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Reply to L Bennedsen et al

Dear Sir:

We thank Bennedsen et al for their letter regarding our study (1), which explains possible mechanisms behind the association between short sleep duration and obesity in male teenagers. We agree that our results of increased energy expenditure (EE) and decreased drive to eat combined with decreased spontaneous energy intake are not supportive of the current evidence in the field, especially in younger populations (2). Bennedsen et al in their letter raise some important methodologic considerations in this regard.

We found that the difference in EE between sleep conditions (ie, 4 h compared with 8 h of sleep/night) was fully explained by the prolonged waking time in the short sleep condition. We agree that there are conflicting results regarding the impact of sleep restriction on EE, but a total sleep-deprivation protocol was applied in the study by Jung et al (3) as well as in the study by Benedict et al (4). This crucial methodologic difference prevents any direct comparison with our study. Also, the study by Benedict et al (4) did not measure 24-h EE. In the study by Bosy-Westphal et al (5), individual regression lines of VO2 compared with heart rate were used to calculate total EE. This method to determine 24-h EE under mainly sedentary conditions is far from optimal (6). Thus, in line with the results from our study we do not believe that acute partial sleep restriction brings about intrinsic physiologic changes in EE, which has also been reviewed by our group previously (7). Future studies will nevertheless be needed to confirm this observation, with careful attention to the sleep-restriction protocol and methods used.

We are well aware that pretrial sleep habits or even sleep-related conditions such as obstructive sleep apnea syndrome (OSAS) can differ between and within individuals and thus potentially interfere with the results. We also acknowledge the fact that OSAS can affect children and teenagers without either the children or their parents being aware of this. Consequently, we did submit the subjects to polysomnographic assessment the 2 nights before the 24-h measurement of EE. Furthermore, we evaluated pretrial sleeping habits by using the Pittsburgh Sleep Quality Index and also questioned the participants about potential sleep-related illnesses. Neither screening assessment of sleep nor polysomnographic measurements raised ground for concern regarding pretrial sleeping habits or OSAS among these lean and healthy boys. Furthermore, we conducted a crossover trial to limit the effects of any between-subject differences.

In the present study we confined the subjects in respiration chambers and strictly controlled their food intake to be able to detect subtle physiologic effects on energy metabolism. Bennedsen et al comment on the discrepancy between assessing energy balance in this controlled setting compared with assessing energy balance under free-living conditions. We agree that a controlled setting does not translate to real-life conditions; however, experimental studies are more robust than observational studies and are instrumental in understanding mechanisms under standardized conditions. We agree that food intake as well as physical activities were both dictated by protocol and restricted by the chamber and, as such, not comparable to free-living conditions. However, we do believe that this approach is suitable for the purpose of the study and can answer critical questions and provide an interesting addition to the current literature.

We also acknowledge that inducing sleep restriction for a few days does not mimic the mild chronic sleep deprivation in habitual short sleepers. The aim of the present study was to evaluate potential mechanisms involved in the sleep-obesity relation, and this requires strictly controlled conditions, which, for obvious reasons, cannot be carried out for long durations. Nevertheless, Bennedsen et al raise an important question as to whether the putative causes of the sleep-obesity association are related to behavioral aspects rather than intrinsic physiologic changes. Strictly controlled trials are not suitable for addressing behavioral aspects but are necessary to evaluate potential mechanisms. Future studies that use a different approach will have to be conducted to elucidate the question as to whether changes in food choice or timing of food intake (eg, late-night snacking) after sleep restriction would cause alternate effects in energy balance and thus help explain the gap between experimental studies and the unequivocal epidemiologic evidence.

Although we agree with Bennedsen et al that further experimental studies are needed to draw any definitive conclusions on the behavioral aspects potentially underpinning the sleep-obesity relation, we do believe that our study has provided important findings that may even help to develop future studies such as those outlined by Bennedsen et al. In our opinion, a primary target of future studies should be the nonhomeostatic, reward-driven regulation of eating behavior that could be affected by sleep restriction. A better characterization of short sleepers will also be needed, because it appears that only some of these individuals are likely to overeat in response to sleep restriction.

The authors declared no conflicts of interest.

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Dairy food and body fat: when the epidemiologist meets the physiologist

Dear Sir:

The first documentation of a relation between calcium intake and body weight in humans was done in the 1980s when McCarron et al (1) reported a low calcium intake in individuals classified in the upper centiles of the studied population for BMI. However, the study of this issue had its real beginning when Zemel et al (2) published an article reporting animal and human data that showed some benefits of dairy food on body fat via a mechanism involving calcium homeostasis. The hypothesis relating calcium/dairy food to the regulation of energy and fat balance has received subsequent support from physiologic studies that showed an effect of calcium or dairy food on each component of fat balance, ie, fat oxidation (3), intestinal fat excretion (4), and fat intake (5).

Numerous intervention studies have also been implemented to verify if the supplementation of dairy food can influence body weight and fat. They have been collectively reanalyzed by Chen et al (6) who reported in a recent issue of the Journal a meta-analysis of 29 controlled trials. Obes Rev 2009;10:475–86. Numerous intervention studies have also been implemented to verify if the supplementation of dairy food can influence body weight and fat. They have been collectively reanalyzed by Chen et al (6) who reported in a recent issue of the Journal a meta-analysis of 29 controlled trials. Obes Rev 2009;10:475–86.

The absence of difference in body weight and fat loss between long-term nonrestricted dairy- and non–dairy-supplemented studies may give the impression that there is no detectable effect of dairy food in this context. In addition to the documentation of this issue, the authors also presented the most realistic explanation of this result: “In addition, an extra dairy intake in ad libitum dietary intervention might have led to an increased energy intake, which would have resulted in weight gains or have offset the protective effect of the dairy intervention” (6). For example, they cited the study by Barr (8) in which participants assigned to the dairy intervention group had an energy intake that exceeded by ~100 kcal/d their baseline intake, whereas such an increase was not seen in the control group. When this finding is compared with the estimated additional energy deficit observed in the short-term trials considered in the meta-analysis, it is clear that such an a priori increase in energy intake constitutes an experimental handicap that is quantitatively sufficient to entirely compensate for the expected benefits of dairy food. In this context, the fact that weight changes were comparable between groups despite this experimental manipulation may also be perceived as a favorable effect of dairy food. After all, the prevention of an expected weight gain and the accentuation of an expected weight loss have in common the increase in the importance of energy expenditure/fat oxidation relative to energy and fat intake.

In summary, the study by Chen et al (6) suggests that “when the epidemiologist meets the physiologist,” this may increase the likelihood to deduce realistic implications in a free-living context from what is objectively shown in the laboratory. In this case, the results confirm that the contribution of dairy food to diet-induced weight loss is significant but should be a matter of reasonable expectations. In fact, regular dairy consumption appears once again to be an important part of a healthy lifestyle that facilitates the regulation of energy/fat balance and that can make a difference in variations of body fat over time.

There were no conflicts of interest to declare.

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