Can poor sleep, body fat mass, and low physical activity contribute mutually to an increase in childhood obesity?

Sono ruim, adiposidade corporal e inatividade física podem contribuir mutuamente para o aumente da obesidade infantil?

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Alisson Luiz Aquino da Silva
Master in Physical Education
Institution: Universidade Católica de Brasília (UCB)
Address: QS 7 Lt 1, s/n, EPCT, Taguatinga - DF, CEP: 71966-700
E-mail: alissoncvpsa@gmail.com

Elaine Cristina Vieira
Post-Doctorate in Physical Education
Institution: Universidade do Estado de Minas Gerais (UEMG)
Address: Av. Padre Cleus Francis Cox, 300, Poços de Caldas – MG, CEP: 37701-355
E-mail: vieiraec@yahoo.com

Wainny Rocha Guimarães Ritter
PhD in Physical Education
Institution: Centro Universitário de Mineiros (UNIFIMES)
Address: Rua 22, Setor Aeroporto, 75833, 130, Mineiros – GO, CEP: 75830-000
E-mail: wainny@unifimes.edu.br

Suliane Beatriz Rauber
PhD in Physical Education
Institution: Centro Universitário do Distrito Federal (UDF)
Address: SGA SUL, 903, Conj E, Lt 80, Asa Sul – DF, CEP: 70390-035
E-mail: professora.suliane@gmail.com

John Eugene Lewis
Post-Doctorate in Educational and Psychological Studies
Institution: University of Miami Miller School of Medicine, Department os Psychiatry and Behavioral Sciences
Address: 1120 NW 14th Street, Miami, FL 33136
E-mail: jelewis@miami.edu

Carmen Silvia Grubert Campbell
Postdoctoral Fellowship in Health Promotion in Clinical Research
Institution: Universidade Católica de Brasília (UCB)
Address: QS 7 Lt 1, s/n, EPCT, Taguatinga - DF, CEP: 71966-700
E-mail: campbellcsg@gmail.com

ABSTRACT
Childhood obesity is a worldwide epidemic and a public health problem. Sleep is an important factor in this process, and its quality and duration are associated with metabolism, eating behavior, cognition, and psychology. Physical activity encompasses hormonal, thermoregulatory, fatiguing, and restorative processes, is a metabolic regulator of sleep, and modestly reduces body fat through elevated energy expenditure and metabolic regulation. The relationships among obesity, sleep, and physical activity in childhood need to be better understood. Physical activity regulates circadian rhythms and metabolic alterations that can improve sleep quality and minimally reduce adiposity and the incidence of obesity. Poor sleep quality is associated with higher body mass index and lower levels of physical activity, contributing to increased caloric intake through hormonal deregulation related to eating behavior. Thus, poor sleep contributes to higher energy intake and lower daily caloric expenditure through a sedentary lifestyle.

Keywords: childhood obesity, sleep, physical activity, lifestyle, chronic disease.

RESUMO
A obesidade infantil é uma epidemia mundial e um problema de saúde pública. O sono é um factor importante neste processo, e a sua qualidade e duração estão associadas ao metabolismo, ao comportamento alimentar, à cognição e à psicologia. A actividade física engloba processos hormonais, termorreguladores, fatigantes e restauradores, é um regulador metabólico do sono e reduz modestamente a gordura corporal através de um gasto energético elevado e da regulação metabólica. As relações entre obesidade, sono e actividade física na infância precisam de ser melhor compreendidas. A actividade física regula os ritmos circadianos e as alterações metabólicas que podem melhorar a qualidade do sono e reduzir minimamente a adiposidade e a incidência da obesidade. A má qualidade do sono está associada a um maior índice de massa corporal e a menores níveis de actividade física, contribuindo para o aumento da ingestão calórica através da desregulação hormonal relacionada com o comportamento alimentar. Assim, um sono de má qualidade contribui para uma maior ingestão energética e um menor gasto calórico diário através de um estilo de vida sedentário.

Palavras-chave: obesidade infantil, sono, actividade física, estilo de vida, doença crónica.

