; Tsai, Peper, & Lin, 2016; Wilson, & Peper, 2004). This indicates that accessing positive memories while in a slouched posture requires more areas of the brain to be active to accomplish this task than it would in an erect posture.

This paper focuses on the possible affect fmodulation of oscillatory theta rhythms, 4.0–7.5 Hz slow waves, and how this modulation is altered by the lifestyle factors of sleep and body posture.

#### **Theta and Brain Function**

Theta brain waves have been broadly categorized into two types: hippocampal theta rhythms and cortical theta rhythms (Theta Waves, n.d.). Hippocampal theta is an oscillatory slow-wave rhythm that is generated subcortically, as the name implies, in the hippocampus. The majority of research on hippocampal theta comes from studies conducted on rodents and neurosurgical/epileptic patients that have had electrodes implanted deep into the hippocampus or surrounding subcortical structures; scalp EEGs cannot detect this type of theta. While its exact functions in humans are still hotly debated and heavily associated with sleep (Buzsáki, 2002), it is important to note that the presence of theta may not be indicative of a less active or idle brain. It has been suggested that hippocampal theta is actually linked to attention and arousal (Jacobs, Lega, & Watrous, This type of theta has been linked to sensorimotor functions (Bland & Oddie, 2001; Burgess, Barry, & O'Keefe, 2007; Ekstrom et al., 2005; Hoffman et al., 2013; Jacobs et al., 2017; Lopour, Tavassoli, Fried, & Ringach, 2013; Mormann et al., 2005; Watrous, Fried, & Ekstrom, 2011), spatial navigation (Aitken, Zheng, & Smith, 2018; Ekstrom et al., 2005; Jacobs et al., 2017; Kahana, Sekuler, Caplan, Kirschen, & Madsen, 1999; Vass et al., 2016; Watrous et al., 2011), episodic memory coding (Jacobs et al., 2017), and, of particular interest, head position all through its connection to the vestibular system (Aitken et al., 2018).

Alternately, cortical theta rhythms can be detected on a scalp EEG, despite being generated subcortically. An important distinction is that this type of theta is, in actuality, alpha rhythms that have slowed into the theta band (personal communication with Jay Gunkleman, July 11, 2019). We will continue to refer to these rhythms as theta on the basis that they are functionally similar to hippocampal theta. A form of this cortical theta has been termed "frontal midline theta" (FMT) and has been the subject of research. Ishihara and Yoshii (1972) first introduced the term to describe the prominent theta oscillations found primarily at the Fz electrode site while doing arithmetic. This finding, as well as numerous subsequent studies, has led to the association of FMT with working memory (Gevins, Smith, McEvoy, & Yu, 1997; Hsieh, Ekstrom, & Ranganath, 2011; Jensen & Tesche, 2002; Raghavachari et al., 2001) and sustained attention over a long period of time (Hseih & Ranganath, 2014). Another speculation is that FMT has a function in temporal order maintenance: individual items in a sequence are associated with each successive theta phase (Hseih et al., 2011).

The commonality among these neural functions is the cooperation of disparate areas of the brain. While engaging in a task like navigation, a person uses numerous cognitive functions simultaneously: visual perception (to see their environment), motor functions (to coordinate body movement), vestibular system (to maintain balance while in motion and detect the speed of movement), sensory integration, etc. It has been posited that theta rhythms help to coordinate brain-wide network functioning (Buzsáki, 1996; Zhang & Jacobs, 2015), wherein theta rhythms act as a "carrier frequency" that allows disparate areas of the brain to interact (Goldstein & Walker, 2014).

### Vestibular System, Head Position, and Theta Production

Recent research has found high comorbidity between psychiatric issues, such as depression, anxiety, and panic disorder, and vestibular system dysfunction (Mast, Preuss, Hartmann, & Grabherr, 2014). This could account for vestibular symptoms, such as dizziness, often experienced by those with anxiety disorders. Those with panic disorder and certain specific phobias (i.e., agoraphobia, acrophobia, basophobia, etc.) have been shown to have multisensory integration issues (Jacob, Furman, Durrant, & Turner, 1996; Mast et al., 2014), particularly concerning inadequate visual and

proprioceptive input. The connection between the vestibular system and emotional processing is much more direct; a region of the brain stem (an area known to produce theta rhythms), the parabrachial nucleus (PBN), provides a direct link between the vestibular system and the emotional processing structures of the brain (Balaban, 2004; Mast et al., 2014) that have been implicated in affective disorders. Therefore, it may be that dysfunction in the vestibular system correlates with impaired emotional processing.

