COMMENTARY

Increasing Sleep Duration for a Healthier (and Less Obese?) Population Tomorrow

Commentary on Cappuccio FP, Taggart FM, Kandala N-B, et al. Meta-analysis of short sleep duration and obesity in children and adults. Sleep 2008;31:619-626.

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EVER-INCREASING REPORTS LINKING SHORT SLEEP DURATION WITH INCREASED OBESITY HAVE PROMPTED HEATED DEBATES, EDITORIALS, AND MANY NEWS reports. With the current demand for rapid "bench to bedside" translation of research findings, the short sleep-obesity link has been widely disseminated already. The general public, health providers, and policy makers have practical questions: Does sleeping too little make you fat? Can you lose weight by sleeping longer? After nearly 700 studies, what answers can the field of sleep research offer?

Cappuccio and colleagues[1] have done the heavy lifting for us by gleaning the literature and abstracting data on 634,511 participants from 30 studies. The aims of their report, in this issue of *Sleep*, were to assess whether evidence solidly supports an association of short sleep duration and obesity, and to quantify the association using pooled data. The meta-analysis results, using dichotomous variables for sleep duration and obesity, show that compared with children sleeping >10 hours, those sleeping 10 or fewer hours/night have 89% greater odds of being obese (OR = 1.89, 95% CI = 1.47-2.43, P < 0.0001) and adults sleeping five or fewer hours versus >5 hours have 55% greater odds of being obese (OR = 1.55, 95% CI = 1.43-1.68, P < 0.0001). Regression analyses with continuous variables using pooled data on adults predicted that a 1-hour/night increase in sleep was associated with a decrease of 0.35 BMI units. The authors concluded that there is consistent evidence for an increased risk of obesity with short sleep duration, but that a causal link is unproven. More prospective studies incorporating repeated measures of sleep duration and rigorous control for confounders were recommended. The authors' answer to questions about increasing sleep to lose weight would likely be "we do not know yet."

Also in this issue of *Sleep* is a letter from Horne expressing his consternation that the sleep-obesity link, even if causal, has been overrated in importance.[2] In his letter, "Too weighty a link between short sleep and obesity," he touches on key points common to any risk factor reduction strategy. Horne notes that the prevalence of the "exposure" is low (few people are short sleepers), the effect size is small, and suggests other outcomes of short sleep (e.g., accidents) are worse than a slight weight gain. My guess is that his answer to the individual wondering if they can sleep away their obesity would be "no."

The comprehensive review article by Cappuccio and colleagues[1] and the letter to the editor by Horne[2] underscore the tough issues in translating study findings into action and provide an opportunity to step outside the clinical box and consider determinants of population health. The leap from published findings to policy has always been contentious, cheered on by advocates and delayed by objective purists waiting for a better model fit.[3] In addition to the soundness of findings, other factors weigh in on whether or not policy change and population initiatives are warranted, or if more research is needed. Let us consider the feasibility of increasing sleep to decrease obesity from the broader population perspective, for example, as seen by federal agencies developing Healthy People 2020 (see www.healthypeople.gov)—a program to set goals for improved population health with an emphasis on obesity in children—or policy makers considering regulation of work schedules that curtail sleep duration.

Central to the goal of assessing the benefit of risk factor reduction is whether the factor has a causal role in the outcome. Cappuccio et al[1] conclude that a causal link has not been *proven*. But, can this ever really be achieved? Causal inference in epidemiology—much simpler in the previous era of infectious diseases with necessary and sufficient causes—has evolved into a maze of argumentation, rooted in the history and philosophy of science, with no bottom line.[4] Earlier schemes and checklists for scientific inferences are flawed in light of the complexity of biologic associations with gene-environment interactions and an ever-changing socioeconomic context. Biomedical scientists, described as "practitioners who use a variety of criteria to evaluate data in conditions that provide less than total certainty" must deal with a gradient in level of certainty for causality. And researchers have differed in where they set the level for the sleep-obesity link, with some concluding that there is sufficient evidence for causality and initiation of prevention strategies.[5]

With even a modest level of causal evidence, how compelling is the case for increasing sleep duration as a strategy to decrease obesity? Overweight and obesity are high on the list of predictors of poor health; diabetes, sleep apnea, hypertension, and other significant disorders have been attributed to excess weight.[6] Although obesity is reversible, voluntary weight loss is difficult to sustain. Thus, preventing some fraction of obesity prevalence would be a great benefit to population health.

