In America’s fast-paced society, sleep is sometimes viewed as a lazy luxury; however, recent research shows that sleep is more than a passive time sink. Accumulating research indicates that long-term sleep loss is associated with the development of obesity and diabetes. Moreover, several experimental sleep loss studies show that total and partial sleep deprivation may modulate hormone secretions that impact glucose utilization and insulin sensitivity, appetite regulation, and resting energy expenditure. In addition to insulin, circulating leptin, ghrelin, thyroid-stimulating hormone, and cortisol levels are influenced by sleep patterns. Collectively, hormone manipulations observed with sleep loss may predispose individuals to obesity and type 2 diabetes.

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Several groups in the American population, including college students, swing-shift workers, and parents of small children, struggle to get regular or adequate sleep. The National Sleep Foundation recommends 8 hours of sleep per night. According to the Centers for Disease Control and Prevention, however, the percentage of Americans who slept 6 hours or less each night from 1985 to 2006 increased significantly (Centers for Disease Control, unpublished estimates from the 2006 National Health Interview Survey; 2008). Furthermore, greater than 30% of Americans aged 30 to 64 years slept less than 6 hours per night in 2006 (Centers for Disease Control, unpublished estimates from the 2006 National Health Interview Survey; 2008). Given the prevalence of chronic sleep loss in America, it is troubling that many studies provide evidence that acute and chronic sleep loss may initiate hormonal irregularities.

In the advanced stages of type 2 diabetes, insulin production suffers as a result of pancreatic damage, and the afflicted person may require insulin injections to clear BG adequately.

To assess the impact of a 24-hour sleep deprivation period on IR, one group of researchers used the insulin suppression test. The insulin suppression test typically requires a continuous infusion of epinephrine, propranolol, insulin, and glucose. Epinephrine and propranolol suppress endogenous insulin release from the pancreas. Whereas the exogenous insulin and glucose infusion is constant in all individuals, steady-state glucose (SSG) concentration is dependent on how well the infused insulin allows insulin-sensitive cells to take up glucose in the blood. As such, people with IR will display higher SSG concentration than will individuals with normal insulin function. Investigators studied 28 healthy, young (19–23 years of age) men and women, and the participants were equally divided into a normal-sleep control group or a sleepless experimental group. Participants were matched for blood pressure, age, body mass index, and sex. Although there were no initial significant differences in SSG concentrations at the baseline measurement, the participants who experienced 24 hours of sleep deprivation had significantly greater change in SSG.
concentration than individuals in the normal-sleep night group (P = .01). If sleep loss causes a transient state of IR, the pancreas may need to release extra insulin to clear BG. In consequence, many researchers assert that chronic sleep loss may contribute to the development of type 2 diabetes and metabolic syndrome, which are both also linked to obesity and overweight.5

High Carbohydrate Intake
In conjunction with IR, sleep loss may also increase appetite and induce cravings for high-carbohydrate foods.6 If this is the case, the problem of IR would be exacerbated if the sleep-deprived person consumes more high-carbohydrate food than usual. In this scenario, the beta cells of the pancreas would be stressed by a higher carbohydrate influx, which would necessitate a compensatory increase in insulin release.

Elevated Cortisol
Sleep deprivation may also increase the stress hormone cortisol, which potentiates increases in blood pressure and BG.7 Chronically elevated cortisol secretion is associated with obesity and diabetes. Some research shows that one sleepless night may significantly increase circulating cortisol levels. For example, a study of 33 healthy men examined plasma cortisol levels before and 36 hours after 3 experimental conditions: 8 hours of sleep (designated as “normal”; n = 9), 4 hours of sleep (n = 7), or total sleep deprivation (n = 17).5 The 3 groups of men had comparable body mass index and age ranges, and none of the participants reported problems with sleep. The experimental protocol allowed the researchers to examine the participants in a laboratory setting for 32 hours continuously, and measurements were taken from 6:00 PM to 11:00 PM on 2 consecutive nights. All groups of men showed typical fluctuations in circulating cortisol: cortisol was low in the late evening and early night, reached maximal levels in the morning, and tapered off throughout the day. However, the men in the restricted sleep conditions did not experience comparable cortisol reductions to those of the men in the normal-sleep group. The evening following the 4-hour sleep protocol, the participants’ plasma cortisol levels were elevated 37% above the baseline measurement (155 ± 17 vs 113 ± 14 nmol/L; P = .03). Total sleep deprivation resulted in a 45% increase in cortisol levels compared with the baseline level (168 ± 19 vs 116 ± 11 nmol/L; P = .003). Unfortunately, cortisol acts as a stimulus to break down muscle protein, which subsequently contributes to elevated BG and may lower metabolism, as a result of muscle loss. Furthermore, research also suggests that excess endogenous glucocorticoids may induce IR directly.7