1 INTRODUCTION

Worldwide, more than 380 million children and adolescents up to the age of 19 were overweight or obese in 2016[1]. Thus, obesity is a global public health problem[2–4], increasing the incidence of associated diseases and comorbidities and reducing quality of life[2,5–7]. In Brazil, the incidence of obesity in the adult population increased by 60% in the last decade[8]. In 2015, 23.7% and 8.7% of Brazilian adolescents aged 13 to 17 were overweight (body mass index [BMI] ≥85th and <97th percentiles) and obese (BMI ≥97th percentile), respectively[9].

Obesity is multifactorial, primarily associated with lifestyle[10–15], and has direct consequences on sleep, giving rise to multiple complications. Sleep patterns outside the age-appropriate recommendation impair sleep[16], causing metabolic changes and lead to obesity. The

mechanisms involved in this process in childhood and adolescence are not fully understood.

Physical activity promotes important changes not only in maintaining body weight through energy expenditure but also plays a key role in the release of hormones important for the sleep cycle, such as melatonin and noradrenaline[17–19]. Reducing sleep hours, increasing obesity, and lowering physical activity levels are part of the health reality around the world, leading to reduced quality of life and increased incidence of associated diseases.

Thus, physical activity, in addition to positively impacting factors related to obesity, exerts a beneficial effect on sleep as well. Therefore, the objective of this literature review is to examine the relationships among sleep, body fat mass, and sedentary behavior in promoting childhood obesity.

2 METHODS

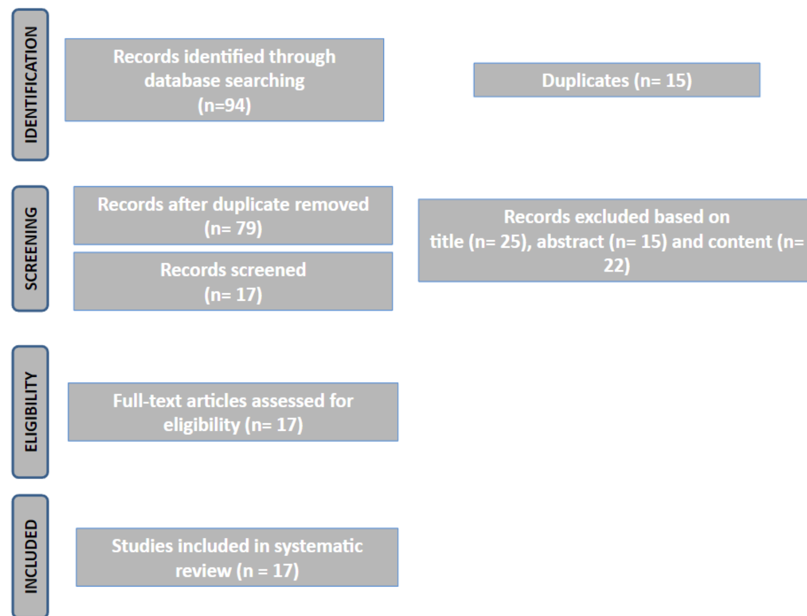
2.1 DATA SOURCES

This review consisted of a search for articles from PubMed (United States of America National Library of Medicine), DOAJ (Directory of Open Access Journals), Lilacs (Latin American and Caribbean Literature in Health Sciences), and Scielo (Scientific Electronic Library Online). The descriptors in the search from HSD (Health Sciences Descriptors) / MeSH (Medical Subject Headings) were sleep, physical activity, and childhood obesity. The descriptors were used independently or combined by AND. The search sequence was: sleep AND physical activity AND childhood obesity; sleep AND childhood obesity; and sleep AND physical activity.

2.2 STUDY SELECTION

Inclusion criteria for the articles were: (1) clinical trial or cross-sectional study in humans; (2) subjects were from birth to 18 years of age; (3) published from 2010 to 2020; and (4) written in English, Portuguese, or Spanish. In total, 94 possible articles were found, and after inclusion criteria were considered, 17 were kept in this review as described in Figure 1.

Figure 1. Consort Flow Diagram article process selection



3 RESULTS

The main findings are shown in Table 1. Poor sleep quality is associated with higher BMI and lower levels of physical activity, contributing to increased caloric intake through hormonal deregulation related to eating behavior. Thus, poor sleep contributes to higher caloric intake and lower daily caloric expenditure through a sedentary lifestyle.