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With reduction of obesity a worthy goal, can "increased sleep" make enough of a difference? Horne[2] is underwhelmed with the magnitude of the sleep-obesity link. Questioning the importance of small effect sizes is not uncommon. Reaction to findings that some risk factor causes an elevation of 3-4 mm Hg in systolic blood pressure with $P < 0.001$, often goes like this—"yes, it's statistically significant, but is it *clinically* significant?" What is missing is the consideration of whether the effect is of *public health* significance.

Geoffrey Rose, a British epidemiologist and pioneer in prevention, clarified the distinction between "individual" and "population" in interpreting effect sizes.[7] Study findings from samples of individuals are average values that express the mean of a target population; Rose stressed the value of small shifts in those population means toward healthier values. Consider the meta-analysis estimate[1] that sleeping one extra hour would lower the average BMI of adults by 0.35 units, equivalent to 1-3 kg of weight depending on height. Clinically important on an individual level? No, but definitely of public health significance. Rose and Day[8] demonstrated with data from 32 countries, collected in 1988, that a reduction of just 1 kg in the mean weight of a typical European population (e.g., mean BMI = 25) was equivalent to a decline in the prevalence of overweight from 6% to 4%: a reduction of 33%. This benefit would be more dramatic in the US today, where the BMI distribution has a mean of 28 and a disproportionately extended tail of extreme obesity.

Prevalence of the risk factor is an important component in extrapolating the impact of a particular effect size. Horne notes 5% to 8% of adults are short sleepers, and expresses with various comparisons why he believes that the reduction of obesity attributable to increasing sleep would be trivial.[2] Citing study findings of a 2-fold increased prevalence of obesity for children sleeping <10 versus >10 hours/night, he correctly pointed to the less impressive results when expressed as absolute, rather than relative differences: 6% of obese children were short sleepers compared to 3.6% of the non-obese. Stressing that relatively few obese people are short sleepers and few short sleepers are obese, he implies that overall little benefit in terms of obesity could be gained by changing sleep duration. Recall, however, that not all smokers get lung cancer, and lung cancer has causes other than smoking.

With a standard formula,[9] I calculated the population attributable risk for children and adults, based on exposure prevalence from published data that 5% to 15% of children[10] and 5% to 8% of adults are short sleepers[2] and the pooled odds ratios[1] (1.9 for children and 1.6 for adults). The results indicate that 5% to 13% of the total proportion of obesity in children and 3% to 5% in adults, could be attributable to short sleep. *A reduction of at least 5% of the population burden of obesity would be of clear public health significance.*

Enthusiasm for gambling on a risk reduction strategy with only modest causal evidence should be tempered by the cost-benefit ratio. The benefits of reducing short sleep extend far beyond a reduction in obesity, as Horne pointed out,[2] and costs are minimal: loss of an hour or so of wake time among those spending 19 or more hours awake per day. With public health in mind, need we delay attempts to increase sleep duration in short sleepers as a population goal until we have more longitudinal data? Undoubtedly, better studies will be conducted, effect sizes will be more precise, and casual certainty will be fine-tuned. In the meantime, with 700 studies behind us and well summarized by Cappuccio and colleagues,[1] certainty that short sleep is not healthy, that short sleepers are more likely to be obese, (with a good chance that short sleep is the culprit in a meaningful number of children and adults), we *do* have enough to advocate for population-targeted educational initiatives and policies to increase sleep duration as a means of reducing obesity. No, I would not tell an individual that they can sleep away their obesity, but, with a moderate degree of certainty, I expect that if short sleepers increase their sleep time, the population as a whole will be healthier and rank lower in mean BMI. In the words of one renown for his logic, "the needs of the many *outweigh* the needs of the one" (Spock, *Wrath of Khan*, 1982).

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