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REFERENCES


Sleep restriction and appetite control: waking to a problem?

Dear Sir:

In the December issue of the Journal, Schmid et al (1) reported that short-term sleep restriction did not increase spontaneous food intake under laboratory conditions in healthy men. In addition, short-term sleep restriction affected neither appetite sensations nor the circulating concentrations of leptin and ghrelin. These findings contradicted their initial hypothesis and the seminal study by Spiegel et al (2), who elegantly showed that 2 consecutive nights of sleep restriction to 4 h instead of 10 h induced an 18% decrease in the circulating concentrations of the anorexigenic hormone leptin and a 28% increase in concentrations of the orexigenic hormone ghrelin in conjunction with increased sensation of hunger and appetite. Furthermore, these findings were not concordant with the growing body of evidence showing that sleep restriction alters neuroendocrine hormones and appetite in a way that could favor a positive energy balance and potential weight gain in the long run (3–8).

Although the authors tried to explain this discrepancy, an important flaw of the study design that was not mentioned in the article should be highlighted. This pertains to the fact that the participants were overfed at the time during which appetite was assessed, and blood samples were collected for the analysis of appetite-related hormones. Indeed, on the whole, the subjects ingested a rather high amount of energy that on average exceeded their estimated daily energy demand by ~60% in both study conditions. We believe that such an excess caloric intake alone may explain the absence of difference in leptin and ghrelin concentrations and consequently in appetite sensations. The pattern of response in these 2 hormones (leptin was increased and ghrelin was suppressed without showing the typical diurnal variations) is also concordant with this state of overfeeding. This collection of data contrasts with the study of Spiegel et al (2), in which men were subjected to mild caloric restriction in the form of intravenous glucose infusion. Accordingly, these results must be interpreted with caution, and the effects of sleep restriction on appetite control cannot be adequately determined on the basis of this study.

The study results also emphasize the difficulty of assessing in an experimental context the influence of short sleep duration on energy balance. The mismatch between energy input and output as a result of short sleeping will probably be more accurately determined under free-living conditions and chronic short sleep duration (as opposed to an acute manipulation in a laboratory setting). The propensity of many individuals to overeat in the setting of sedentary living with unlimited food availability also suggests that the nonhomeostatic feeding behavior—ie, eating in the absence of hunger—could play an important role. This concept is supported by recent data showing that recurrent bedtime restriction under free-living conditions did not down-regulate the satiety hormone leptin nor up-regulate the appetite-stimulating hormone ghrelin but increased intake of calories from snacks (9). Likewise, a study carried out in 1985 observed that habitual short-sleepers (average of 6 h/night) ate proportionally more often (ie, >3 meals/d with more frequent nibbling) than did long-sleepers (10).

Future studies will thus have to differentiate the variations in energy intake associated with short sleep duration between 1) the increased time and opportunities to eat (due to extra waking hours) as well as eating as a result of cues other than those that are appetite related and 2) the homeostatic regulation of feeding (hormonal signals that increase appetite). With the advent of functional magnetic resonance imaging, the documentation of food-related reward activation in the brain after sleep restriction will open new research avenues. Moreover, future experimental studies that examine the influence of objectively measured restricted sleep on both sides of the energy balance equation should focus more on children and adolescents to build up the cause-and-effect evidence between short sleep duration and obesity. No interventional study to date has tested the effects of sleep loss on energy balance in children/adolescents despite the fact that the relation between short sleep duration and obesity is more robust in this population. Future studies should also address the question of whether increasing sleep time in sleep-deprived obese individuals will reduce the amount of body fat or influence the concentration of hormones that help to control appetite. Whether people can voluntarily change their sleeping hours is also unknown; therefore, the causes of sleep curtailment should be investigated. If most of the extra waking hours take place at the end of the day, future studies should document late-night snacking. This will be especially important if most of wakefulness is spent in sedentary activities, such as watching television, in which snacking is common.
LETTERS TO THE EDITOR

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Reply to J-P Chaput et al

Dear Sir:

We thank Chaput et al for their letter regarding our study (1), which investigates possible mechanisms behind the association between short sleep duration and obesity that is inferred from epidemiologic studies (2). Our results of unchanged food intake and comparable ghrelin and leptin concentrations after acute sleep restriction at first glance do not support the currently widely discussed hypothesis that links short sleep duration to increased food intake and, in consequence, to body weight gain. Although we fully agree with Chaput et al that further experimental studies are needed to draw any definitive conclusions on the matter, we do believe that our study has yielded important results that may even help to develop future studies such as those outlined by Chaput et al. A primary target of these studies should be the nonhomeostatic, reward-driven regulation of eating behavior that could be affected by sleep restriction.

There is growing evidence that human eating behavior apart from the neuroendocrine homeostatic pathways of brainstem and hypothalamus (3) is profoundly influenced by signals that are related to the hedonic value of food intake and that are processed in higher brain centers such as mesolimbic areas (4) and parts of the prefrontal cortex (5). These areas project with a high neuronal density to hypothalamic nuclei where neuroendocrine-homeostatic and hedonic signals are integrated. Sleep regulation is well known to interact with reward processing, an effect that is put to clinical use in the treatment of depression. The orexin/hypocretin system may be of particular relevance in this context inasmuch as it is critically involved in both sleep-wake regulation and reward processing (6). Human behavioral data provide first evidence for an enhancing influence of sleep loss on hedonically driven eating behavior. Thus, a recent study by Nedeltcheva et al (7) showed an increased consumption of snacks in subjects who spent 14 d with bedtime restriction to 5.5 h/d in a sleep laboratory with ad libitum food intake. Of note, increased snacking occurred in the absence of any alterations in circulating ghrelin and leptin concentrations, that likewise remained unaffected by sleep loss in our study, which suggests hedonic rather than homeostatic mediators of the shift in food selection. Although the laboratory setting of ad libitum food intake in our study was comparable to that of the foregoing study (7), we did not observe a similar effect on food consumption apart from a slightly increased intake of fat. In both studies, subjects displayed food intake that clearly exceeded their energy demand, consuming ∼3000 kcal/d in our study and 3600 kcal/d in the previous study (7), an effect most probably due to continuous access to a broad variety of palatable foods. However, whereas our subjects were monitored for one full day, the participants of the previous study spent 14 d in the laboratory. It seems reasonable to assume that over such an extended laboratory session, subjects adapt to the stimulatory influence of readily available, highly palatable foods. In support of this view, in the study by Nedeltcheva et al (7), the order of conditions turned out to be a significant factor to influence energy intake and body weight changes, leading the authors to conclude that “the excessive consumption of calories from meals and snacks was augmented by the novelty of the experimental environment and emerged as an important predictor of individual weight gain” (p 132). Nevertheless, sleep loss significantly increased snack intake, which suggests that sleep restriction prevents the adaptation to palatable food cues that have a strong hedonic component.

Importantly, in our study, the time periods when food could be consumed by the subjects were strictly paralleled—ie, no food intake was permitted during sleep restriction. In contrast, in the study by Nedeltcheva et al (7), subjects were allowed to eat when sleep restriction was implemented at nighttime. Interestingly, the difference in snack intake between the sleep loss and regular sleep conditions reached significance only during nighttime but not during daytime, indicating that the timing of sleep restriction strongly influences its effect on calorie intake.