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RESEARCH ARTICLE

SLEEP RESTRICTION AND ITS ASSOCIATION WITH ADOLESCENT OBESITY: A REVIEW

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ABSTRACT

Adolescence typically describes the years between ages 13 and 19 and can be considered the transitional stage from childhood to adulthood. Optimal sleep is needed to promote good health in children and adolescents. Inadequate sleep is seen as a consequence of "modern life". The present review discusses role of short sleep duration as an independent risk factor for obesity and weight gain. A literature search was conducted for all articles published between 2000 and 2016 using the key words "sleep restriction" and ("duration" or "hour" or "hours") and "obesity" or "weight" in the PUBMED database. Additional references were identified by reviewing bibliographies. Studies reporting the association between sleep duration and at least one measure of weight were included. Sleep restriction increased desire for (high-calorie) food choices, particularly increased intake of carbohydrates and fat, and cravings for calorie-dense foods (which are higher in glycaemic index and glycaemic load) suggesting sleep deprivation leads to weight gain making these foods more rewarding. Adequate sleep in adolescence would be the preventive factor for obesity.

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INTRODUCTION

School-aged children (6-12 years of age) average approximately 10-11 hours of sleep in a 24-hour period. Research studies suggest that adolescents generally require about 9-9.25 hours of sleep per night; however, many are getting less than 8 hours of sleep per school night. Sleep duration changes with age and reflects alterations in physiology and health. (Medscape, 2005) Our bodies require sleep in order to maintain proper function and health. Sleep efficiency is defined as the percent of time spent in bed asleep. Sleep restriction means limiting time in bed to the actual amount of sleep obtained in order to increase sleep efficiency (Cline, 2009). Insufficient sleep in teenagers is associated with increased risk of self-harm, suicidal thoughts, and suicide attempts, besided increasing the risk of accidents, injuries, hypertension, obesity, diabetes, and depression. (Paruthi S etal 2016)Inadequate sleep is seen as a consequence of "modern life," associated with technologies of the time. (Matricciani L *et al*. 2012).

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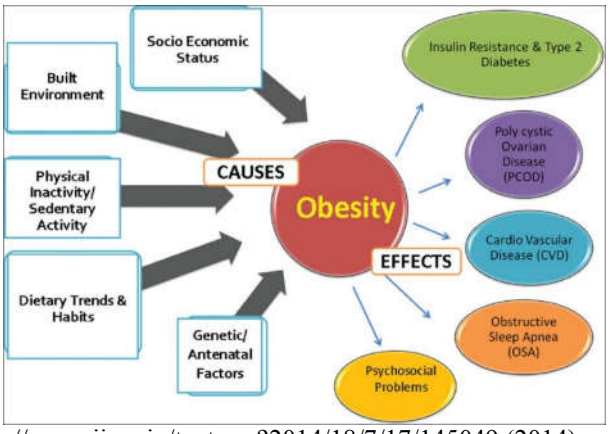
Chronic sleep loss in adolescence is a serious threat to the academic success, health, and safety of our nation's youth and an important public health issue**.** Factors affecting are electronic media use, caffeine consumption, and health-related consequences, such as depression, increased obesity risk, and higher rates of drowsy driving accidents. (Owens, 2014). Insufficient sleep is a putative risk factor for weight gain. Increases in hunger and food intake in sleep-deprived individuals exceed energy demands of extended wakefulness and therefore involve eating for pleasure rather than to fulfil a caloric need. (Hanlon *et al*., 2016). Childhood obesity is a global phenomenon affecting all socio-economic groups, irrespective of age, sex or ethnicity. Many co-morbid conditions like metabolic, cardiovascular, psychological, orthopaedic, neurological, hepatic, pulmonary and renal disorders are seen in association with childhood obesity. (Raj *et al*. 2010) Members of the American Academy of Sleep Medicine developed consensus recommendations for the amount of sleep needed to promote optimal health in children and adolescents using a modified RAND Appropriateness Method.