One of the functions of the vestibular system, specifically that of the vestibular organs within the inner ear, is to allow people to sense head acceleration; from this, the head position can then be calculated (Aitken et al., 2018). While there is currently very little research on how EEG brain waves change as head position shifts, Spironelli, Busenello, and Angrilli (2016) found that lying in a supine position correlated with decreased cortical activity and increased alpha and delta wave amplitude compared to seated position. Given these effects, it may be reasonable to think that tilting the head downwards, in a chin-to-chest direction, would also produce noticeable changes in brain wave activity.

#### **REM Sleep and Emotional Reactivity**

Many people report that when they have been sleep-deprived, having stayed up much too late, they woke up feeling cognitively absent, physically exhausted, and probably quite cranky. Subjective self-reports have consistently linked sleep deprivation with increased emotional volatility and impairments to attention, alertness, and memory (Horne, 1985). Although the irritability is often explained as the result of being tired, it may be that being easily agitated is not a side effect of being tired; rather, it is a direct side effect of the lack of sleep itself.

The limbic lobe has been implicated in a variety of emotional functioning processes; during REM sleep, emotion-related subcortical structures (the amygdala, striatum, and hippocampus) and cortical areas such as the medial prefrontal cortex (mPFC) and the insula (Dang-Vu et al., 2010; Goldstein & Walker, 2014; Miyauchi, Misaki, Kan, Fukunaga, & Koike, 2009; Nofzinger, 2005) show approximately similar, if not higher, levels of activity as with resting wakefulness (Dolcos, LaBar, & Cabeza, 2005). This further suggests that the emotional processing that occurs during REM sleep is equally as impactful as that which occurs during wakefulness.

One week of sleep deprivation (4 to 5 hours of sleep [Dinges et al., 1997; Motomura et al., 2013]) has been

shown to amplify the sensitivity and reactivity of dopaminergic limbic structures (Gujar, Yoo, Hu, & Walker, 2011), particularly the amygdala. This is coupled with dulling of the regulatory executive functions of the prefrontal cortex, resulting in decreased alertness, attention, and memory recall. Yoo, Gujar, Hu, Jolesz, and Walker (2007) estimated that one night of sleep deprivation increases the sensitivity and reactivity of the amygdala by 60%, a condition made more severe by the decreased regulatory connectivity to the mPFC. This results in an overreactive, hypersensitive amygdala being allowed to run rampant, unchecked by the mPFC; denying oneself the proper allotment of sleep could mean not allowing certain processes to occur.

One of the often-unnoticed functions of REM sleep is a process called "next day emotional recalibration"; this process functions to recalibrate the brain's sensitivity in response to emotional events and it primes the brain to react appropriately to emotional experiences that occur the next day (Goldstein & Walker, 2014). Not only that, but it also "strip[s] away the visceral charge (the emotion) from affective experiences of the prior day(s), depotentiating their emotional strength while still consolidating the information (the memory) contained within that experience," (Goldstein & Walker, 2014, p. 9). This is particularly relevant to emotionally challenging or distressing experiences (Phelps, Delgado, Nearing, & LeDoux, 2004; Van der Helm & Walker, 2012; Walker, 2009) since "deficits in the extinction and ability to appropriately utilize surrounding information underlie fear-related disorders such as specific phobia and PTSD" (Goldstein & Walker, 2014, p. 8). In other words, REM sleep may serve as an emotional reset button that removes the emotions associated with memories from that day while also preparing for emotional experiences that one will encounter the next day. It may be that those who are deprived of this function are much more likely to develop more severe psychiatric symptoms.

Sleep abnormalities have been associated with many affective psychopathologies. The altered reactivity of the mesolimbic structures and reduced connectivity to the prefrontal cortex is a neurological pattern often seen in disorders such as depressive disorders (Siegle, Thompson, Carter, Steinhauer, & Thase, 2007), bipolar disorder (Drevets, Savitz, & Trimble, 2008), anxiety disorders (Davidson, 2002; Etkin & Wager, 2007; Nitschke et al., 2009; Paulus & Stein, 2006), substance use disorder (SUD; Arnedt, Conroy, & Brower, 2007; Brower & Perron, 2010), and PTSD (Rauch et al., 2000; Shin, Rauch, & Pitman, 2006). In

these disorders, sleep abnormalities are highly comorbid and are, in some cases, part of the diagnostic criteria.

