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Chronic sleep fragmentation promotes obesity in young adult mice.

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Source

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Abstract

Objectives: Short sleep confers a higher risk of obesity in humans. Restricted sleep increases appetite, promotes higher calorie intake from fat and carbohydrate sources, and induces insulin resistance. However, the effects of fragmented sleep (SF), such as occurs in sleep apnea, on body weight, metabolic rates, and adipose tissue distribution are unknown. Design and Methods: C57BL/6 mice were exposed to SF for 8 weeks. Their body weight, food consumption, and metabolic expenditure were monitored over time, and their plasma leptin levels measured after exposure to SF for 1 day as well as for 2 weeks. In addition, adipose tissue distribution was assessed at the end of the SF exposure using MRI techniques. Results: Chronic SF induced obesogenic behaviors and increased weight gain in mice by promoting increased caloric intake without changing caloric expenditure. Plasma leptin levels initially decreased and subsequently increased. Furthermore, increases in both visceral and subcutaneous adipose tissue volumes occurred. Conclusions: These results suggest that SF, a frequent occurrence in many disorders and more specifically in sleep apnea, is a potent inducer of obesity via activation of obesogenic behaviors and possibly leptin resistance, in the absence of global changes in energy expenditure.