Table no 1. Obesity and its effect



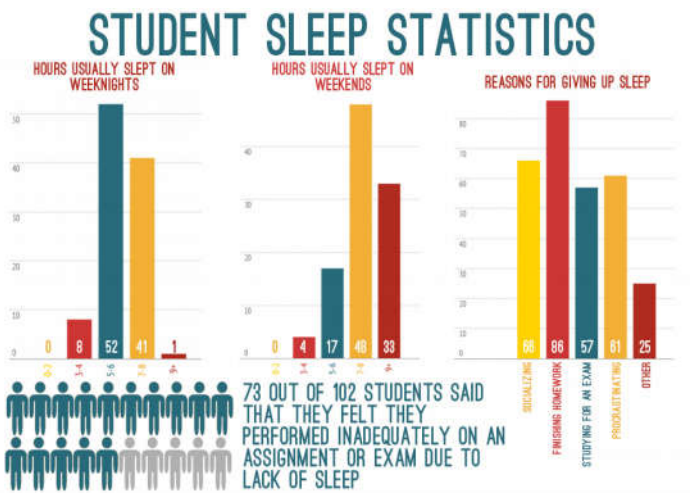
http://www.ijem.in/text.asp?2014/18/7/17/145049 (2014)

After review of 864 published articles,by (Paruthi S etal, 2016)recommended sleep durations as:

- Infants 4 months to 12 months- 12 to 16hours per 24 hours (including naps)
- Children 1 to 2 years- 11 to 14hours per 24 hours
- Children 3 to 5 years- 10 to 13hours per 24 hours
- Children 6 to 12 years- 9 to 12hours per 24 hours
- Teenagers 13 to 18 years- 8 to 10hours per 24 hours

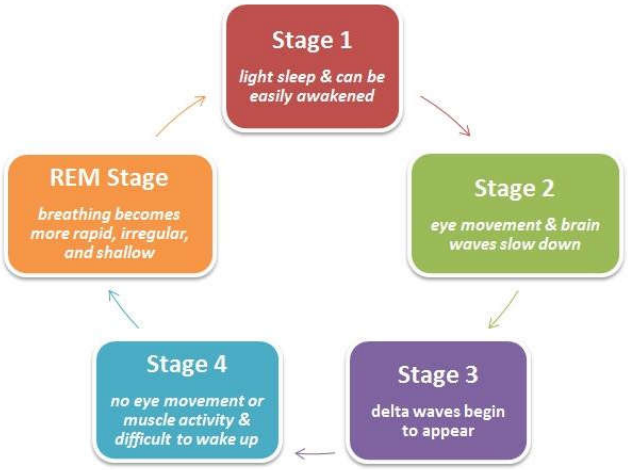
The data that are available for this age group (10 to 17 years) are dominated by the observation that they go to bed considerably later in the evening (particularly if they have access to television or live in latitudes where summer evenings can be very long) and so tend to be sleep-deprived on school days. At weekends, they catch up on lost sleep by extended time spent in bed (lie-ins). At the weekends also, the melatonin rhythm is phase delayed compared with during the week. Although most of the students were neither morning type nor evening type, that is, they were 'intermediate type', the scores decreased significantly with age, indicating a change towards becoming evening type (boys: r = -0.99, $P < 0.001$; girls: r = -0.83, $P = 0.041$, Pearson's correlation coefficients). Female students older than 10 years tended to be more evening types than their male counterparts, but this difference decreased with age and was reversed in student's aged18 years old. This change in female MEQ scores at around 10 years might relate to menarche (Waterhouse *et al*. 2012).

Table no.2 Students Sleep Statistics



Sleep is not homogeneous, and this has been investigated by recording surface electrical activity on the scalp using an electroencephalogram (EEG). In normal sleep, there is an ultradian rhythm of cycling between slow wave sleep (SWS) and rapid-eye-movement (REM) sleep stages, the REM-non REM cycle. This cycle lasts approximately 90 minutes, about five cycles occurring during the course of a normal night's sleep. The composition of successive cycles varies, with the amount of SWS decreasing and the amount of REM sleep increasing. (Waterhouse *et al*. 2012). First comes non-REM sleep, followed by a shorter period of REM sleep, and then the cycle starts over again. Dreams typically happen during REM sleep. During the deep stages of NREM sleep, the body repairs and regrows tissues, builds bone and muscle, and strengthens the immune system (Blahd, 2016).

