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Journal search and commentary

Article reviewed: Impact of sleep dept on metabolic and endocrine function ☆

Richard P. Allen

Johns Hopkins University, School of Medicine, Baltimore, MD, USA

Category

Sleep loss, metabolism and sleep

Objective

Evaluate the effects of chronic sleep loss on metabolic and endocrine function

Study design

Within subject evaluation before sleep loss, after 6 days of only 4 h sleep and after 6 days of recovery with 12 h sleep periods.

Study population

Eleven healthy males, ages 18–27 years.

Methods

Subjects had regulated sleep periods for 16 consecutive nights in a clinical research center: three at 8 h, six at 4 h and seven at 12 h for sleep. Sleepiness, sympathovagal balance (RR interval variability) and saliva free-cortisol concentrations were evaluated during each condition. Carbohydrate metabolism and hormonal profiles were evaluated only at the ends of the sleep loss and the sleep recovery conditions.

Results

Sleep loss (4 h sleep periods) compared to recovery (12 h sleep periods) condition showed significantly lower glucose tolerance ($P < 0.02$), decreased thyrotropin concentrations ($P < 0.01$), increased evening cortisol ($P < 0.001$), and increased sympathetic nervous system activity ($P < 0.02$). The 4- compared to the 8-h sleep condition showed subjective reports of greater sleepiness ($P < 0.01$), higher afternoon cortisol ($P < 0.03$) and marginally higher sympathetic activity ($P < 0.12$).

Conclusion

The chronic sleep loss had harmful effects on both carbohydrate metabolism and endocrine function. The authors also conclude that since similar changes occur with aging, sleep loss may exacerbate severity of some age-related chronic conditions.

Comment

This is a well-executed study and the findings are extremely important for sleep medicine. There are two very surprising aspects of this study. First, the

☆ Spiegel K, Leproult R, Van Canter E. Impact of sleep debt on metabolic and endocrine function. Lancet 1999;354:1435–1439.

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adverse effects with only 6 days of sleep loss were very significant. For example the rate of glucose clearance after injection was reduced by 40%, glucose effectiveness was reduced by 30% and the insulin response to glucose was 30% lower. The latter is an early marker of diabetes and these changes are the same magnitudes as those reported for aging and gestational diabetes. Despite no differences in the insulin secretory responses the peak glucose response to the breakfast was dramatically higher consistent with a diagnosis of impaired glucose tolerance.

Similarly the 24-h thyrotropin levels were reduced by about 35% and the usual nocturnal increase was virtually abolished while the free thyroxine index was significantly increased suggesting significant effects on thyroid-hormone concentrations. The 0900–1400 h RR variability measure of sympathovagal activity was also significantly increased by about 17% ($P < 0.02$). Moreover, not only were quiescent times for cortisol secretion reduced by about 15% ($P < 0.03$) but the evening and late afternoon levels were significantly increased ($P < 0.00l$) with values at some time points showing 100% increases for the sleep loss condition. If these effects occur in people prone to diabetes or hypertension it seems likely the chronic sleep loss could significantly exacerbate or even precipitate the development of their disorder.

Clearly this study needs to be replicated and the effects of degree and duration of sleep loss examined. The authors propose three pathways for the CNS effects of sleep loss to effect peripheral function: (1) Increases in sympathetic vs. parasympathetic tone may adversely affect cardiovascular and kidney function. (2) Decreased cerebral use of glucose may lead to chronic peripheral exposure to higher glucose levels. (3) Disruption of the hippocampal negative feedback regulation of the hypothalamo-pituitary-adrenal axis. These certainly need further evaluation. Possible phase delay or desynchrony affecting insulin response might also explain some of these results, particularly the abnormal insulin response that occurred only for breakfast and not for later meals.

The second surprising aspect of this study is why it has taken this long for the field of sleep medicine to address the effects of chronic sleep loss on health and in particular on peripheral metabolic and endocrine functioning. The shift work literature includes several studies suggesting adverse health effects presumably related in part to chronic sleep loss, which the authors probably could have noted in this article, but otherwise this has been a neglected area of study. There has also been some work relating insulin resistance to sleep apnea, which should probably be reconsidered in light of these findings. Sleep apnea and other sleep disorders with chronic loss of adequate sleep may produce conditions where these effects of chronic sleep loss might occur. As the authors suggest, the field of sleep medicine must recognize that sleep is not only for the brain, but also for the rest of the body. Sleep loss affects brain and peripheral function.