Table 1. Main Findings of Seventeen Selected Articles

Author	Sample (n)	Age (years)	Body state	Results
Vézina-Im et al., 2017	228	3-5	overweight, obesity	Higher sleep duration was significantly associated with a lower z score of BMI.
Hart et al., 2013	37	8-11	eutrophic, overweight, and obesity	Higher sleep durations are associated with lower self-reported daily caloric intake, reduced fasting leptin levels, and reduced body mass.
Laurson et al., 2014	674	7-12	overweight, obesity	Children with healthy eating habits, sleep, and physical activity are less prone to obesity: 16% of boys and 9% of girls compared to 53% of boys and 42.5% of girls with unhealthy lifestyles.

Wang et al., 2017	5,518	9-12	eutrophic, overweight, and obesity	Longer sleep duration was associated with z BMI, WC; and later sleep was associated with higher z BMI, WC and body fat percentage. There was no association between sleep quality and adiposity.
Ruiz et al., 2014	90	12-17	eutrophic, overweight, and obesity	Overweight was associated with less sleep on weekdays in adolescence and higher deficit sleep and sleep debt. Low HDL cholesterol and insulin resistance were significantly associated with sleep debt. Among adolescents with a sleep deficit, the risk of being overweight was 2.7 times higher.
Pileggi et al., 2013	542	10	eutrophic, overweight, and obesity	Short sleep (≤9 h) was associated with an increase in BMI compared to normal sleep (≥9 h).
Pulido-Arjona et al., 2018	2,779	9-18	eutrophic, overweight, and obesity	Boys who had the recommended duration of sleep had a reduced risk of hyperglycemia compared to boys with long duration of sleep. In addition, in comparison to young people without sleep problems, excessive daytime sleepiness was related to low HDL-C levels in boys and high levels of TG in girls. Girls with irregular sleep patterns had reduced HDL-C levels.
Gustafsson et al., 2015	568	10-15	eutrophic, overweight, and obesity	Higher daytime sleepiness was associated with lower health-related quality of life.
Sayin & Buyulinan, 2016	108	10-15	obesity	The values of aspartate aminotransferase, alanine aminotransferase, TG, and HOMA-IR were higher in subjects who passed ≥5 h/day on screens. Children aged 10 to 13 years who slept <9 h/day were more likely to have high levels of insulin and HOMA-IR and lower HDL cholesterol levels compared with subjects who slept 9-10 h/day and ≥10 h/day. A negative correlation was found between sleep time and media time.
Zhang et al., 2018	13,001	6-10	eutrophic, overweight, and obesity	Sleep times on non-school days ≥10 h and good eating habits reduced the chances of being overweight and obese. Practicing physical activities ≥ 2 h daily on non-school days reduced the chances of being overweight and obese.
Raine et al., 2017	154	8-9	eutrophic, overweight, and obesity	After 9 months of physical activity, there was a decrease in adiposity. Children in the control group had an increase in visceral adipose tissue. Changes in visceral adipose tissue were related to changes in cognitive performance (inhibitory control) of children with obesity in the intervention group.

Carsson et al., 2016	4,169	6-17	eutrophic, overweight, and obesity	The time spent in sedentary behavior or mild physical activities was associated with risk indicators for obesity, behavioral losses, higher systolic blood pressure, reduced aerobic fitness, and cardiometabolic disorders. The time spent in moderate to vigorous physical activity was positively associated with aerobic fitness and negatively associated with risk indicators for obesity and cardiometabolic disorders. Poor sleep (≤9 h/night) was negatively associated with risk factors for obesity, behavioral impairments, and elevated systolic blood pressure.
Lin et al., 2018	433	10-13	eutrophic, overweight, and obesity	Bedtime, sleep duration, sleep timing, and sleep efficiency were not significantly associated with active outdoor play the next day. A significant association was found between active play outdoors and the following night time in bed, suggesting that each time increase in active play outdoors was associated with an increase of 4 minutes in the time in bed.
Hart et al., 2016	37	8-11	eutrophic, overweight, and obesity	A reduction in sleep duration was associated with longer time watching television and a lower level of physical activity.
Patsopoulou et al., 2016	181	13-15	overweight and obesity	At 3 months, the physical activity and diet and physical activity groups significantly reduced mean BMI, WC, systolic and diastolic blood pressure, heart rate, and cardiorespiratory capacity, respectively, while greater reductions in BMI were observed at 6 months.
Tan et al., 2017	104	5	eutrophic, overweight, and obesity	The physical activity program reduced BMI, WC, %BF, and fat mass; and reduced the rate of body mass growth of eutrophic and children with obesity. Training significantly reduced systolic blood pressure and heart rate responses during exercise in children with obesity, improving performance in cardiorespiratory fitness and lower limb strength tests; while lean and trained children improved more fitness measures.
Zehsaz, Farhangi & Ghahraman, 2016	32	9-12	obesity	Physical activity significantly decreased anthropometric, body composition, and metabolic indicators with an increase in the positive expression of omentin-1 level with a reduction of fasting insulin.