#### **Posture and Mood**

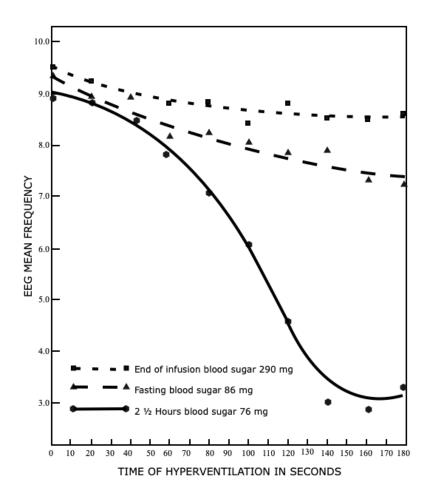
Peper and Lin (2012) demonstrated that if people tried skipping versus walking in a slouched posture, subjective energy after the exercise was significantly higher. Among the participants who had reported the highest level of depression during the last two years, there was a significant decrease of subjective energy when they walked in a slouched position as compared to those who reported a low level of depression. There was also a shift in the EEG power during recall of positive memories in the collapsed state. Tsai, Peper, and Lin (2016) showed that when participants sat in a collapsed position, evoking positive thoughts required more "brain activation" (i.e., greater mental effort) compared to that required when walking in an upright position. While in the upright position, it is easier to access positive thoughts and experience significantly improved cognitive performance. Namely, in the upright position, participants report that performing mental serial subtraction is significantly easier than in the slouched position (Peper, Harvey, Mason, & Lin, 2018). Although it was highly significant for the whole group (125 students). posture did not affect those students who reported that they did not have test anxiety, math difficulty, and "blanking out" scores.

Most likely, the collapsed posture evokes a defense reaction that occurred previously during an experience of defeat and hopelessness. Then, if one is in a slouched position, it is easier to access hopeless, helpless, powerless, and defeated

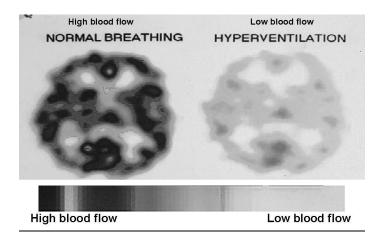
memories than in an upright position (Peper et al., 2017). Thus, posture becomes the conditioned stimulus to trigger the emotions and body state associated with fear and defeat. Even physically, the person experiences reduced strength to resist the downward pressure on their arm when standing collapsed versus erect (Peper, Booiman, Lin, & Harvey, 2016). The slouched position also tends to increase shallow thoracic breathing and slightly reduce heart rate variability (Peper et al., 2017).

For some people, shallow, rapid breathing could cause overbreathing (hyperventilation) which lowers the partial pressure of carbon dioxide (pCO2) and may interact with low blood sugar to reduce EEG frequency. Namely, if blood sugar is very low and a person hyperventilates, the EEG frequency decreases into the theta range, as shown in Figure 1, which would also reduce the cerebral circulation as shown in Figure 2.

In addition, blood circulation may be affected in the slouched position when the person looks upward and scrunches their neck. Harvey, Peper, Booiman, Heredia Cedillo, and Villagomez (2018) showed that when participants scrunched their necks for 30 s, 98.4% of the participants experienced a significant increase in symptoms of pressure in the head, stiff neck, eye tension, and headaches. Although the symptoms may be caused by muscle tension, it may also be caused by vertebrobasilar insufficiency due to the scrunching of the neck, which causes transient or permanent reduction or cessation of blood supply to the hindbrain through the left and right vertebral arteries (VA) and the basilar artery (Kerry, Taylor, Mitchell, McCarthy, & Brew, 2008).



**Figure 1.** EEG mean frequency change at different blood sugar levels. Adaptation of graph from 'Hyperventilation: Analysis of clinical symptomatology,' by G. L. Engel, E. G. Ferris, and M. Logan, 1947, *Annals of Internal Medicine, 27*(5), p. 690.



