Sleep Medicine Reviews 36 (2017) 3–12

Contents lists available at ScienceDirect

Sleep Medicine Reviews

journal homepage: www.elsevier.com/locate/smrv

CLINICAL REVIEW

Is sleep education an effective tool for sleep improvement and minimizing metabolic disturbance and obesity in adolescents?

Teresa Arora [a, b], Shahrad Taheri [a, b, c, *]

[a] Department of Medicine, Weill Cornell Medicine, New York, USA
[b] Weill Cornell Medicine, Doha, Qatar
[c] Department of Medicine, King's College London, London, UK

ARTICLE INFO

Article history:
Received 30 September 2015
Received in revised form
27 April 2016
Accepted 19 August 2016
Available online 30 August 2016

Keywords:
Sleep duration
Sleep improvement
Sleep education
Sleep hygiene
Pediatrics
Adolescence
Obesity
Circadian rhythms
Metabolism
Diabetes mellitus

SUMMARY

The prevalence of childhood obesity has increased significantly in recent years. Obesity is associated with a range of adverse physiological, psychological and social outcomes and places a huge economical burden on healthcare systems around the world. Insufficient sleep duration is common in adolescents and exacerbated by contemporary lifestyles, but may be a contributor to obesity onset and metabolic disruption. We briefly review the current evidence surrounding the associations between sleep and obesity as well as diabetes. Sleep improvement programs have been suggested as a potential avenue to raise awareness of the importance of sleep and ultimately enhance sleep behaviors/routines. A review of the current literature supporting the efficacy of such programs is tentative. Furthermore, very few studies have investigated if sleep enhancement has downstream positive effects on metabolic function or body weight in adolescents. We highlight biological and social factors that intensify sleep loss in adolescents and recommend that these be targeted components in future interventions aimed at improving adolescent sleep.

Introduction

The global economic burden of obesity is estimated at $2 trillion, accounting for almost 3% of total healthcare costs [1]. The rising prevalence of obesity and type 2 diabetes mellitus in pediatric populations are of particular concern [2]. The World Health Organization (WHO) estimates that there are 42 million overweight children under the age of 5 y [3]. Previously, type 2 diabetes diagnoses were confined to older age groups, but type 2 diabetes is now increasingly observed in pediatric populations [2]. Persistence of pediatric overweight/obesity into adulthood has been observed [4]. Whilst there is a complex genetic contribution to metabolic disease [5], lifestyle behaviors have driven the rising prevalence of obesity and diabetes and have been the main target for interventions aiming at minimizing these conditions [6–8]. It is well established that positive energy balance (excessive energy intake and insufficient energy expenditure) is a major contributor to diabetes and obesity [5]. Sleep has, however, emerged as a tertiary lifestyle factor. Educating individuals about the importance of sleep has been suggested [9–12], particularly as a potential avenue to address adolescent obesity [13,14]. There is some literature available regarding the efficacy of sleep improvement programs and the potential influence upon addressing obesity and metabolic regulation, which is the primary focus of this review.

Sleep is a complex phenomenon that is regulated by two mechanisms named process S (homeostatic drive) and process C (circadian drive) [15]. The homeostatic drive is appetitive with sleep drive increasing with greater wakefulness. The accumulated sleep debt, developed during the day, is paid off during sleep, when the homeostatic drive is maximal. The circadian drive determines timing of sleep ensuring wakefulness in the evening, where the homeostatic drive signals the need for sleep, and in the morning on awakening to support alertness. In adolescence, sleep-

Abbreviations: BMI, body mass index; PSQI, Pittsburgh sleep quality index; SE, sleep efficiency; SOL, sleep onset latency; SWS, slow wave sleep; TIB, time in bed; TST, total sleep time.

* Corresponding author. Research Division, Weill Cornell Medicine in Qatar, Qatar Foundation-Education City, PO Box 24144, Doha, Qatar. Fax: +974 4492 8970.
E-mail address: staheri@me.com (S. Taheri).

http://dx.doi.org/10.1016/j.smrv.2016.08.004
1087-0792/

wake patterns are shifted and delays in sleep initiation and wakefulness are driven by multiple intrinsic and extrinsic factors (see Table 2). Extreme misalignment of process C has been simulated under laboratory conditions in at least two small studies of young adults [16,17]. Both studies highlighted adverse metabolic outcomes that could contribute to the onset of obesity [17] and type 2 diabetes [16]. Whilst the shift in process C, commonly observed in developing adolescents, is less extreme compared to that simulated in the studies described, a small but chronic shift in process C may have meaningful cardio-metabolic consequences. Recent longitudinal data have confirmed that persistent later bedtimes across adolescence are linked to subsequent increases in body mass index (BMI) and an increased risk of obesity [18]. Interventions to minimize delays to process C and realignment of the two processes involved in sleep can improve this behavior in adolescents.