Appetite Manipulation
In addition to insulin and cortisol modulation, poor sleep affects a number of hormones that are known to directly influence feeding. As a result, the sleep-deprived individual may battle an elevated appetite when BG control is deregulated.8

Lowered Leptin
Scientists used to consider fat tissue an inert storage depot for excess energy. Now, researchers and medical professionals recognize that adipose is a powerful hormone-releasing endocrine organ, and its hormones have an influence on diet and activity behaviors. Leptin is one hormone secreted from the fat tissue, and it binds to receptors on the hypothalamus, which is the appetite center of the human body. Generally, leptin levels are greater with high body fat mass and recent feeding. Therefore, high circulating leptin values indicate that an individual is in a state of positive energy balance. Under normal conditions, elevated leptin levels blunt appetite and increase energy use, mostly through nonexercise activities.3,5 In a crossover study, conducted in a sleep laboratory, 11 lean men were permitted to sleep for 12, 8, or 4 hours for 6 nights in a row.8 Compared with the 12-hour sleep condition, the 4-hour sleep protocol caused a 20% drop in circulating leptin levels, independent of changes in fat mass or energy intake. In response to sleep loss, the body receives a signal that energy levels are low. In fact, the leptin reduction observed following 6 nights of sleep loss was comparable to the leptin reduction observed in healthy young men after 3 days of a 900-kcal/d of energy restriction, as reviewed by Spiegel et al.8 In consequence to the reduction of leptin following sleep loss, appetite may increase.

Increased Ghrelin
It is convenient to think of ghrelin, secreted by the stomach, as a hormone that opposes leptin. Higher-than-normal ghrelin levels signal an energy deficit to the body; hence, a person is likely to eat more and move less when circulating ghrelin is high.10 One crossover study revealed that 1 night of total sleep deprivation induced a 22% increase in circulating ghrelin levels and increased hunger in 9 healthy men.11 Furthermore, another randomized, crossover study of 12 healthy men subjected to two 4-hour sleep nights showed that sleep loss was associated with a mean leptin drop (18%; P = .04), an increase in circulating
Taken together, ghrelin (28%; $P < .04$), and increased hunger (24%; $P < .01$) and appetite (23%; $P = .01$).\textsuperscript{4} Taken together, one may speculate that concomitant ghrelin and leptin alterations after a period of inadequate sleep may stimulate overeating.

**Increased Eating Time and Frequency**

If an individual spends more time awake, he/she is apt to take in an extra meal or two, especially if abnormal hormonal signals stimulate hunger and carbohydrate cravings. Therefore, a sleep deficit of 3 to 4 hours may leave the sleep-deprived predisposed to consuming poor-quality food, with little motivation to move and expend the extra calories. In addition, both epidemiological and laboratory-based studies show that people actually eat more, on average, with less sleep.\textsuperscript{12}

