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Obesity is rapidly becoming the commonest preventable cause of death, yet we have no effective measures to curb this pandemic. This is because obesity results from complex interactions between genes, behaviour and the environment. One important factor that may be contributing to hunger, food selection and energy expenditure is the lack of sleep. Sleep is not (as commonly believed) a passive state; it is a highly active and complex state whose precise physiological functions have remained a mystery. Sleep loss, however, can have serious consequences for health and mortality. The role of acute sleep loss in poor cognitive performance and as a cause of motor vehicle accidents is increasingly appreciated, but little is known about the consequences of chronic partial sleep loss (which we all commonly experience). In the second half of the last century, because of increasing pressures on our time (work, school, family, television, and now computer games and the internet), we have reduced the time we spend sleeping by up to two hours a night, on average. A substantial proportion of adults now sleep less than eight hours a night. At the same time as sleep duration has decreased, the prevalence of obesity has increased. This may be a coincidence, but the connection between short sleep duration and obesity has been seen in very large population studies across all age groups, even in children as young as five years old:

- In a French study of 1,031 children aged five years, children sleeping less than 11 hours a night were significantly more obese.
- In a Japanese study of 8,274 children, aged six to seven years, the odds of obesity were about three times greater for sleep of less than eight hours a night, compared with sleep of 10 hours.
- A Spanish study of 1,772 adolescents (15 years or older) reported an association between short sleep duration and obesity.
- Using sleep data of 1.1 million adults from the American Cancer Society, a relationship between short sleep duration and obesity was observed. This study also showed an association between sleep duration and mortality.
- In a sample of 496 Swiss adults followed up over 13 years, there was an increase in body mass index (BMI) from 21.8 kg/m^2 (age 27 years) to 23.3 kg/m^2 (age 40 years), with a concurrent decrease in sleep duration from 7.1 to 6.9 hours a night in men, and from 7.7 to 7.3 hours in women. This study noted that the association between short sleep duration and obesity diminished with ageing; therefore this association may be most important early on in life where it may have greatest impact on setting future eating habits.

> On average, over the past 50 years, we have reduced the time we spend sleeping by up to two hours a night

How does short sleep duration impact on body weight? Are appetite and metabolic hormones involved? Two opposing hormones in the regulation of appetite are leptin and ghrelin. Leptin is released by adipocytes (fat cells) to signal the state of fat stores, while ghrelin is released by the stomach to signal hunger. Low leptin and high ghrelin signal an energy deficit to the hypothalamus in the brain, resulting in hunger. In one small study, total sleep deprivation in 10 healthy volunteers reduced their leptin levels, but these returned towards normal levels after recovery from sleep loss. In another study, volunteers were restricted to four hours of sleep for six nights. This resulted in decreased leptin and increased ghrelin levels, which corresponded to increased hunger and an appetite for ➜

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urrently facing an obesity pandemic that is likely to r medical, social and economic consequences. Of cern is the alarming increase in obesity in children. *aheri*, working in the Henry Wellcome Laboratories Neuroscience and Endocrinology, is exploring the vel idea that a lack of sleep may influence obesity.

➜ high-calorie foods. The important conclusion from these studies is that sleep loss may impact on the secretion of hormones that regulate appetite and energy expenditure.

However, the question must be asked: are the changes in appetite hormones with sleep loss observed in small, highly controlled, laboratory studies reflective of real life? During Taheri's work at the Howard Hughes Medical Institute at Stanford University, in collaboration with the well-established Wisconsin Sleep Cohort study, he studied over 1,000 adults from the general population in the sleep laboratory and obtained information regarding their sleep habits through sleep diaries and questionnaires. They also measured hormones that are important in the regulation of appetite and energy expenditure, and confirmed that short sleep duration is associated with increased body weight. Most importantly, they showed for the first time in a large population that there is an explanation behind the association between short sleep duration and obesity. Lack of sleep affects leptin and ghrelin; with short sleep duration, leptin is lower and ghrelin higher. With sleep loss, low leptin and high ghrelin can give powerful dual signals that the body has an energy deficit, thus increasing food intake. In societies with easy access to high-calorie foods, this may contribute to the development of obesity.

The intriguing and fascinating relationship between sleep duration, metabolic hormones and obesity, with potentially important public health implications, remains to be investigated further. Other hormones beside leptin and ghrelin and other factors may also be involved. Obesity is likely to become the commonest preventable cause of premature death in the Western world, while its prevalence is increasing globally. Taheri's research in Bristol aims to unravel the mechanisms involved in the interaction between sleep and metabolism, while exploring whether sleep manipulation can be of benefit. Manipulating sleep in the prevention of obesity, and as an adjunct to lifestyle change and medical and surgical therapy, may be a novel approach to address the current obesity pandemic. But there are many other unknown factors that contribute to the world's accelerated weight gain. Here in Bristol we have recognised that tackling obesity requires a multidisciplinary approach. As a consequence, a collaborative group has been formed across several departments and faculties to increase our understanding of obesity and to devise effective strategies to deal with this pandemic. ■

www.bris.ac.uk/obesity

Body mass index

BMI is a measure of obesity. It is calculated as weight [kg] divided by height [m] squared [^{2}].

Example:
1. Work out your height in metres and multiply the figure by itself.
2. Measure your weight in kilograms.
3. Divide the weight by the height squared (ie the answer to Step1).

For example, if you are 1.7 m (5 feet 7 inches) tall and weigh 65 kg (10 stone), the calculation would be: 1.7 x 1.7 = 2.89, therefore your BMI would be 65 divided by 2.89 = 22.5. Your weight would be within the ideal range shown in the table below.

Recommended BMI chart	BMI
Underweight	less than 18.5
Ideal	18.5-25
Overweight – should lose weight	26-30
Obese – should lose weight now	31-40
Very obese – lose weight now	greater than 40

In addition to the BMI, the waist circumference adds to increased risk of obesity complications and mortality. In men, waist circumference above 102 cm (40 inches) and in women above 89 cm (35 inches) confers increased risk.