

TITLE:

Chronic insomnia is associated with nyctohemeral activation of the hypothalamic-pituitary-adrenal axis: clinical implications.

Remember, the word nyctohemeral in the title simply means "24 hours a day." The "hypothalamic-pituitary-adrenal axis" (or HPA axis) is the body's main stress-response system. So the title is saying that insomnia is associated with constant, 24-hour-a-day activation of the body's stress response system, and the research will look at the clinical implications of that fact.

TEXT:

Although insomnia is, by far, the most commonly encountered sleep disorder in medical practice, our knowledge in regard to its neurobiology and medical significance is limited. Activation of the hypothalamic-pituitary-adrenal axis leads to arousal and sleeplessness in animals and humans;...

An essential point: "Activation of the hypothalamic-pituitary-adrenal axis leads to arousal and sleeplessness in animals and humans." Whenever the body's stress-response system is activated, there will be increased arousal, that is, increased activation of mental and physical faculties. That is perfectly normal. However, when that stress-response is prolonged or unceasing, there is excess arousal, and that can lead to sleeplessness.

...however, there is a paucity of data regarding the activity of the hypothalamic-pituitary-adrenal axis in insomniacs.

"Paucity" is a lack or shortage. So this research is designed to fill in some of the blanks in our knowledge about the role of the HPA axis in sleep..

We hypothesized that chronic insomnia is associated with increased plasma levels of ACTH and cortisol.

That is the hypothesis, the thing they are going to try to confirm or disconfirm by experiment. ACTH and cortisol are both adrenal hormones that produce added arousal. We need these to adrenal hormones to remain alert and awake, and to maintain an appropriate level of arousal during waking hours. However, increased or excessive levels will produce excess arousal, and insomnia, according to the hypothesis.

Eleven young insomniacs (6 men and 5 women) and 13 healthy controls (9 men and 4 women) without sleep disturbances, matched for age and body mass index, were monitored in the sleep laboratory for 4 consecutive nights, whereas serial 24-h plasma measures of ACTH and cortisol were obtained during the fourth day.

This describes the way they did the experiment and collected their data.

Insomniacs, compared with controls, slept poorly (significantly higher sleep latency and wake during baseline nights).

Now they tell us the results of the experiment. "Higher sleep latency" means it took longer for insomniacs to fall asleep than a control group, and they had longer periods of wake during the night. No surprise there. That's what insomnia is all about.

The 24-h ACTH and cortisol secretions were significantly higher in insomniacs, compared with normal controls (4.2 +/- 0.3 vs. 3.3 +/- 0.3 pM, P = 0.04; and 218.0 +/- 11.0 vs. 190.4 +/- 8.3 nM, P = 0.07). Within the 24-h period, the greatest elevations were observed in the evening and first half of the night.

First significant finding: Insomniacs had higher levels of adrenal hormones ACTH and cortisol levels than controls, 24 hours a day. Greatest elevations were in the evening, and first half of the night.

Also, insomniacs with a high degree of objective sleep disturbance (% sleep time < 70), compared with those with a low degree of sleep disturbance, secreted a higher amount of cortisol. Pulsatile analysis revealed a significantly higher number of peaks per 24 h in insomniacs than in controls (P < 0.05), whereas cosinor analysis showed no differences in the temporal pattern of ACTH or cortisol secretion between insomniacs and controls.

Third finding: Insomniacs had a greater number of peaks of ACTH and cortisol secretion than controls over a 24-hour period. However, distribution of the peaks over time did not differ from control group—it retains a normal circadian pattern. Circadian means "over the course of a 24-hour day".

SUMMARY OF THE FINDINGS:

We conclude that insomnia is associated with an overall increase of ACTH and cortisol secretion, which, however, retains a normal circadian pattern.

In other words, ACTH and cortisol undergo the same cyclical rising and falling pattern over the course of a day and night in both insomniacs and normal sleepers, but levels are higher in insomniacs.

RESEARCHERS' CONCLUSIONS:

1. These findings are consistent with a disorder of central nervous system hyperarousal rather than one of sleep loss, which is usually associated with no change or decrease in cortisol secretion or a circadian disturbance.

Remember that phrase, "Central nervous system hyperarousal."

2. Chronic activation of the hypothalamic-pituitary-adrenal axis in insomnia suggests that insomniacs are at risk not only for mental disorders, i.e. chronic anxiety and depression, but also for significant medical morbidity associated with such activation.

This speaks for itself.

3. The therapeutic goal in insomnia should be to decrease the overall level of physiologic and emotional arousal, and not just to improve the nighttime sleep.

Ah-ha! That gives me an idea! In our sleep system, why don't we offer some exercises that "decrease the overall level of physiologic and emotional arousal" all day long, in addition to those that improve sleep at night. Can you think of any exercises that might be helpful for that purpose?

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