**Reduction of Resting Energy Expenditure and Non–Exercise-Activity Thermogenesis**

**Decreased Spontaneous Energy Expenditure**

Sleep loss may impact spontaneous movement, such as non–exercise-activity thermogenesis (NEAT), and energy expenditure, in addition to appetite and eating behaviors. Any movement that is not planned exercise is considered to be NEAT. As such, fidgeting, pacing, and other habits are NEAT activities. Most of an individual’s daily energy needs are for the resting energy expenditure, which is the minimum amount of energy required to keep a body awake and alive; NEAT is second, and formal exercise is third. If NEAT activities subside significantly, the result may be more detrimental than abstaining from exercise. As reviewed by Knutson et al,\textsuperscript{3} individuals who have trouble sleeping report significantly less engagement in physical activity. For instance, when a person is sleep deprived, metabolically imbalanced, nd tired at 3:00 pm, will he/she feel like playing with a child or getting up to talk to a coworker? It is not likely, and the person will sacrifice NEAT energy use. Although one may speculate that sleep loss affects energy use in humans, there are not enough data to make definitive conclusions.

**Reduced Thyroid-Stimulating Hormone**

The thyroid axis begins with the secretion of thyroid-releasing hormone (TRH) from the hypothalamus, which signals the pituitary gland to secrete thyroid-stimulating hormone (TSH). In turn, TSH prompts the thyroid gland to produce and release thyroxine (T4) and triiodothyronine (T3). The T3 and T4 thyroid hormones control the overall energy use of most human cells and provide negative feedback to both the pituitary and hypothalamus, so TRH and TSH are not released when T3 and T4 levels are adequate. In contrast, when low circulating T3 and T4 are sensed by the hypothalamus and pituitary, TRH and TSH are secreted to induce thyroid hormones production and secretion. Clinically, plasma TSH levels are often used as an indicator of thyroid function. One study\textsuperscript{13} revealed that healthy men who slept about 4 hours per night over a 6-day period, in contrast to men who had 9 hours of sleep per night, had a 30% drop in TSH secretion. As a result, reduced TSH release may result in reduced thyroid hormone release. On the other hand, further research is needed to determine if T3, T4, and energy expenditure are influenced by sleep loss.

**Elevated Metabolism in the Second Half of Sleep**

Rapid-eye-movement (REM) sleep is greater during the second half of the sleep night. Rapid-eye-movement sleep is important for a number of reasons; however, resting energy expenditure is also higher during REM than during the deeper sleep that predominates the earlier sleep cycles.\textsuperscript{14} Furthermore, a study of 335 children and adolescents, aged 7 to 17 years, revealed that 1 hour less of total sleep was associated with a 1.85 odds ratio of overweight, and 1 less hour of REM sleep was associated with an odds ratio of 2.91.\textsuperscript{15} Although a prospective study is required to demonstrate that reduced REM sleep time causes obesity, the association between reduced REM sleep and obesity is interesting and presents another area of future research opportunity.

**Postpartum Weight Loss**

Gunderson et al\textsuperscript{16} studied 940 postpartum women longitudinally and revealed an association between sleep quantity and postpartum weight loss. Although women who slept more than 8 hours per night did not have an increased risk of postpartum weight gain, women who slept less than 5 hours per day ($n = 112$) after giving birth were more than twice as likely as women who slept at least 7 hours ($n = 319$) to retain an extra 13 lb at 1 year after giving birth. Furthermore, the women who managed to collect at least 7 hours throughout the day and night ($n = 545$) were more likely to return to their prepregnancy weights within 1 year. Although the study shows only an association between adequate sleep and postpartum weight loss, it suggests that sleep may be a factor in postpartum weight loss.

**Excessive Sleep**

On the other hand, some evidence also suggests that excessive sleep may be detrimental. One analysis of the
Specifically, Nutrition Today, As reviewed by Experimental evidence suggests that sleep loss The literature sex and age both impact The scientific literature suggests that acute Ten Steps to Better Sleep How Sleep Deprivation Manipulates Appetite and Weight Ten Steps to Better Sleep