**Figure 2.** Blood flow through the brain in healthy subjects as measured with single photon emission computed tomography (SPECT) by looking at the brain from above during normal breathing and hyperventilation. The darker color indicates increased blood flow. During hyperventilation (PCO2 < 20 torr) there was a significant decrease in blood flow throughout the brain. Reprinted with permission from Scott Woods, M. D., Yale University, unpublished data, 1987.

#### **Discussion**

The precise nature of the relationship between slouched posture, sleep, and depressive symptoms remains unclear, although the literature suggests a cyclical, reciprocal dynamic that is modulated by the involvement of theta rhythms (see Figure 3).

Maintaining poor sleep habits, either by hypersomnia or insomnia, may contribute to disrupted emotional

functioning or increase the severity of existing depressive symptoms through the heightened sensitivity of limbic structures. Additionally, poor sleep, particularly in the case of insomnia, can lead to fatigue, thus increasing the likelihood of having slouched posture. This paper focused on the consequences of insomniac sleep habits, so the effects of hypersomnia within this dynamic remains unclear. An avenue of future research could be to investigate how, or if, hypersomnia fits into this model.

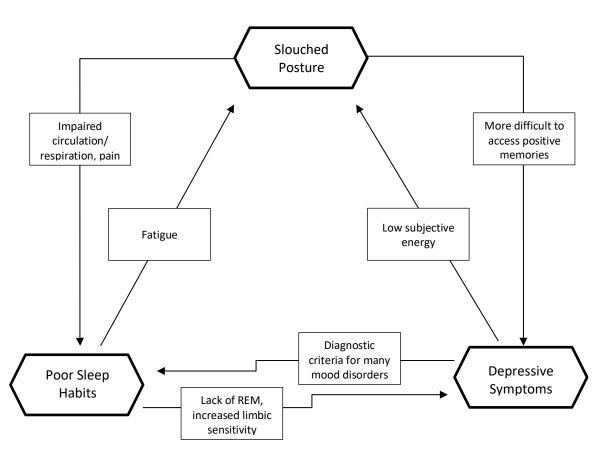


Figure 3. The relationship and feedback loops between posture, sleep and depressive symptoms.

Slouched posture has been found to have a number of physiological effects that could interfere with one's ability to get adequate sleep, increase shallow respiration, impair cerebral circulation, and increase symptoms such as headaches, back/shoulder/neck pain, etc. Although theta rhythms are heavily associated with sleep, they are not known to facilitate the descent into sleep stages. Given that theta rhythms have been found to correspond with heightened mental activity and that hyperventilation, and subsequent shallow breathing, can lead to increased theta rhythms, we can see how poor posture could cause poor sleep through the

generation of excessive theta. We also see how the association of slouched posture with feelings of defeat and hopelessness can lead to a fixation on memories connected with such feelings, making it more difficult to recall more positive memories to mind.

Affective mood disorders are highly comorbid with dysfunctional sleep, both in the forms of hypersomnia and insomnia; this correlation is so robust that sleep issues are part of the diagnostic criteria. This aspect appears to resemble a negative feedback loop in which symptom severity increases as sleep

decreases. The reports of low subjective energy in those with depressive symptoms can lead to slouched posture in a similar fashion to lack of sleep. While sleep deprivation seems to cause bodily fatigue, depression seems to elicit a type of mental fatigue. This could mean that slouched posture is the more likely point of access to this dynamic. Developing slouched posture, routinely poor sleep habits, or simply developing depressive symptoms through neurochemical or physiological process are all viable entry points to this vicious interrelationship.

A fairly new field of study, affective neuroscience, seeks to determine the neurological basis for mood and emotions. This area could be the catalyst for further research on the relationship between posture, sleep, and mood disorders. The effects of head tilt need to be investigated more thoroughly; perhaps habitual downward head tilt and the prolonged compression of the frontal lobes against the inside of the skull could have bruise-like effects. If this is the case, then poor posture could potentially have side effects more similar to that of concussive brain injuries.

The connection between limbic structures and the vestibular system could provide avenues for novel treatment approaches. Evidence suggests that stimulation of the vestibular system can alleviate depressive symptoms in some cases. In the instances that affective mood disorders are comorbid with vestibular dysfunction, it is possible that treating the vestibular issues could also treat the mood disorder. Further research needs to be done to determine the treatment efficacy of this approach.

