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PEDIATRIC EDITORIAL

Have we been barking up the wrong tree: can a good night's sleep make us slimmer?

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Imagine that you suddenly read in Nature that scientists have discovered a new environmental agent that has an adverse effect on the hypothalamic system for appetite regulation, and makes the affected subjects extremely vulnerable to weight gain when exposed to a high-fat diet, large portion sizes and physical inactivity. The agent was found to cause a desensitising of a number of receptors mediating the satiating effect of leptin, GLP1, CCK and PYY, and by this means it disconnects the brain from most of the communication from the periphery, so that the brain is unable to respond adequately when the gastrointestinal tract, and glycogen and fat stores are all full of nutrients. The agent could be a virus, a pollutant, a trans fatty acid or another environmental factor to which exposure has increased dramatically over the past 60 years, coinciding with the obesity epidemic. Would you be surprised? Or would you feel that we now have a sensible explanation for what is really going on?

Most authorities, including task forces, WHO and consensus panels, conclude that most of the overweight and obesity problems could be prevented if we had less fat in our diet, consumed fewer sugar-rich beverages, ate smaller portion size and did our 30–60 min exercise every day. This may be true for those who are slightly overweight. But can it prevent and cure obesity? Most systematic reviews and meta-analyses of reduction in dietary fat suggest that a weight loss of approximately 3–5 kg can be expected with a reduction in the proportion of energy from dietary fat of 10 percentage points (e.g. from 40 to 30%). Estimates of the impact of training and physical activity on body weight do not really show any major effect. The genetic component is well recognised but it can be excluded as a cause of the obesity epidemic, as the gene pool has not changed over the past 60 years. So what can explain that some people gain 20 or 50 kg?

The study by Chaput *et al.*,[1] published in this issue of The Journal, analyses data from an observational, cross-sectional study of 422 children, aged 5–10 years, from Quebec. They find that those who slept less than 10–13 h/day had an increased risk of overweight and obesity. Children who slept 10.5–11.5 h/day had a 42% increased risk, whereas those who slept only 8–10 h/day had a 245% increased risk. In addition, they found an inverse association between sleep duration and body weight, and between sleep duration and waist circumference in boys. If these risk estimates are correct, then too little sleep may be quite important as a predisposing condition for weight gain and obesity, and it may even be a key to an effective prevention strategy. However, this is an observational, cross-sectional study, and even though the associations remained after adjustment for various confounding factors, such a study cannot prove any causal relationship. Another shortcoming of observational studies is that they are based on self-reported information, whether regarding sleep quality or sleep duration. Only recently a case reference study has shown that obese subjects with type 2 diabetes do seem to have an impaired quality of sleep as compared to simple-obese subjects, as assessed by night time radar measurements.[2] There are actually a number of longitudinal studies in the literature showing that poor quality and short duration of sleep are risk factors for weight gain and type 2 diabetes in children.[3] It has also been shown that poor quality of sleep is associated with increased risk of obesity in adults. In a German cohort of about 4000 men and 4000 women, sleeping difficulties were associated with an increased risk of developing type 2 diabetes by a mean follow-up period of 7.5 years.[4] In the Nurses Health Study, short sleep duration (<5 h/day) was associated with an increased risk of type 2 diabetes over a 10-year period.[5]

Before a causal relationship can be established, it is necessary to have some plausible biological mechanism linking impaired sleep with disrupted appetite regulation and sedentary behaviour, and to have some proof of this from randomised trials. Although such trials might be difficult to execute, they are certainly possible. Recent theory about the link between sleep and weight gain suggests that the crucial links are leptin and ghrelin, and hormonal factors may turn out to be only one part of the full story. There are already a few short-term trials, which have shown that sleep restriction is associated with an increased sympatho-adrenal secretion of cortisol and catecholamines,[6,7] and also with decreased evening leptin and increased ghrelin levels.[8] Such changes might lead to caloric overconsumption. Impaired sleep may actually be a sign of mental stress, and it may prove difficult to disentangle the causes of impaired sleep from its effects. Many other environmental factors may become apparent, everything from mental stress, excessive intake of alcohol or coffee, drug use, physical inactivity, pain, etc., can adversely affect sleep.

A contributing explanation may be that when we sleep more, we simply have less time in which to eat. In addition, too little sleep and impaired quality of sleep may affect our chronobiology and adipocyte function adversely.

How much of the current obesity epidemic can be explained by impaired sleep? Figuring this out is one of the major research challenges. There is no doubt that the prevalence of sleep disturbances has increased dramatically over the same period in which we have observed the major part of obesity epidemic.

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