

The Centers for Disease Control and Prevention (CDC) data collected with the National Health Interview Survey (NHIS) from 2004-2006 demonstrates that an estimated 70 million Americans are negatively affected by chronic sleep loss or sleep disorders.

The data collected was analyzed for the prevalence of smoking, alcohol use, physical inactivity, and obesity as they relate to usual sleep duration. Furthermore, the data was stratified by gender, age, and race/ethnicity. The goal of the study was “to identify variations in prevalence of these health risk behaviors by usual sleep duration and to identify subgroups for which these associations may be particularly noteworthy.” Although determinations of causality cannot be inferred from correlation studies, conclusions can be drawn about the possible clustering of behaviors that result in negative health outcomes.

For both genders, smoking prevalence was substantially higher in those who slept less than six hours; and especially among younger age groups. Adults 18-44, sleeping less than 6 hours were 38% more likely to be current smokers than same aged adults who slept 7-8 hours per night.

Similarly, the data found that men of all ages who slept less than 6 hours per night were 31% more likely to also consume five (5) alcohol drinks in one day. The likelihood was slightly higher for the 18-44 demographic. Women’s results were not significantly different for those who slept little and those who slept adequately.

Interestingly, physical inactivity rates were highest for both genders when individuals slept either less than 6 hours, or 9 hours or more. This was independent of race or ethnicity. Leisure-time inactivity rates for non-Hispanic white adults who slept less than 6 hours were about 42%, and 45% for those who slept 9 hours or more. Non-Hispanic black adults who slept 9 hours or more had 58% rates of inactivity and those who slept less than 6 hours experienced inactivity rates of 49%.

As far as obesity is concerned, one in four adults studied was found to be obese. Adults who slept less than 6 hours were characterized by a 33% obesity rate, while those who slept 7-8 hours were characterized by a 22% rate. Fortunately, research has gone into elucidating some of the underlying physiological mechanisms that clarify why a lack or surplus of sleep may manifest changes in hunger patterns and alter body composition.

Sleep patterns and hunger mechanisms are interrelated. Several studies over the past few years have studied the relationship between sleep and eating patterns. This knowledge has become increasingly relevant because over the past few decades, an increase in industrialization has been accompanied by a decrease in sleep hours and, coincidentally, an increase in obesity.

Independent studies at the University of Chicago, Stanford University, and the University of Toronto have demonstrated that experimental sleep restriction is associated with an adverse impact on glucose homeostasis, insulin sensitivity, and neuroendocrine control of appetite.

The major endocrine indices focused on by these studies were leptin, ghrelin, adiponectin, orexins, and neuropeptide  $\gamma$  (NPY). NPY is a neurotransmitter found in the brain that stimulates food intake (1). Even small doses injected into the hypothalamus have been found to stimulate feeding in animal studies. Ghrelin is a hormone produced mainly by cells lining the human stomach and epsilon cells of the pancreas that stimulates appetite. It is essentially antagonistic to the hormone leptin. Leptin, released from adipocytes, inhibits neurons containing NPY - essentially inhibiting appetite. It is known as the “satiety” hormone. Obese individuals are characterized by chronic high serum levels of leptin resulting in receptor down-regulation - a decreased sensitivity similar to Type 2 Diabetic insulin-resistance. This decreases “satiation” likely perpetuating their obesity. Furthermore, the visceral fat located around the organs is dysfunctional in regards to its release of leptin. It releases very little leptin which may also be a mechanism perpetuating appetite and further weight gain. Combined, high levels of visceral and subcutaneous adiposity can result in a dysfunctional hunger mechanism characterized by unusual highs and lows with regard to levels of leptin. Adiponectin binds cellular receptors increasing AMP kinase activity which, among other actions, increases fatty acid oxidation and decreases cholesterol synthesis. It also has a similar yet additive effect on NPY neurons as leptin. Finally, orexins promote both wakefulness and hunger. Orexin-secreting cells are inhibited by leptin.

Epidemiologic studies in both children and adults demonstrate consistently that a lack of sleep (characterized as 5 hours vs. an 8 hour control group) increases the risk of diabetes. Sleep curtailment is also associated with neuroendocrine control of appetite dysfunction. Serum leptin and ghrelin levels are markedly decreased and increased respectively in sleep deprived groups. One study found that there was a 15.5% lower leptin level and 14.9% higher ghrelin level in 5 hour sleep groups vs. 8 hour sleep groups (2).

In this way, chronic sleep deprivation alters the ability of leptin and ghrelin to accurately signal caloric need. This may contribute to an inaccurate perception of caloric requirements and often leads to overeating. This pattern can lead to chronic hyperphagia (overeating) and actually create a negative feedback loop that culminates in obesity. Multiple epidemiologic studies have shown an association between short sleep periods and higher body mass index after controlling for a variety of possible confounders (1).

In conclusion, maintaining a healthy sleep schedule is vital in maintaining not only psychological health, but physiological well-being as well. Sleep is intimately tied to hunger-mechanisms and therefore dietary balance. The general recommendation seems to be 8 hours of sleep on a consistent day-to-day cycle to maintain circadian rhythms and neurological control of perceived caloric need. The data for the NHIS from the CDC can be found at [www.cdc.gov/nchs/pubs/pubd/hestats/sleep04-06/sleep04-06.htm](http://www.cdc.gov/nchs/pubs/pubd/hestats/sleep04-06/sleep04-06.htm)

**References**

1. Molecular characterization of the ligand-receptor interaction of the neuropeptide Y family.

Cabrele C, Beck-Sickinger AG.

Department of Pharmacy, ETH Zurich, Switzerland.

2. Short sleep duration is associated with reduced leptin, elevated ghrelin, and increased body mass index.

Taheri S, Lin L, Austin D, Young T, Mignot E.

Howard Hughes Medical Institute, Stanford University, Palo Alto, California, USA.

3. Hypocretin/Orexin: a molecular link between sleep, energy regulation, and pleasure.

Ganjavi H, Shapiro CM.

University of Toronto, Toronto, Canada.