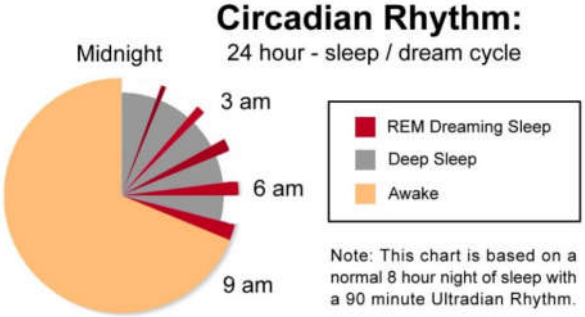
Table no. 3. Stages of sleep



https://www.europeanbedding.sg/blogs/articles/103744774-learn-how-to-sleep-better-by-understanding-sleep-cycles-stages

ActiGraph's innovative actigraphy monitoring system delivers 24-hour, real world information about sleep/wake behavior, circadian rhythms, and daytime physical activity. With its ability to capture high quality continuous sleep/wake data for days and weeks at a time, the actigraph system is a cost-effective and non-invasive in-home monitoring solution that offers a level of accuracy and reliability that cannot be achieved through subjective patient reports. (SL-1). Cortisol is the end product of the hypothalamic-pituitary-adrenal (HPA) axis, and its endogenous release is regulated by corticotrophin-releasing hormone (CRH), produced by and released from the paraventicular nucleus and corticotrophin (ACTH) released from the pituitary.

Table no. 4 Circadian Rhythm



http://www.youcanluciddream.com/about/dreaming-and-the-sleep-cycle (2012)

AUTHOR (YEAR) TITLE NAME OF THE JOURNAL	METHODOLOGY	MAJOR FINDINGS AND CONCLUSION OF THE STUDY
Dean W. Beebe etal(2013) Dietary Intake Following Experimentally Restricted Sleep in Adolescents Sleep. 2013 Jun 1; 36(6): 827–834	64 participants,healthy adolescents 14-16 years, completed a 3-week experimental sleep manipulation protocol,they were told to arrive at the study location by 8:30am; this became the rise time during all 3 weeks. Week 1 (baseline) was designed to stabilize sleep patterns.During weeks 2 and 3, participants' prescribed bedtimes on Monday-Friday nights were systematically changed to allow 6.5 h in bed (sleep-restriction condition, SR) versus 10 h in bed with lights out and all phones and electronic screens off (healthy sleep duration, HS).	Sleep was monitored for all nights of the study.Each participant wore on the non-dominant wrist a Micro Motionlogger Sleep Watch (Ambulatory Monitoring Incorporated, NY), which collected data on movements (Zero-Crossing Mode) in 1-min bins.To find nightly sleep onset,offset, and total sleep period (rise time minus sleep onset time).A 24-h diet recall was conducted after both experimental sleep conditions. Compared with dietary intake following several nights of nearly 9 h of sleep, adolescents' diet were characterized by higher glycaemic index, glycaemic load, and greater consumption of calories and carbohydrates. Sleep restriction caused increased consumption of desserts and sweets.
Hart CN etal(2013) Changes in children's Sleep Duration on Food Intake, Weight, and Leptin. Paediatrics. 2013 Dec;132(6):e1473-80	37 children, 8 to 11 years of age (27% overweight/obese) completed a 3-week study. Children slept their typical amount at home for 1 week and were then randomized to either increase or decrease their time in bed by 1.5 hours per night for 1 week, completing the alternate schedule on the third week. Primary outcomes were dietary intake as assessed by 24-hour dietary recalls, food reinforcement (i.e., points earned for a food reward), and fasting leptin and ghrelin. The secondary outcome was child weight.	Participants achieved a 2 hour, 21 minute difference in the actigraph defined sleep period time between the increase and decrease sleep conditions ($P < .001$). Comparison reported consuming an average of 134 kcal/day less ($P < .05$), and exhibited lower fasting morning leptin values ($P < .05$). Measured weights were 0.22 kg lower during the increase sleep than the decrease sleep condition ($P < .001$). There were no differences in food reinforcement or in fasting ghrelin. Compared with decreased sleep, increased sleep duration in school-age children resulted in lower reported food intake, lower fasting leptin levels, and lower weight.
