

EDITORIALS

Is prolonged lack of sleep associated with obesity?

Possibly, and it could have other effects on long term ill health

Francesco P Cappuccio *cephalon chair of cardiovascular medicine and epidemiology*, Michelle A Miller *associate professor (reader) in biochemical medicine*

University of Warwick, Sleep Health and Society Programme, UHCW Campus, Coventry CV2 2DX, UK

In the linked longitudinal study (doi:10.1136/bmj.d2712), Carter and colleagues assess the association between reduced sleep and differences in body composition and the risk of becoming overweight in young children.¹

We spend about a third of our life asleep. We spend more time asleep as babies and children and then generally settle into a pattern of seven to eight hours a night. Sufficient sleep is necessary for optimal daytime performance and wellbeing, yet the amount of sleep that people get varies greatly.²

The quantity and quality of sleep have shown secular trends alongside changes in modern society that require longer hours of work, more shift work, and “24/7” availability of commodities. These changes have reduced the average duration of sleep across westernised populations, with increased reporting of fatigue, tiredness, and excessive daytime sleepiness.² This sleep curtailment has been attributed mainly to lifestyle changes.² Too little sleep is associated with adverse health outcomes, including total mortality,³ stroke and coronary heart disease,⁴ type 2 diabetes,⁵ hypertension,⁶ respiratory disorders, poor self rated health,² and obesity in adults and children.⁷

Obesity in childhood has now reached epidemic proportions and is a cause of psychosocial problems including low self esteem. It often continues into adulthood, where it causes major morbidity, disability, and premature death. Several studies have reported associations between short duration of sleep and the risk of obesity in children.⁷ Most studies have been cross sectional and therefore unable to support a cause-effect association. Nevertheless, some prospective longitudinal studies in children have shown that short duration of sleep may precede the development of overweight or obesity. The potential public health implications of a causal association would be far reaching. However, the studies have had limitations in that sleep assessment has often been based on parental reports or self reports rather than on direct measurements, and body mass index (BMI) has been the main outcome measure for obesity, with little additional specific measure of adiposity or fat distribution.

Carter and colleagues report the results of a longitudinal analysis of sleep duration in relation to the development of obesity in 244 children in New Zealand, followed from 3 to 7 years of

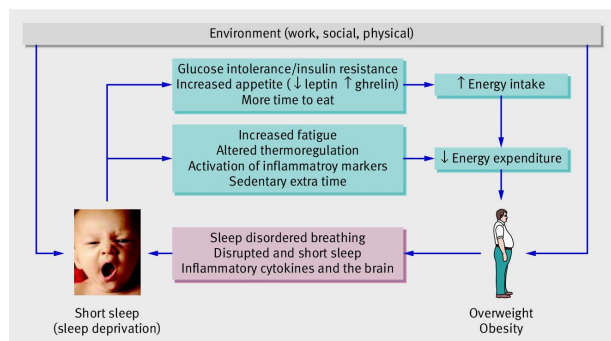
age.¹ The sleep duration of these children was directly measured with an accelerometer three times (at ages 3, 4, and 5) and BMI was measured yearly. In addition, yearly measurements were made of physical activity (by accelerometry), fat mass and fat-free mass (by bioelectrical impedance and dual energy x ray absorptiometry), and other confounders. After adjustment for multiple confounders, longer sleep duration at age 3-5 was associated with a reduction in BMI (0.48 for each extra hour of sleep) and a 61% reduction in the risk of being overweight at age 7. These differences resulted from an effect on fat mass rather than fat-free mass. The effect was independent of sex, maternal education and BMI, smoking during pregnancy, income, ethnicity, birth weight, physical activity, and some aspects of the children’s diet. The authors conclude that young children with inadequate sleep are at increased risk of overweight as a result of increased fat deposition.

The study is a valuable contribution to the understanding of the causal pathway whereby reduced sleep duration may directly contribute to overweight and obesity in children. The study is longitudinal, thus avoiding reverse causality. It also avoids the potential inaccuracies of reported sleep duration by using accelerometers and sleep diaries, which are more reliable, direct and repeated measures of time in bed and time asleep. Outcome is also carefully assessed by standard anthropometry and two direct measures of fat distribution. Important confounders are considered. Finally, the effect size is biologically relevant and, if reversible, of potential public health interest.

Important limitations are often unavoidable. For example, the response rate was 59%, too low by conventional standards to reassure the readers of lack of self selection bias. This might limit the generalisability of the findings. Moreover, the authors assumed a linear effect of sleep duration on outcome. This assumption may not hold if sleep duration exerted a threshold effect that led to a U shaped association, as is the case with other outcomes in adults.³⁻⁶ The residual confounding by socioeconomic status may not be fully accounted for by maternal circumstances (education and income) because the household socioeconomic status is often determined by indicators such as paternal income, access to commodities, house size, and

crowding. These are generally associated with sleep duration, at least in adults.²

How would short duration of sleep cause obesity? It has been suggested, mainly through short lived severe sleep deprivation experiments in young volunteers, that short duration of sleep may cause an increase in energy intake and a reduction in energy expenditure through activation of hormonal responses (such as reciprocal changes in leptin and ghrelin). This might lead to an increase in appetite; glucose intolerance and insulin resistance⁸; activation of low grade inflammation; and a reduction in energy expenditure also mediated by the modulation of thyrotropin, cortisol, and noradrenaline (figure).^{2,9}



Finally, one obesity subtype might be associated with emotional distress, poor sleep, fatigue, hyperactivity of the hypothalamus-pituitary-adrenal axis, and hypercytokinaemia whereas the other subtype is associated with non-distress, better sleep but more sleepiness, normoactivity or hypoactivity of the hypothalamus-pituitary-adrenal axis, and hypercytokinaemia.¹⁰ Sleeping less would give people more time to eat and engage in other sedentary activities, as exemplified by children and adolescents who like to stay up late to play on their computer or watch TV while snacking (although this scenario was excluded in the current study). The association between short duration of sleep and obesity is open to the possibility of a reverse causality pathway, whereby obesity causes short or disrupted sleep (or both) because of breathing problems at night and the effect of inflammatory markers on the brain's regulation of the circadian rhythm.¹¹ It is intriguing that although prospective studies in children have shown significant effects, the results in adult cohorts are less consistent.² In addition, the effects of short term acute and extreme changes in sleep duration

may not reflect what happens under less severe and more sustained chronic sleep deprivation.

The ultimate proof of principle should come from a randomised trial of sleep “prolongation” on weight change.¹² The problem is that currently we do not have an effective and reproducible intervention tool. Future research should therefore explore and validate new behavioural (non-drug based) methods to prolong childrens’ and adults’ sleeping time. In the meantime it would do no harm to advise people that a sustained curtailment of sleeping time might contribute to long term ill health in adults and children.

Competing interests: All authors have completed the ICMJE uniform disclosure form at www.icmje.org/coi_disclosure.pdf (available on request from the corresponding author) and declare: no support from any organisation for the submitted work; no financial relationships with any organisations that might have an interest in the submitted work in the previous three years; no other relationships or activities that could appear to have influenced the submitted work.

Provenance and peer review: Commissioned; not externally peer reviewed.

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Cite this as: *BMJ* 2011;342:d3306