#### **Clinical Implications for Neurofeedback Training**

When training clients with neurofeedback to reduce theta and increase beta or other EEG frequencies. EEG mastery and clinical efficacy may be increased if the biological matrix is optimized to inhibit theta. This includes sleep hygiene, diet to hypoglycemic states, mastery of diaphragmatic breathing to avoid hyperventilation, and posture. We recommend that neurofeedback practitioners, before beginning neurofeedback, first assess sleep patterns and explore strategies to reduce sleep deprivation and to monitor diet. Then, teach the client self-care skills to optimize health. This means educating the client in 1) sleep hygiene, 2) appropriate diet choices to reduce hypoglycemic states, and 3) awareness of posture and how to achieve and maintain an erect, upright posture to enhance empowerment and increase vertebral artery circulation to inhibit theta.

These self-care skills will optimize the physiology to reduce central theta.

#### **Acknowledgments**

The authors would like to thank Dr. Lisa Wines, PhD, LPC, CSC, for her contributions and edits to this paper.

#### **Author Disclosure**

Authors have no grants, financial interests, or conflicts to disclose.

#### References

- Aitken, P., Zheng, Y., & Smith, P. F. (2018). The modulation of hippocampal theta rhythm by the vestibular system. *Journal of Neurophysiology*, 119(2), 548–562. https://doi.org/10.1152 /in.00548.2017
- Arnedt, J. T., Conroy, D. A., & Brower, K. J. (2007). Treatment options for sleep disturbances during alcohol recovery. *Journal of Addictive Diseases*, 26(4), 41–54. https://doi.org/10.1300/J069v26n04\_06
- Balaban, C. D. (2004). Projections from the parabrachial nucleus to the vestibular nuclei: Potential substrates for autonomic and limbic influences on vestibular responses. *Brain Research*, *996*(1), 126–137. https://doi.org/10.1016/j.brainres.2003.10.026
- Bland, B. H., & Oddie, S. D. (2001). Theta band oscillation and synchrony in the hippocampal formation and associated structures: The case for its role in sensorimotor integration. Behavioural Brain Research, 127(1–2), 119–136. https://doi.org/10.1016/s0166-4328(01)00358-8
- Brower, K. J., & Perron, B. E. (2010). Sleep disturbance as a universal risk factor for relapse in addictions to psychoactive substances. *Medical Hypotheses*, *74*(5), 928–933. https://doi.org/10.1016/j.mehy.2009.10.020
- Burgess, N., Barry, C., & O'Keefe, J. (2007). An oscillatory interference model of grid cell firing. *Hippocampus*, *17*(9), 801–812. https://doi.org/10.1002/hipo.20327
- Buzsáki, G. (1996). The hippocampo-neocortical dialogue. Cerebral Cortex, 6(2), 81–92. https://doi.org/10.1093/cercor/6.2.81
- Buzsáki, G. (2002). Theta oscillations in the hippocampus. *Neuron*, 33(3), 325–340. https://doi.org/10.1016/S0896-6273(02)00586-X
- Dang-Vu, T. T., Schabus, M., Desseilles, M., Sterpenich, V., Bonjean, M., & Maquet, P. (2010). Functional neuroimaging insights into the physiology of human sleep. *Sleep*, 33(12), 1589–1603. https://doi.org/10.1093/sleep/33.12.1589
- Davidson, R. J. (2002). Anxiety and affective style: Role of prefrontal cortex and amygdala. *Biological Psychiatry*, 51(1), 68–80. https://doi.org/10.1016/s0006-3223(01)01328-2
- Dinges, D. F., Pack, F., Williams, K., Gillen, K. A., Powell, J. W., Ott, G. E., ... Pack, A. I. (1997). Cumulative sleepiness, mood disturbance, and psychomotor vigilance performance decrements during a week of sleep restricted to 4–5 hours per night. Sleep, 20(4), 267–277. https://doi.org/10.1093/sleep/20.4.267
- Dolcos, F., LaBar, K. S., & Cabeza, R. (2005). Remembering one year later: Role of the amygdala and the medial temporal lobe memory system in retrieving emotional memories. *Proceedings of the National Academy of Sciences*, 102(7), 2626–2631. https://doi.org/10.1073/pnas.0409848102
- Drevets, W. C., Savitz, J., & Trimble, M. (2008). The subgenual anterior cingulate cortex in mood disorders. *CNS Spectrums*, 13(8), 663–681. https://doi.org/10.1017/s1092852900013754

Ekstrom, A. D., Caplan, J. B., Ho, E., Shattuck, K., Fried, I., & Kahana, M. J. (2005). Human hippocampal theta activity during virtual navigation. *Hippocampus*, 15(7), 881–889.

https://doi.org/10.1002/hipo.20109

Engel, G. L., Ferris., E. G., & Logan, M. (1947). Hyperventilation: Analysis of clinical symptomatology. *Annals of Internal Medicine*, 27(5), 683–704.