L Mc Donald etal (2015) Nighttime Sleep Duration and Hedonic Eating in Childhood International Journal of Obesity (London). 2015 Oct; 39(10): 1463–1466.	Data were from 1008 families who had provided complete information on their children's sleep and appetite at this age, Nighttime sleep was calculated from parent-reported bedtime and wake time at age 5 years. Study assessed FR (food responsiveness) with the FR scale of the Child Eating Behaviour Questionnaire. We also included the SR (sleep responsiveness) scale of the Child Eating Behaviour Questionnaire as an indicator of 'homeostatic eating'.	There was a significant linear relationship between nighttime sleep duration and FR(food responsiveness), such that shorter sleep was associated with higher FR at age 5 years. There was no significant association between nighttime sleep duration and SR. It can be concluded that shorter sleep is associated with higher FR but not with SR. FR is part of the pathway mediating the effect of shorter sleep on adiposity.
Bridget Morrissey etal (2016) Sleep Duration and Risk of Obesity among a sample of Victorian School Children BMC Public Health. 2016; 16: 245.	A random sample of 156 primary schools were invited to participate, they measured Anthropometric measures (height, waist circumference, BMI), Sleep duration was taken where sufficient sleep (10 or more hours per night)and less than this is insufficient sleep.	Children with insufficient sleep are more likely to be categorised as being overweight or obese, less physically active and more sedentary was partly supported. One third of participants were categorised as insufficient sleepers. Among this sample sleep duration was inversely associated with weight status, though not between objectively measured PA (Physical activity) and ST (sedentary time). Insufficient sleep was significantly higher among children with a computer/EGD (electronic gaming device) in their bedroom.
Erin C. Hanlon etal(2016) Sleep Restriction Enhances the Daily Rhythm of Circulating Levels of Endocannabinoid2-Arachidonoylglycerol Sleep. 2016 Mar 1; 39(3): 653–664.	Healthy non obese men and women between the ages of 18 to 30 y. Participants were limited to sedentary activities during waking hours and were housed in a private room. No naps were allowed. In both conditions, irrespective of sleep/wake time, only dim light was allowed in the subject's room from wake until 10:30 and again from 18:00 until bedtime. Cortisol, leptin, and ghrelin were assayed, whereas CBs (cannabinoid)were assayed at 60-min intervals. Caloric content of the diet was calculated. Sleep was recorded by polysomnography.	All demonstrated a robust effect of insufficient sleep on hormones. These differences in the 24-h profiles of 2-AG(2-arachidonoylglycerol) and 2-OG (2-oleoylglycerol) between the two sleep conditions occurred. The well documented circadian rhythm of plasma cortisol levels, with a nocturnal nadir and a morning peak, was present in both sleep conditions. The study of sleep restriction versus normal sleep examined the hypothesis that increases in hunger, appetite, and food intake, in a state of sleep debt, are associated with increased activity of the eCB(endocannabinoid) system.
Stacey L. Simon etal(2015) Sweet/Dessert Foods Are More Appealing to Adolescents after Sleep Restriction PLoS One. 2015; 10(2): e0115434.	The 3-week sleep protocol was followed 'Teens' circadian phase was first stabilized in a baseline week, during which they could self-select their bedtimes, but had to come to our office for an 8:30 AM visit. The outcomes for this study obtained via a computerized food-appeal rating-system. Two matched sets with 84 photos of 42 sweets/desserts and 42 non-sweets (fruits/vegetables, meat/eggs) were compiled. One set was viewed at each experimental week visit and teens were asked to rate how appetizing each picture looked on a 1–4 scale ("gross," "OK," "good," "delicious"). Teens rated their hunger.	Sleep manipulation affected food appeal and hunger ratings. Images of sweets/desserts were rated more appealing but the effect of sleep manipulation was non-significant for self-reported hunger and the appeal of non-sweet foods. SR (Sleep Restriction) particularly increases intake of carbohydrates and fat, and cravings for calorie-dense foods, suggesting sleep deprivation makes these foods more rewarding.

