Increased Morning Cortisol Level

Effect of Sleep Fragmentation or Stress Response to the Last Annoying Stimulus?

To the Editor:

Fragmented sleep has great effects on daily life, including sleepiness, impaired cognitive function, decreased mood, and elevated BP.1-3 In an excellent study in a recent issue of CHEST (January 2010), Stamatakis and Punjabi1 show that two nights of nonspecific sleep fragmentation in healthy volunteers led to decreased insulin sensitivity and glucose effectiveness despite normal sleep duration. This study helps to elucidate the influence of sleep fragmentation per se on glucose metabolism without the confounding effect of sleep duration or hypnotic insult, as seen in the case of obstructive sleep apnea. The authors concluded that increased sympathetic activity and adrenocortical activity likely mediate the adverse effects of poor sleep quality, based on the finding of increased morning cortisol levels and increased sympathetic activity.

They used auditory and mechanical stimuli to elicit EEG microarousals with a frequency of ≥ 30 events/h. If the stimuli failed, the subsequent stimulus got larger by increasing the tone volume of auditory stimuli or combining the two kinds of stimuli. In this study, most of the stimuli were effective, including the last one at the end of the sleep period, which was probably the largest stimulus in intensity. When a human is exposed to noxious or potentially noxious stimuli, there is an increased secretion of corticotropin and, consequently, a rise in the circulating cortisol. Approximately 90% to 95% of the cortisol in the plasma binds to plasma proteins, which slows the elimination of cortisol from the plasma. Therefore, cortisol has a relatively long half-life of 60 to 90 min and thus a lasting action.4 An increased morning cortisol level (measured at 8:00 AM in this study) may be the consequence of the last effective stimulus, elicited 1 or 2 h prior to measurement, rather than that of sleep fragmentation. As the authors stated, elevations of cortisol, even within the normal physiologic range, can decrease insulin sensitivity, enhance hepatic gluconeogenesis, and inhibit insulin secretion.5 To settle this dispute, one additional night of nonfragmented sleep following the two nights of fragmented sleep is needed as a sleep-recovery period, as stated in some sleep deprivation studies.6-8 In addition, one single stimulus is given near the end of the night, which is of the same intensity and timing as the last stimulus in the previous night of sleep fragmentation. If an elevated cortisol level is still observed in the coming morning (day 5), the disturbed glucose metabolism may not be attributed to the effect of sleep fragmentation.

Besides, evening cortisol concentration following sleep deprivation was raised in sleep deprivation studies, reflecting an impairment of negative feedback control of the hypothalamic-pituitary-adrenal axis.9,10 This finding was not observed in this study, suggestive of a different mechanism underlying sleep deprivation and sleep fragmentation, or just an unequal stress in intensity, which deserves further exploring.

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REFERENCES


Quantifying Latent TB Infection

The Dilemma Continues

To the Editor:

The study in the May 2010 issue of CHEST by Gallant et al,1 whereby they simultaneously compared the quantitative results of in vivo and in vitro assays for TB infection generates a lot of interest. There are a couple of issues that need to be addressed in this study:

The authors have used Bacillus Calmette-Guérin (BCG), purified protein derivative (PPD), and early-secreted antigenic target-6 (ESAT-6) as the triggers for the release of interferon (IFN)-γ in the in vitro assays. The authors have justified the use of PPD in both the in vitro and in vivo assays because it may avoid the antigen specificity as the confounding factor, but the use of BCG as an in vitro trigger remains questionable because the BCG status of the population in question is not known.

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