Early Bed for Early Birds: Curbing the Evening Calories

Adolescent obesity remains an area of significant concern across the globe, as are the long-term consequences of adolescent obesity. Recent prospective data from a large cohort of adolescents showed that being obese as an adolescent conferred a strong likelihood of being overweight/obese by the age of 24 years [1]. Even apart from its social and psychological burden, as well as the negative impact on academic performance [2], obesity at a young age is associated with several adverse medical outcomes. In a cohort of 6,502 young men followed up in Denmark, 48% of those who were obese had developed diabetes, cardiovascular disease, or venous thromboembolism or had died before 55 years old [3]. In another cohort including 37,674 young men, there was an independent association between body mass index at 17 years old and angiography-proven coronary heart disease (β = 1.355, p = .004) [4]. Recent data from the U.S. National Health and Nutrition Examination Survey suggest that obesity at a young age is associated with seven to nine times (depending on degree of obesity) greater years of life lost compared with obesity at an older age [5].

Efforts to confront adolescent obesity are complicated by the significant social, psychological, and physiological changes that characterize this important developmental period. Two emerging factors that may play a role in adolescent obesity are sleep duration and the circadian clock. Adolescence is commonly associated with a physiological phase delay, resulting in adolescents going to sleep later in the evening and, if left to their own devices, waking up later in the morning. This phase delay is exacerbated by social factors such as the increasing popularity of modern technologies such as mobile telephones and computers. When combined with early school start times, this phase delay during adolescence can result in sleep loss. And, sleep loss has been shown to be associated with obesity and metabolic disturbance in both population [6] and human sleep laboratory studies [7]. Consequently, improving and extending sleep among adolescents may be a key strategy for reducing obesity [8]. However, the circadian clock may also play an important role in metabolic regulation and obesity. Adolescents who have an evening preference—evening chronotypes or “night owls”—tend to have unhealthy dietary habits and a greater propensity to obesity when compared with morning chronotypes or “morning larks” [9].

In this issue of the Journal of Adolescent Health, Beebe et al. [10] examine whether circadian preference determines how individuals respond to manipulation of sleep duration. Participants were permitted to sleep for 6.5 hours per night (sleep restricted) for five nights and 10 hours of sleep per night (healthy sleep) for five nights in a random counterbalanced order. Each sleep manipulation condition was separated by a two-night washout period. Adherence to the sleep study protocol was monitored using wrist accelerometry (actigraphy), allowing the adolescents to sleep within their usual sleep environment. The researchers determined sleep phase preference using the midpoint of the baseline sleep period during the usual sleep pattern. Caloric intake was measured during each of the conditions. The average baseline sleep duration was about 2 hours shorter (7 hours) than current adolescent sleep recommendations (9 hours), indicating that the sample was sleep deprived before entering the randomized experimental sleep condition. The mean sleep extension was about 2.5 hours beyond baseline sleep. When sleep was extended, adolescents accomplished earlier average sleep times of 22:15 compared with when sleep was restricted, where the mean sleep onset time was 00:44; wake times remained consistent. The caloric intake of the “night owls” was not altered by sleep manipulation. For “morning larks,” sleep extension (the “healthy” sleep condition) was associated with lower caloric intake. The authors conclude that sleep extension might be more beneficial for adolescents who are “morning larks” rather than “night owls.” It also appears that the caloric intake of “night owls” could be resistant to sleep extension. Thus, an obesity intervention that incorporates sleep manipulation should not concentrate only on sleep duration but must also be mindful of circadian preference.

Several factors must be considered when interpreting the results of Beebe et al. The authors originally randomized 88
participants but ended up with 67 (the majority, 61%, female). Importantly, 15% of those randomized were either non-adherent to the sleep protocol or dropped out. Enforcing changes in sleep timing and duration among adolescents is challenging given the real-life context of school attendance, academic workload, and social demands. The study manipulated sleep on just five nights per sleep condition. This approach may not accurately reflect the long-term effects of sleep alterations, which are likely to be more relevant to a chronic condition such as obesity. The population studied was nonobese, so it is unclear whether the findings will hold true for obese adolescents. The study concentrated on manipulating sleep timing, but delaying wake time may be more beneficial for adolescents’ metabolism. The use of actigraphy to determine sleep duration and timing is one of the study’s strengths. However, using actigraphy to determine circadian preference, although practical, is not as accurate as dim-light melatonin onset. Furthermore, actigraphy may overestimate sleep extension during periods of wakefulness (lying in bed) in the absence of movement. Although the study used a validated approach to collect caloric intake data, collecting accurate nutrition data is generally difficult. And, the investigators did not examine food selection, which may be more nutritionally important over the long term. The authors did not determine pubertal status, which may play a role in these processes. The impact of sleep extension on mood, cognition, and quality of life also was not examined. An investigation into the impact of sleep manipulation on metabolic markers and regulators of appetite and energy expenditure would also have been informative.

Although a significant body of evidence demonstrates a linear association between short sleep duration and obesity in children and adolescents, emerging data suggests that the circadian clock and circadian preference are also important factors, particularly in relation to dietary behaviors [9]. This is unsurprising given the complex interaction between sleep and circadian regulation. Future studies examining the association between sleep duration and obesity should consider the impact of circadian preference. In obesity prevention and management interventions, manipulating sleep timing may not provide benefits for all adolescents without concurrent changes in wake times. Furthermore, a simple intervention that merely asks adolescents to adjust their sleep times may create resistance and may not be beneficial or sustainable. A more personalized approach that takes sleep timing, circadian preference, and social factors into account is likely to be more successful.

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References