Continue……

Stephanie M. Greer etal(2013) The Impact of Sleep Deprivation on Food Desire in the Human Brain National Community. 2013; 4: 2259	Twenty-three healthy participants, Participants completed two experimental sessions 1) a night of normal sleep in the lab monitored by PSG and 2) a night of total sleep deprivation monitored by lab personnel from 9pm and wrist actigraphy. Participants ate according to their normal diet throughout enrolment. During the sleep deprivation night, participants were provided with a controlled snack from 2:30–3:00am.	Sleep loss may lead to the development and/or maintenance of obesity through the potentially maladaptive selection of foods carrying obesogenic (weight-gain) potential, thereby explaining the large-scale significant association between reduced sleep time and obesity reported in population level studies. Sleep deprivation induced a concomitant behavioral profile of increased desire for weight-gain promoting (high-calorie) food choices.
Eliane A. Lucassen etal (2014) Sleep Extension Improves Neurocognitive Functions in Chronically Sleep-Deprived Obese Individuals PLoS One. 2014; 9(1): e84832.	Sleep duration was assessed by two-week sleep diaries and by concomitant usage of wrist actigraphyvia recording gross locomotor activity in one-minute epochs.	Study hypothesize that compensatory ability of brain areas devoted to attention may be limited in obese, sleep-deprived individuals, resulting in a larger impairment compared to non-obese, sleep-deprived individuals. Chronically sleep deprived obese individualexhibit substantial neurocognitive deficits that are partially reversible upon improvement of sleep in a non-pharmacological way.
A. N. Vgontzasetal(2007) Daytime Napping after a night of Sleep Loss Decreases Sleepiness, Improves Performance, and Causes Beneficial Changes in Cortisol and Interleukin-6secretion American Journal of Physiology, 3 January 2007 Vol. 292 no. 1, E253-E261	Forty-one normal sleepers (20 men, 21 women) 18–30 years participated in a sleep deprivation experiment that lasted 7 days. Adequate sleep time and regular sleep schedules were verified with a sleep log and actigraphy for 2 weeks before the study. After four consecutive nights in the sleep laboratory (1 adaptation and 3 baseline nights), the subjects were deprived of sleep during the entire fifth night, whereas they were allowed to sleep again on nights 6 and 7 (recovery nights) the subjects were ambulatory. Also, they were instructed not to change their diet, and their three daily meals were at about 700, 1200, and 1800.	The results suggest that a 2-h mid-afternoon nap after a night of sleep loss restores, to a significant degree, alertness and tends to improve, to a lesser degree, performance. These changes are associated with parallel increases in cortisol levels and decreases in IL-6 levels, thus both promoting alertness and better performance. A 2-h mid-afternoon nap improves alertness and, to a lesser degree, performance and reverses the effects of one night of sleep loss on cortisol and IL-6 secretion.
Jonathan Liberzon etal(2008) Naturalistic Stress and Cortisol Response to Awakening: Adaptation to Seafaring Psycho neuro endocrinology. 2008 Aug; 33(7): 1023–1026.	31 subjects (18 females) enrolled from 18 to 38 years. Subjects provided saliva samples on six separate mornings, at zero, 30 and 45 minutes after awakening. Subjects were divided into three groups or “watches”, assigned to three rotating, offset shifts (morning, mid-day and night) to provide round-the-clock coverage of sailing duties.	A clear increase in salivary cortisol was detected after awakening in the majority of samples. No differences were found between men and women. CRA(Cortisol Response to Awakening) were not correlated with sex, sleep parameters or subjective stress and control. CRA was greater during onshore workdays compared with weekend days.
J-P Chaput etal (2015) Associations between Sleep Patterns and Lifestyle Behaviors in Children: an International Comparison International Journal of ObesitySupplement. 2015 Dec; 5(Supplement 2): S59–S65.	Included 5777 children aged 9-11years. Nocturnalsleep duration (hours per night), sleep efficiency (%) and bedtime (h:min) were monitored over 7 consecutive days using an accelerometer. Lifestyle behaviors included moderate-to-vigorous physical activity (MVPA), total sedentary time (SED), self-reported screen time (ST) and healthy/unhealthy diet patterns (HDP/UDP).	Overall, participants averaged 8.8 hours of sleep with 96.2% sleep efficiency and a mean bedtime of 2218 hours. Results using categories of sleep patterns were consistent with the linear associations. Results also revealed that associations between sleep patterns and MVPA, SED and ST were significantly different between study sites, with stronger associations in high-income countries compared with low/middle-income countries. Sleep characteristics are important correlates of lifestyle behaviors in children. Differences between countries suggest that interventions aimed at improving sleep and lifestyle behaviors should be culturally adapted.
Sharon L Camhi etal(2000) Factors affecting Sleep Disturbances in Children and Adolescents Sleep medicine journal April 1, 2000Volume 1, Issue 2, Pages 117–123	Children, ages 3–14, were administered a health questionnaire which contained sleep problems, respiratory diseases and symptoms. Participants were classified as having sleep disturbances if they reported disorders of initiating and maintaining sleep (DIMS), excessive daytime sleepiness (EDS) or snoring.	The overall prevalence rates were 16.8, 4 and 22.9% for DIMS, EDS, and snoring, respectively. We found a significantly higher prevalence of DIMS in 11–14-year-old girls (30.4%) and snoring (32.3%) in 3–6-year-old boys. Study concluded that children as in adults, respiratory symptoms are associated with sleep disturbances. The increased insomnia seen in adult women may begin in early adolescence.
Andrew D. Calvin etal(2013) Effects of Experimental Sleep Restriction on Caloric Intake and Activity Energy Expenditure Chest. 2013 Jul; 144(1): 79–86.	Individuals were between 18 and 40 years, of normal weight. Subjects underwent a screening evaluation consisting of a physical examination, dietary surveys, an assessment of haemoglobin concentration, a urine pregnancy test, and an overnight polysomnogram (PSG). Subjects were allowed ad lib food and drink without restrictions. The duration was too short to reliably see changes in fat mass and weight.	Weight among those randomized to sleep restriction increased over the experimental period. Ghrelin levels remained similar between the acclimation and experimental periods in the sleep-deprived group. Modest sleep restriction in relatively young healthy individuals is accompanied by increases in caloric intake without any significant changes in energy expenditure.

