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VIEWPOINT

Sleep in children and adolescents: A worrying scenario
Can we understand the sleep deprivation–obesity epidemic?

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Kotagal and Pianosi [1] recently completed a clinical review on sleep disorders in children and adolescents. The authors are to be commended on providing a timely reminder of this often overlooked, but highly important area of medical practice. An account was given of how to clinically assess sleep problems in this age group, which is highly important given that research suggests we do not adequately tackle this problem in our clinical practice. The more intricate sleep disorders like narcolepsy and sleep phase disorder were made accessible as was the more common and widely applicable notion of sleep hygiene. Kotagal and Pianosi suggest that by improving our patients sleep hygiene we can improve their health in many systems – nervous, cardiovascular, respiratory and metabolic – and by this reduce the burden of many of our most prevalent diseases including diabetes and ischaemic heart disease.

We feel, however, that one important aspect was missing from the impact of poor sleep. Many large scale epidemiological studies have linked shorter sleep with an increased prevalence and incidence of obesity. Obesity has become a significant public health problem, in particular, it is associated with the development of type 2 diabetes mellitus, coronary heart disease (CHD), an increased incidence of certain forms of cancer, respiratory complications (obstructive sleep apnoea) and osteoarthritis of large and small joints [2,3].

For the past couple of decades, a growing epidemic of childhood obesity has been noted [4–8]. This is particularly worrying not just because of the immediate negative impact, but because of the deleterious effects that childhood obesity can have on future health [9,10]. The most effective way of tackling obesity is to identify contributory risk factors and then devise strategies to reduce them [11]. There are only a few longitudinal studies in children to comprehensively study predictors of the development of obesity [12]. Reilly et al. [13] have made some ground in this area. Using the Avon Longitudinal Study of Parents and Children (ALSPAC), conducted in the UK, 8234 children were followed from birth and factors identified that contribute to obesity at age seven. A wide variety of

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potential risk factors were studied — socioeconomic variables, intra-uterine and perinatal factors, family characteristics, infant feeding and childhood lifestyle (including sleep duration). Using the definition of obesity as a body mass index (BMI) $\geq$ 95th percentile of a UK reference population, it was found that short sleepers (<10.5 h/night) at age 30 months were more likely to be obese at age 7 years than those who slept longer (>12 h/night). The estimated risk was 45% greater. The association remained independent of all other factors.

There have been several other epidemiological studies that have reported an association between short sleep duration and increased BMI, in both children and adolescents [14—16].

A cross-sectional study [14] of 6862 German children aged 5—6 years determined that those sleeping least (<10.5 h/night) were more likely to be classified as obese. This study however uses a slightly different method of quantifying obesity — within the top 3 percent of population BMI, which can make the results more difficult to reconcile.

Studies in adolescents have discovered similar findings. Adolescents are a population that may be at particular risk because most may get significantly less sleep than they need, leading to chronic partial sleep loss [17]. Gupta et al. [15] examined 383 11—16-year-old boys and girls in the cross-sectional Heartfelt study. Obese adolescents experienced less sleep than non-obese adolescents. For each hour of lost sleep, the odds of obesity increased by 80%. Unfortunately, this study did not attempt to control for mental health issues, like depression, which are well known to affect sleep [18].

Interestingly, research has indicated there may be differences in the relationship between short sleep and obesity between boys and girls. Knutson [16], using data from 4486 adolescents from the National Longitudinal Study of Adolescent Health, converted BMIs into age- and sex-specific *z*-scores to allow more suitable stratification of risk. When these *z*-scores were compared with the respective sleep duration, it was found that every hour increase in sleep duration was associated with a 10% reduction in risk of being overweight in boys. No comparable effect was found in girls. Again, mental health status was not included as a variable in this study, and there are gender-specific differences in the sleep relationship with depression in adolescence [19]. Nevertheless, the results of this study remain of interest and warrant further investigation.

Restricted sleep and obesity could be associated for several reasons. First, recent investigations documenting the connection may be mediated by a neuroendocrine system. Spiegel et al. [20] reported metabolic and hormonal consequences of acute sleep restriction. Young healthy men limited to 4 h of sleep for six consecutive nights experienced impaired glucose tolerance, reduced glucose utilisation, an exaggerated acute insulin response to glucose, and increased sympathetic activity. Spiegel et al. drew similarities between these disturbances to those of aging or gestational diabetes. In addition, over time it was thought the poorer carbohydrate handling and raised sympathetic activity may cause hypertension, diabetes or obesity.

Furthermore, sleep restriction reduces the orexigenic hormone leptin, which is secreted from adipocytes [21] to decrease appetite and increase energy expenditure. Thus leptin, in part, may mediate the sleep—obesity relationship. Levels of ghrelin, another potent stimulator of appetite were elevated in short sleepers [21], thus increasing appetite.

It is ironic that a reduction in the most sedentary activity of all — sleep — should be associated with weight gain. In the midst of all this science it is important that we do not discount the more obvious explanation — that being awake for longer, gives us more time to eat. Whilst this may be true for contemporary children who would spend more of their 'awake' time in sedentary activities [22], like watching TV, sitting at the computer and playing computer games — with the consequent comfort of eating, it is also true that more recent suggestions challenge the fact that the association between short sleep and obesity has anything to do with eating more or exercising less, but it would find more complex pathophysiological explanations in reduced basal metabolic rate or in reduced non-exercise associated thermogenesis.

The complex interplay between hormones, appetite, exercise and thermogenesis, and any subsequent weight gain is not fully understood, and should be targeted for future research.

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