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CLINICAL REVIEW

The metabolic consequences of sleep deprivation

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Summary The prevalence of diabetes and obesity is increasing at an alarming rate worldwide, and the causes of this pandemic are not fully understood. Chronic sleep curtailment is a behavior that has developed over the past 2–3 decades. Laboratory and epidemiological studies suggest that sleep loss may play a role in the increased prevalence of diabetes and/or obesity. Current data suggest the relationship between sleep restriction, weight gain and diabetes risk may involve at least three pathways: (1) alterations in glucose metabolism; (2) upregulation of appetite; and (3) decreased energy expenditure. The present article reviews the current evidence in support of these three mechanisms that might link short sleep and increased obesity and diabetes risk.

Introduction

The prevalence of obesity and type 2 diabetes is increasing worldwide but particularly in the US.[1] Obesity and diabetes are both associated with increased age-adjusted mortality risk as well as a substantial economic burden.[2] The causes of this pandemic are not fully explained by changes in traditional lifestyle factors such as diet and physical activity. One behavior that seems to have developed during the past few decades and has become highly prevalent, particularly amongst Americans, is sleep curtailment. In 1960, a survey study conducted by the American Cancer Society found modal sleep duration to be 8.0–8.9 h,[3] while in 1995 the modal category of the survey conducted by the National Sleep Foundation poll had dropped to 7 h.[4] Recent analyses of national data indicate that a greater percentage of adult Americans report sleeping 6 h or less in 2004 than in 1985.[5] Today, more than 30% of adult men and women between the ages of 30 and 64 years report sleeping less than 6 h/night.[5] The decrease in average sleep duration in the US has occurred over the same time period as the increase in the prevalence of obesity and diabetes.

The present review examines the existing evidence for a link between short sleep and increased

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risk of obesity and diabetes and explores putative causal mechanisms. By "short sleep", we mean sleep durations under 7 h/night. There is substantial evidence in support of an association between long sleep (>8 h) and increased morbidity and mortality,[6–8] but the mechanisms linking long sleep and poor health are likely to be distinct from those mediating the adverse effects of short sleep.

Figure 1 provides a schematic representation of the three major pathways that could mediate an adverse effect of sleep loss on the risk of obesity and diabetes. Obesity is in itself a major risk factor for type 2 diabetes but recent data indicate that short sleep may impair glucose metabolism and increase the risk of diabetes independently of changes in body mass index (BMI). Sleep restriction may affect energy balance and result in weight gain because of an upregulation of appetite, more time to eat and a decrease in energy expenditure. Significant weight gain may in turn result in insulin resistance, a condition that increases the risk of developing diabetes and may promote further adiposity. This cascade of negative events is likely to be accelerated in many overweight and obese individuals by sleep-disordered breathing (SDB), a reported independent risk factor for insulin resistance.[9,10] The present article will only focus on sleep loss resulting from behavioral sleep restriction rather than from the presence of a sleep disorder.

We will first review the experimental and epidemiologic evidence for an association between short sleep, alterations in glucose metabolism and increased diabetes risk. As a cautionary note, translating the effects of experimental sleep restriction in the laboratory to the real world is not straightforward. Furthermore, laboratory studies of sleep restriction cannot be conducted for periods of time extending beyond 1–2 weeks. Epidemiologic studies that involve population-based samples often do not provide evidence for causal direction. In many such studies, the effects of sleep loss cannot be distinguished from effects of sleep disturbances. A presentation of the evidence linking short sleep, upregulation of appetite and increased BMI will follow. Finally, we will address the possibility that individuals exposed to insufficient sleep and the resulting sleepiness and fatigue may also have lower levels of energy expenditure than well-rested adults, particularly in an environment that promotes physical inactivity.

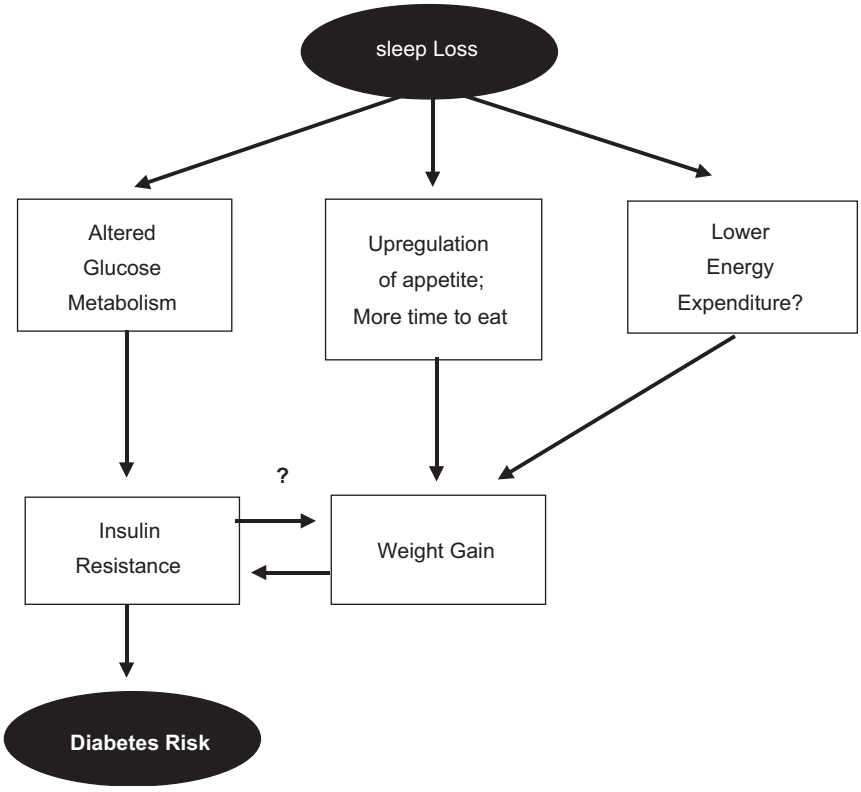

Figure 1 Schematic of the potential pathways leading from sleep loss to diabetes risk.