In this review, we briefly outline the evidence surrounding the relationship between sleep parameters (sleep duration, sleep quality [sleep efficiency], and sleep architecture, specifically slow wave sleep [SWS]) and metabolic health outcomes with mechanistic explanations. We then discuss the preliminary evidence relating to the efficacy of sleep education programs, for improving sleep duration as a potential avenue to address metabolic health and disease. Finally, we discuss limitations of the existing studies and provide recommendations for future research when considering the development, design and application of such programs for delivery to adolescent populations. We highlight the importance of intrinsic and extrinsic drivers of sleep loss in adolescence, and suggest these to be key targets in future interventions aimed at improving sleep and ultimately metabolic health in this vulnerable age group.

Evidence of a sleep-obesity association in pediatrics

Obesity is a complex disease arising from multiple direct and indirect factors (see Fig. 1). Adolescents are susceptible to developing obesity and are vulnerable to its effects [19]. Many adolescents fail to achieve sufficient sleep quantity [10,11] and this, as well as poor sleep quality and late circadian preference has been linked to a plethora of adverse health outcomes and metabolic disruption [9,20–22]. Two early epidemiological studies reported a sleep-obesity relationship in children [23,24]. Whilst these studies highlighted an important novel relationship between sleep and obesity, which indicated sleep as a fundamental behavior for pediatric health, weaknesses of these, and similar studies, include: 1) use of parental report for sleep duration, which may be inaccurate [25]; 2) use of BMI to determine excess adiposity, which may be problematic, particularly in children where physical development occurs on a wide spectrum and is confounded by children entering puberty at younger ages than before [26]; 3) lack of examination of temporal associations from cross-sectional study designs [23,24]; and 4) study design for purposes other than examining the role of sleep in health. Some of these drawbacks have now been addressed [9,27,28] and have provided further evidence to support a link between sleep loss and obesity in pediatric populations. Furthermore, meta-analyses have shown that insufficient sleep in pediatrics is associated with an increased risk of obesity, ranging from 58% to 89% [29,30].

Sleep and diabetes in pediatrics

Obesity is closely linked to insulin resistance and type 2 diabetes, an increasingly common condition in pediatric populations [2]. The relationship between sleep and type 2 diabetes has previously been investigated [31–33]. Sleep architecture has been assessed in 118 children (mean age 13.1 ± 3.3 y) using polysomnography [32]. Those with greater sleep efficiency (percentage of time spent sleeping of the total amount of time spent in bed) and longer sleep duration had significantly lower 2-hour glucose levels, after adjustment. Insulin sensitivity was greater in those with more SWS, where $\beta = 0.024$, $p = 0.012$ and in those with better sleep efficiency $\beta = 0.013$, $p = 0.016$ [32]. Other groups have reported similar findings for SWS and insulin resistance/secretion in various populations (healthy weight, overweight, and obese) [31,33].

Mechanisms involved in sleep and metabolic health

The suprachiasmatic nucleus regulates sleep-wake timings and disruption to these patterns, whether in an experimental setting or undertaken voluntarily in the natural environment, may influence biological oscillations of the appetite-regulating hormones and feeding behaviors. In particular, leptin levels in healthy adults peak during sleep (between 22:00 and 03:00 h) and are lower during the day (08:00–17:00 h) [34]. Conversely, ghrelin levels are lower during sleep, peaking pre-prandially and decrease after energy intake [35].

Disruption to these hormones occurring from experimental sleep loss have been linked to an increased appetite for energy-dense foods in adults [20]. Whilst Beebe and colleagues examined changes in food intake following experimental sleep reduction in adolescents, metabolic hormones were not assessed [36]. The authors applied a cross-over study design with experimental sleep restriction (6.5 h/night monitored by wrist actigraphy) in mid-adolescents (aged 14–16 y) and reported a significantly increased number of self-reported daily sweet/dessert portions, and foods with a higher glycemic load, compared to when adolescents were in the 'healthy' sleep condition (10 h) [36]. Further recent work by the same group, revealed consistent findings showing an elevation in calorie intake following sleep restriction [37].

Sleep loss has also been associated with reduced glucose tolerance (40% reduction in glucose clearance after an intravenous glucose tolerance test following sleep restriction [four hours for six nights] versus sleep recovery [12 h for six nights]) as well as significant elevations in evening cortisol levels in adults [38]. In the normal state, cortisol levels are lowest in the evening. Alterations in sleep, a predominantly brain phenomenon, impinge on peripheral hormones via the autonomic nervous system, regulated by the hypothalamus. Sleep deprivation results in over-activation of the sympathetic nervous system [38], purported to promote insulin resistance and obesity-driven metabolic syndrome [38,39].

Mechanistic studies examining neural responses of sleep reduction upon appetite in young adults have shown that specific brain regions, known for appetite regulation and food desirability, are also affected by sleep loss [40]. Alterations to these brain regions may mediate changes in desirability and selection of unhealthy food types [40]. In turn, these behaviors can promote weight gain and subsequent obesity particularly if sleep loss persists and becomes habitual.

Effectiveness of sleep improvement programs

A consistent negative linear relationship has been observed between sleep quantity and obesity in pediatrics [9,23,24,28], with supporting prospective [28,41–43] and meta-analytic evidence [29,30]. Inter-related factors surrounding sleep, obesity and metabolic disruption are now well understood, but the question now is, if delivery of sleep improvement programs is an effective tool for improving sleep and ultimately resolving obesity and/or improving metabolic health.