- Etkin, A., & Wager, T. D. (2007). Functional neuroimaging of anxiety: A meta-analysis of emotional processing in PTSD, social anxiety disorder, and specific phobia. *American Journal* of Psychiatry, 164(10), 1476–1488. https://doi.org/10.1176/appi.ajp.2007.07030504
- Gevins, A., Smith, M. E., McEvoy, L., & Yu, D. (1997). High-resolution EEG mapping of cortical activation related to working memory: Effects of task difficulty, type of processing, and practice. *Cerebral Cortex*, 7(4), 374–385. https://doi.org/10.1093/cercor/7.4.374
- Goldstein, A. N., & Walker, M. P. (2014). The role of sleep in emotional brain function. *Annual Review of Clinical Psychology*, *10*, 679–708. https://doi.org/10.1146/annurev-clinpsy-032813-153716
- Gujar, N., Yoo, S., Hu, P., & Walker, M. P. (2011). Sleep deprivation amplifies reactivity of brain reward networks, biasing the appraisal of positive emotional experiences. *The Journal of Neuroscience*, 31(12), 4466–4474. https://doi.org /10.1523/ineurosci.3220-10.2011
- Gunkleman, J. (July 11, 2019). Personal communications.
- Harvey, R., Peper, E., Booiman, A., Heredia Cedillo, A., & Villagomez, E. (2018). The effect of head and neck position on head rotation, cervical muscle tension and symptoms. *Biofeedback*, 46(3), 65–71. https://doi.org/10.5298/1081-5937-46.3.04
- Hoffman, K. L., Dragan, M. C., Leonard, T. K., Micheli, C., Montefusco-Siegmund, R., & Valiante, T. A. (2013). Saccades during visual exploration align hippocampal 3–8 Hz rhythms in human and non-human primates. Frontiers in Systems Neuroscience, 7(43). https://doi.org/10.3389 /fnsys.2013.00043
- Horne, J. A. (1985). Sleep function, with particular reference to sleep deprivation. *Annals of Clinical Research*, *17*(5), 199–208. https://www.ncbi.nlm.nih.gov/pubmed/3909914
- Hsieh, L., Ekstrom, A. D., & Ranganath, C. (2011). Neural oscillations associated with item and temporal order maintenance in working memory. *The Journal of Neuroscience*, *31*(30), 10803–10810. https://doi.org/10.1523/jneurosci.0828-11.2011
- Hsieh, L., & Ranganath, C. (2014). Frontal midline theta oscillations during working memory maintenance and episodic encoding and retrieval. *NeuroImage*, *85*, 721–729. https://doi.org/10.1016/j.neuroimage.2013.08.003
- Ishihara, T., & Yoshii, N. (1972). Multivariate analytic study of EEG and mental activity in Juvenile delinquents. Electroencephalography and Clinical Neurophysiology, 33(1), 71–80. https://doi.org/10.1016/0013-4694(72)90026-0
- Jacob, R. G., Furman, J. M., Durrant, J. D., & Turner, S. M. (1996). Panic, agoraphobia, and vestibular dysfunction. *The American Journal of Psychiatry*, 153(4), 503–512. https://doi.org/10.1176/ajp.153.4.503
- Jacobs, J., Lega, B., & Watrous, A. J. (2017). Human hippocampal theta oscillations: Distinctive features and interspecies commonalities. In D. E. Hannula & M. C. Duff (Eds.), The Hippocampus from Cells to Systems: Structure, Connectivity, and Functional Contributions to Memory and Flexible Cognition (pp. 37–67). New York, NY: Springer.
- Jensen, O., & Tesche, C. D. (2002). Frontal theta activity in humans increases with memory load in a working memory task. European Journal of Neuroscience, 15(8), 1395–1399. https://doi.org/10.1046/j.1460-9568.2002.01975.x