<table>
<thead>
<tr>
<th>Table. Ten Steps to Better Sleep</th>
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<tbody>
<tr>
<td>(1) Stick to a regular bedtime</td>
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<tr>
<td>A consistent bedtime is important to maintain regular hormone secretions, including melatonin.</td>
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<tr>
<td>(2) Avoid caffeine late in afternoon</td>
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<tr>
<td>Caffeine has an average circulation half-life of about 6 h and may be elevated to 60 h in liver conditions; therefore, an afternoon dose of caffeine may easily prevent some people from falling asleep.</td>
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<tr>
<td>(3) Avoid alcohol before bed</td>
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<tr>
<td>Although alcohol can induce drowsiness, the effect is often short-lived. Drinking alcohol also prevents progression into the deep-sleep stages.</td>
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<tr>
<td>(4) Keep all lights out</td>
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<tr>
<td>Serotonin production is inhibited by light. Use heavy drapes to block nighttime light pollution and early-day light and a motion-sensitive night light for the bathroom, and avoid clocks with bright numbers.</td>
</tr>
<tr>
<td>(5) Go to the bathroom right before bed</td>
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<tr>
<td>There is less likelihood of rising in the middle of the night if the bladder is voided before bed.</td>
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<tr>
<td>(6) Turn the television off</td>
</tr>
<tr>
<td>The light emitted from the television may stimulate the brain and impair one’s ability to fall asleep.</td>
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<tr>
<td>(7) Stay off the cell phone before bed</td>
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<tr>
<td>Exposure to cell-phone radiation may prevent melatonin production; limit cell phone use before bedtime.</td>
</tr>
<tr>
<td>(8) Exercise</td>
</tr>
<tr>
<td>At least 30 min of daily exercise will help most people fall asleep at night. However, exercise within an hour or two of bedtime may make sleeping more difficult.</td>
</tr>
<tr>
<td>(9) Cool off</td>
</tr>
<tr>
<td>Core body temperature must drop to induce sleep—try to keep the bedroom below 70°F.</td>
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<tr>
<td>(10) Focus on sleep</td>
</tr>
<tr>
<td>Do not use the bedroom for tasks such as study, work, or television watching.</td>
</tr>
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</table>

Nurses Health Study (n = 82,969) reported that sleeping less than 6 hours or more than 7 hours is associated with an increased mortality. Specifically, the relative mortality risk for 5 hours or less per sleep night was 1.15 (95% confidence interval [CI], 1.02–1.29), 1.01 for 6 hours (95% CI, 0.94–1.08), 1.00 for 7 hours (reference group), 1.12 for 8 hours (95% CI, 1.05–1.20), and 1.42 for 9 or more hours (95% CI, 1.27–1.58). Investigations of obesity risk, however, demonstrate an overwhelming increased obesity risk with fewer sleep hours and little association with increased sleep and obesity risk, as reviewed by Taheri. This relationship is evident in children, and there is a linear relationship between reduced sleep hours and obesity risk.

In addition, the optimal number of sleep hours is highly individual, which makes general sleep recommendations difficult to determine. Some individuals do not show symptoms of sleep deprivation with less than 6 hours of sleep per night (short sleepers), whereas some individuals require 9 to 10 hours of sleep each night (long sleepers). As reviewed by Ferrara and De Gennaro, sex and age both impact sleep needs—women tend to sleep longer, and humans generally sleep less as they age. Factors thought to influence sleep needs should be integrated into the design of future sleep research. Furthermore, investigators should ensure continuity in the number of hours participants typically sleep each night before the study begins.

**Summary**

There is a long-term association with chronic sleep loss of less than 6 hours per night and obesity and type 2 diabetes. The scientific literature suggests that acute and chronic sleep loss may impair the hormone homeostasis of otherwise healthy humans. Moreover, studies examining the acute effects of sleep loss reveal impaired glucose tolerance after only 1 night of sleep loss. Experimental evidence suggests that sleep loss elevates the stress hormone cortisol. The literature also indicates that sleep loss may also impact hormones that are directly related to food intake and appetite. Specifically, ghrelin is elevated by about 22% to 28%, and leptin is decreased by about 20%; collectively, these changes may stimulate appetite and increase feeding. In conclusion, additional prospective research is needed to confirm the effects of sleep loss on the endocrine systems; however, the available literature supports the utility of sleep habit evaluation for obese patients and clients (Table).
How Sleep Deprivation Manipulates Appetite and Weight

Energy Needs

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