Both CRH and ACTH induce increased waking, whereas increased endogenous levels of cortisol in humans have been associated with increased arousal and sleep disruption. (Vgontzas *et al*., 2007)Researches on Circadian rhythm has shown thatsleep onset will occur as core temperature falls, melatonin secretion begins, fatigue increases and alertness falls, the individual having been awake for about 16 hours; sleep will end when core temperature rises and melatonin secretion falls. (Waterhouse *et al*. 2012)

SLEEP AND ITS EFFECT ON FOOD INTAKE

An important step in the construction of a mechanistic causal model by which SR (Sleep restriction) might influence dietary choices, suggesting that SR may increase the subjective appeal of already attractive sweet/dessert to investigate food-appeal in sleep-restricted adolescents, suggesting sleep deprivation makes caloric dense foods more rewarding (Simon *et al*. 2015) The proposed shift in hedonic value of sweets/desserts does not exclude other mechanisms by which SR might impact diet (e.g., circadian factors, disinhibition), an area ripe for additional work. As biological and social factors push bedtimes later, teens' schools start earlier, leading them to average 2-hours less sleep on school nights than recommended .Because adolescents establish enduring dietary patterns,and because adolescent obesity is highly persistent ,the effects of SR could have life-long consequences, even if short sleep is limited to the high school years. (Simon S etal, 2015) Thus adolescence are giving rise to obesity by night time eating habits due to electronic media use, caffeine consumption and many more factors.

Recent research has suggested that improved sleep duration may be one such target. In both children and adults, short sleep is associated with greater rates of obesity and prospectively predicts weight gain over time. In adults, acute sleep deprivation increases caloric intake, due particularly to increased consumption of carbohydrates and fat. Ratings of hunger and appetite are also greater after sleep restriction, with the largest cravings for sweet, salty, and high-starch foods. Such foods may be particularly problematic if they are high in simple sugars (high glycaemic index) consumed in large amounts (high glycaemic load). Foods with a high glycaemic index and glycaemic load lead to dramatic fluctuations in serum glucose, and have been linked to the long-term development of several chronic diseases (e.g., diabetes, coronary heart disease, gallbladder disease, some cancers) and short-term increases in hunger.

Because sleep deprivation also alters glucose regulation, it may be as important to determine what kinds of foodare linked to inadequate sleeps as it determines how much food is consumed. (Beebe *et al*. 2013). The potential link between inadequate sleep and dietary patterns takes on particular importance during adolescence due to a convergence of factors. Relatedly, adolescent obesity is a strong predictor of adult obesity and imparts an increased risk of morbidity even for individuals who lose weight later. If sleep duration influences dietary choices, the chronic sleep restriction that is endemic during adolescence, even if limited to the high school years, could have long-term effects on dietary patterns, body mass, and related morbidity. (Beebe *et al*. 2013). Unfortunately, obesity prevention and treatment efforts have tended to be least effective for adolescents. If a causal relationship is found between short sleep and unhealthy dietary choices during adolescence, this could pave the way for innovative approaches to both obesity intervention (e.g., sleep as a treatment target) and obesity prevention (e.g., delaying high school start times to lengthen adolescent sleep). The field has good correlation data; for each hour less sleep received by adolescents, there is an 80% increase in obesity risk. What is needed now is complementary experimental work to establish that short sleep, as opposed to a confounding factor, drives that association. (Beebe *et al*. 2013). Studies showing an association of sleep restriction with adolescent obesity (between 13 to 19 years) are being reviewed in table below.

Conclusion

Sleep deprivation causes weight-gain promoting (high-calorie) food choices, particularly increased intake of carbohydrates and fat, and cravings for calorie-dense foods (which are higher glycaemic index and glycaemic load) suggesting sleep deprivation makes these foods more rewarding. (Simon *et al*., 2015). Adequate sleep pattern and healthy eating habits should be adapted to avoid health complications. Additional research work is required to study the impact of sleep restriction on dietary choices.

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