John, E. R., & Prichep, L. S. (2006). The relevance of QEEG to the evaluation of behavioral disorders and pharmacological interventions. *Clinical EEG and Neuroscience*, *37*(2), 135–143. https://doi.org/10.1177/155005940603700210

- Kahana, M. J., Sekuler, R., Caplan, J. B., Kirschen, M., & Madsen, J. R. (1999). Human theta oscillations exhibit task dependence during virtual maze navigation. *Nature*, 399(6738), 781–784. https://doi.org/10.1038/21645
- Kerry, R., Taylor, A. J., Mitchell, J., McCarthy, C., & Brew, J. (2008). Manual therapy and cervical arterial dysfunction, directions for the future: A clinical perspective. *Journal of Manual & Manipulative Therapy*, 16(1), 39–48. https://doi.org/10.1179/106698108790818620
- Lopour, B. A., Tavassoli, A., Fried, I., & Ringach, D. L. (2013). Coding of information in the phase of local field potentials within human medial temporal lobe. *Neuron*, *79*(3), 594–606. https://doi.org/10.1016/j.neuron.2013.06.001
- Mast, F. W., Preuss, N., Hartmann, M., & Grabherr, L. (2014). Spatial cognition, body representation and affective processes: The role of vestibular information beyond ocular reflexes and control of posture. Frontiers in Integrative Neuroscience, 8, 44. https://doi.org/10.3389/fnint.2014.00044
- Miyauchi, S., Misaki, M., Kan, S., Fukunaga, T., & Koike, T. (2009). Human brain activity time-locked to rapid eye movements during REM sleep. *Experimental Brain Research*, 192(4), 657–667. https://doi.org/10.1007/s00221-008-1579-2
- Mormann, F., Fell, J., Axmacher, N., Weber, B., Lehnertz, K., Elger, C. E., & Fernández, G. (2005). Phase/amplitude reset and theta-gamma interaction in the human medial temporal lobe during a continuous word recognition memory task. Hippocampus, 15(7), 890–900. https://doi.org/10.1002/hipo.20117
- Motomura, Y., Kitamura, S., Oba, K., Terasawa, Y., Enomoto, M., Katayose, Y., ... Mishima, K. (2013). Sleep debt elicits negative emotional reaction through diminished amygdala-anterior cingulate functional connectivity. *PLoS ONE, 8*(2). https://doi.org/10.1371/journal.pone.0056578
- Nitschke, J. B., Sarinopoulos, I., Oathes, D. J., Johnstone, T., Whalen, P. J., Davidson, R. J., & Kalin, N. H. (2009). Anticipatory activation in the amygdala and anterior cingulate in generalized anxiety disorder and prediction of treatment response. *The American Journal of Psychiatry*, 166(3), 302–310. https://doi.org/10.1176/appi.ajp.2008.07101682
- Nofzinger, E. A. (2005). Functional neuroimaging of sleep. Seminars in Neurology, 25(1), 9–18. https://doi.org/10.1055/s-2005-867070
- Paulus, M. P., & Stein, M. B. (2006). An insular view of anxiety. *Biological Psychiatry*, 60(4), 383–387. https://doi.org/10.1016/j.biopsych.2006.03.042
- Peper, E., Booiman, A., Lin, I.-M., & Harvey, R. (2016). Increase strength and mood with posture. *Biofeedback*, 44(2), 66–72. https://doi.org/10.5298/1081-5937-44.2.04
- Peper, E., Harvey, R., Mason, L., & Lin, I.-M. (2018). Do better in math: How your body posture may change stereotype threat response. *NeuroRegulation*, *5*(2), 67–74. https://doi.org/10.15540/nr.5.2.67
- Peper, E. & Lin, I.-M. (2012). Increase or decrease depression: How body postures influence your energy level. *Biofeedback*, 40(3), 125–130. https://doi.org/10.5298/1081-5937-40.3.01
- Peper, E., Lin, I.-M., Harvey, R., & Perez, J. (2017). How posture affects memory recall and mood. *Biofeedback*, 45(2), 36–41. https://doi.org/10.5298/1081-5937-45.2.01
- Phelps, E. A., Delgado, M. R., Nearing, K. I., & LeDoux, J. E. (2004). Extinction learning in humans: Role of the amygdala and vmPFC. *Neuron*, 43(6), 897–905. https://doi.org/10.1016 /j.neuron.2004.08.042
- Raghavachari, S., Kahana, M. J., Rizzuto, D. S., Caplan, J. B., Kirschen, M. P., Bourgeois, B., ... Lisman, J. E. (2001). Gating of human theta oscillations by a working memory task. *The*

