Eating and sleeping—their relationship to ghrelin and leptin

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EATING AND SLEEPING are two kinds of behavior that are essential for the survival of humans and higher animals. Whereas it is obviously excluded that they occur exactly at the same time, there appear to be common regulators of both phenomena. With the identification of ghrelin as the endogenous ligand of the growth hormone (GH)-secretagogue (GHS) receptor by Kojima et al. (9) a new endogenous regulator of food intake and possibly also of sleep was found. In this issue of the American Journal of Physiology-Regulatory, Integrative and Comparative Physiology, Bodosi et al. (3) report a sophisticated study on the relationship between sleep, feeding, ghrelin, and its antagonist in the energy balance, leptin.

The detection of ghrelin was preceded in the 1970s by the synthesis of GHSs and by the cloning of the GHS receptor (12). Although they act on a different receptor, the GHSs and ghrelin share the capacity of GH-releasing hormone (GHRH) to stimulate GH. In addition to this endocrine effect, GHRH stimulates non-rapid eye movement (REM) sleep in various species including humans (11). Similarly sleep-promoting effects of synthetic GHS were found in humans (4, 8). Furthermore, some hints exist for a stimulating influence of GHS on food intake and body weight (6).

Soon after the identification of ghrelin, which is displayed mainly in the stomach and also in other tissues including the hypothalamus, it became clear that it is the most powerful endogenous orexigenic factor known so far. Ghrelin stimulates food intake and conserves fat, resulting in increasing body weight in rodents. Similarly, appetite and caloric intake increased after ghrelin administration in humans. Ghrelin levels were found to be changed in eating disorders, with high concentrations in anorexia and Prader-Willi Syndrome and blunting in obesity (for review, see 14). In contrast to ghrelin, leptin is an orexigenic factor and it is thought that ghrelin and leptin regulate the energy balance in a reciprocal fashion.

Similarly to GHRH, non-REM sleep was enhanced after ghrelin in mice (10) and humans (15). Intact GHRH receptors were shown to be the prerequisite for this effect in mice (10). Also, an effect of leptin on sleep was reported (13). Bodosi et al. (3) compared plasma ghrelin and leptin levels and hypothalamic ghrelin contents, sleep, brain temperature, and feeding throughout the dark-light cycle in rats in three experimental conditions: free-feeding animals with normal diurnal rhythms, restricted feeding, and sleep deprivation. From their findings they conclude that intimate relations between feeding and plasma ghrelin and leptin are corroborated, whereas there are no strong links between sleep and these hormones in the rat. These study results are a challenge to the searches for the answers to new questions.

REFERENCES

6. Dickson SL and Luckman SM. Induction of c-fos messenger ribonucleic acid in neuropeptide Y and growth hormone (GH)-releasing factor neurons in the rat arcuate nucleus following systemic injection


