CORTISOL AND SLEEP: THE HPA AXIS ACTIVITY CONNECTION

The hypothalamic-pituitary-adrenal (HPA) axis interacts with sleep in a multiplicity of ways and a growing body of research suggests reciprocal associations between sleep and activity of the HPA axis.

Normal Sleep Physiology

Normal sleep architecture is characterized by cycles of light sleep, deeper slow-wave sleep and REM sleep. Light sleep includes stage 1 and stage 2 sleep. Stage 1 sleep has mixed frequency theta, slow rolling eye movements and slightly reduced eye movement and chin electromyography (EMG). Stage 2 has mixed frequency electroencephalogram (EEG). Deeper slow-wave sleep includes stages 3 and 4. Stage 3 sleep is characterized by 20-50% delta EEG and stage 4 has greater than 50% delta EEG. The rapid eye movement (REM) cycles have mixed frequency EEG with theta waves in combination with rapid eye movements and nearly absent chin EMG. REM occurs approximately every 90-110 minutes with a predominance of slow-wave sleep in the first half of the night and a predominance of REM sleep in the second half.[1] Corticotropin releasing hormone (CRH) is secreted by the hypothalamus, in particular the paraventricular nucleus (PVN), which acts on CRH receptors in the anterior pituitary to cause the release of adrenocorticotropic hormone (ACTH) into the blood. ACTH acts on the adrenal cortex which produces and releases cortisol. Along with its numerous actions in the body, cortisol has feedback inhibition on the PVN and the anterior pituitary to control CRH or synthesis and release of ACTH. Other areas of the brain also impart feedback to the HPA axis.

The circadian rhythm of cortisol secretion has a waveform pattern with the nadir for cortisol occurring at about midnight. Cortisol levels start to rise about 2-3 hours after sleep onset and continue to rise into the early morning and early waking hours. The peak in cortisol is about 9 a.m. and as the day continues, there is a gradual decline in levels. With the onset of sleep, there is a continued decline until the nadir. Throughout the cycle, there are pulsatile secretions of cortisol of various amplitudes. Cortisol binds to mineralocorticoid receptors (MRs) and glucocorticoid receptors (GRs) and feedback onto the PVN is excitatory or inhibitory depending on the location and type of receptor. Low levels of cortisol in the evening and night are associated with MR binding. When cortisol levels are higher, GRs are activated. In stressful times, GRs may be activated preferentially and thereby increases CRH. This elevated CRH increases sleep EEG frequency, decreases short wave sleep and increases light sleep and frequent waking.

Sleep, the HPA Axis, and Cortisol Rhythm

The initiation of sleep occurs concurrently with a low HPA axis activation and occasional sleeplessness is associated with HPA activation. Nighttime awakenings are associated with pulsatile cortisol release and are followed by a temporary inhibition of cortisol secretion. Cortisol begins its rapid rise after the first morning awakening and continues for about 60 minutes and is called the awakening response.

The HPA axis affects sleep and sleep affects the HPA. HPA axis activation can lead to fragmentation of sleep, decreased slow wave sleep, and a shortened sleep time. To complicate matters, these factors can worsen HPA axis balance. Mental health and circadian patterns are associated.[2] Elevated cortisol levels, particularly in the evening and the first part of the night

time sleep period, is of particular interest.[3,4,5,6] This elevated cortisol may be a marker for increased CRH activity or a marker for increased central norepinephrine.[7]

In summary, HPA axis activation can have a negative impact on sleep, leading to sleep fragmentation, decreased deep slow wave sleep, and shortened sleeping time. Interventions to support the HPA axis, decrease nocturnal CRHactivity, and decrease cortisol may be beneficial in supporting sleep.

Alternative Approach to the Hypercortisol-Induced Occasional Sleep Problems

An effective way to manage cortisol levels is to ensure that the adrenal glands are supported by the proper nutrients. Vitamin B6, pantothenic acid, and vitamin C, often become depleted when the demands on adrenal gland cortisol production are continuous.[8] These nutrients play a critical role in the optimal function of the adrenal gland and in the manufacture of adrenal hormones. L-tyrosine and L-theanine support the adrenal glands.[9] In addition, calcium, magnesium, potassium, manganese and zinc support the cortisol feedback control mechanism.[10]
Ashwagandha (W*ithania somnifera*), also known as Indian ginseng, has been shown to reduce corticosterone, a glucocorticoid hormone structurally similar to cortisol.[11,12] An array of clinical trials and laboratory research also support the use of ashwagandha in supporting mood and energy.[13,14,15,16]

Magnolia (*Magnolia officinalis*), was studied in a randomized, parallel, placebo controlled study in overweight premenopausal women although salivary cortisol levels were not significantly reduced.[17]Magnolia supports mood, sleep, and the normal stress response.[18]
Phosphatidyl serine (PS), also known as lecithin phosphatidylserine, is known to blunt the rise in cortisol and ACTH following strenuous training and significantly reduce both ACTH and cortisol levels after exposure to physical stress.[19,20]Phosphatidylserine also has been shown to improve mood.[21,22]

Many traditional botanicals have been utilized to support the HPA axis including American ginseng, Asian ginseng, astragalus, cordyceps, reishi, eleutherococcus, Holy basil, rhodiola, schisandra, maca, and licorice. Multi-ingredient formulations are common in a whole system approach to restoring HPA axis function, whether elevated or low cortisol levels.
Reducing cortisol levels and supporting the HPA axis can be a very effective approach to supporting sleep.

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