- Journal of Neuroscience, 21(9), 3175–3183. https://doi.org/10.1523/JNEUROSCI.21-09-03175.2001
- Rauch, S. L., Whalen, P. J., Shin, L. M., McInerney, S. C., Macklin, M. L., Lasko, N. B., ... Pitman, R. K. (2000). Exaggerated amygdala response to masked facial stimuli in posttraumatic stress disorder: A functional MRI study. *Biological Psychiatry*, 47(9), 769–776. https://doi.org/10.1016/s0006-3223(00)00828-3
- Shin, L. M., Rauch, S. L., & Pitman, R. K. (2006). Amygdala, medial prefrontal cortex, and hippocampal function in PTSD. *Annals of the New York Academy of Sciences, 1071*(1), 67–79. https://doi.org/10.1196/annals.1364.007
- Siegle, G. J., Thompson, W., Carter, C. S., Steinhauer, S. R., & Thase, M. E. (2007). Increased amygdala and decreased dorsolateral prefrontal BOLD responses in unipolar depression: Related and independent features. *Biological Psychiatry*, 61(2), 198–209. https://doi.org/10.1016/j.biopsych.2006.05.048
- Spironelli, C., Busenello, J., & Angrilli, A. (2016). Supine posture inhibits cortical activity: Evidence from delta and alpha EEG bands. *Neuropsychologia*, 89, 125–131. https://doi.org /10.1016/j.neuropsychologia.2016.06.015
- Theta Waves. (n.d.). Retrieved from http://scottsdaleneurofeedback.com/services/qeeg-brain-mapping/eeg-brainwaves/theta-waves/
- Tsai, H.-Y., Peper, E., & Lin, I.-M. (2016). EEG patterns under positive/negative body postures and emotion recall tasks. NeuroRegulation, 3(1), 23–27. https://doi.org/10.15540/nr.3.1.23
- Van der Helm, E., & Walker, M. P. (2012). Sleep and affective brain regulation. *Social and Personality Psychology Compass*, 6(11), 773–791. https://doi.org/10.1111/j.1751-9004.2012.00464.x

- Vass, L. K., Copara, M. S., Seyal, M., Shahlaie, K., Farias, S. T., Shen, P. Y., & Ekstrom, A. D. (2016). Oscillations go the distance: Low-frequency human hippocampal oscillations code spatial distance in the absence of sensory cues during teleportation. *Neuron*, 89(6), 1180–1186. https://doi.org /10.1016/j.neuron.2016.01.045
- Walker, M. P. (2009). The role of sleep in cognition and emotion. Annals of the New York Academy of Sciences, 1156(1), 168–197. https://doi.org/10.1111/j.1749-6632.2009.04416.x
- Watrous, A. J., Fried, I., & Ekstrom, A. D. (2011). Behavioral correlates of human hippocampal delta and theta oscillations during navigation. *Journal of Neurophysiology*, 105(4), 1747– 1755. https://doi.org/10.1152/jn.00921.2010
- Wilson, V. E., & Peper, E. (2004). The effects of upright and slumped postures on the generation of positive and negative thoughts. *Applied Psychophysiology and Biofeedback, 29*(3), 189–195. https://doi.org/10.1023/B:APBI.0000039057.32963.34
- Woods, S. (1987). [Blood flow through the brain in healthy subjects as measured with SPECT]. Unpublished raw data. New Haven, CT: Yale University.
- Yoo, S.-S., Gujar, N., Hu, P., Jolesz, F. A., & Walker, M. P. (2007). The human emotional brain without sleep—a prefrontal amygdala disconnect. *Current Biology*, 17(20), 877–878. https://doi.org/10.1016/j.cub.2007.08.007
- Zhang, H., & Jacobs, J. (2015). Traveling theta waves in the human hippocampus. *The Journal of Neuroscience*, *35*(36), 12477–12487. https://doi.org/10.1523/jneurosci.5102-14.2015

Received: August 04, 2019 Accepted: September 24, 2019 Published: December 12, 2019