4 DISCUSSION

4.1 DURATION OF SLEEP

Reducing sleep time has recently occurred around the world, with an average loss of one hour per night among children and adolescents[2,18,19]. Simultaneously, the incidences of

sedentarism and obesity are increasing[3]. Children who sleep less are almost twice as likely to be obese[4], and sleeping less than 10 hours per night increases the risk of developing childhood obesity, particularly in boys[5].

Adequate sleep length is essential for the sequential organization of biological pathways to ensure coordinated energy processing and to reduce the risk of endocrine and metabolic diseases[20], resulting in altered ghrelin and leptin production. Sleep controls mechanisms related to ghrelin and leptin, insulin and glycemia[21], and growth hormone (GH) secretion[22]. Children who sleep longer have higher levels of health-related quality of life (HRQoL)[23]. Children 6 to 13 years of age should ideally sleep 9 to 11 hours per night[24].

4.2 METABOLIC CHANGES OF SLEEP REDUCTION

Sleep duration influences the onset of metabolic syndrome in children and young people caused by changes in fasting glucose[25,26], glucose homeostasis, and insulin concentrations[27]. Sleep duration of fewer than 9 hours per day was associated with insulin resistance according to the homeostasis model of insulin resistance (HOMA IR), a low level of high-density lipoprotein cholesterol (HDL-C), and a high level of triglycerides (TG) in children and adolescents with obesity[28,29].

Reducing sleep hours increases the risk of developing obesity in 54%[29] of children due to changes in hormonal regulatory mechanisms that do not complete their cycles in short sleep. Thus, sleep duration is a risk factor associated with the emergence and maintenance of childhood obesity. Vézina-Im et al. (2017)[30] demonstrated an association between sleep time and high adiposity ($p = 0.03$) in 35.5% of 5 year-olds who were overweight (BMI ≥85th and <97th percentiles) or obese (BMI ≥97th percentile). Higher sleep durations were associated with lower BMI z scores, going to sleep earlier, and lower caloric intake at dinner. The same relationship was found by Pillegi et al. (2013)[31] demonstrating a significant association between obesity and chronic sleep impairment in prepubertal children with a significant increase in BMI in children with short sleep duration.

Adequate sleep duration ensures normal hormonal regulation of hunger via ghrelin-leptin and provides better food standards related to health and healthy eating. Hart et al. (2013)[32] demonstrated that sleeping for more than 9 hours in childhood is associated with lower self-reported caloric intake, reduced circulating leptin concentrations, and lower BMI. The resulting imbalance between the regulation of hunger and satiety triggers unhealthy eating behaviors coupled with a sedentary lifestyle, increasingly contributing to obesity. The deposition and distribution of body fat from high daily caloric intake predominant among obese children are

potentiated by short sleep, due to incomplete regulatory hormonal mechanisms[18,19], which shift metabolism to lipogenesis, thereby increasing body fat percentage (%BF) and total circumferences, while changing the dietary pattern. Wang et al. (2017)[33] demonstrated an inverse association between sleep duration and greater BMI z score, waist circumference (WC), and a proportional association between going to sleep later and greater BMI z score, WC, and %BF.

The accumulation of lost hours of sleep related to daily sleep needs is called sleep deprivation (DepS)[34]. Sleep deficit (DefS) is defined when the amount of sleep is below the 10th percentile of recommended nocturnal sleep duration for age[35]. Loss of sleep cannot be compensated, making it impossible to replace sleep hours later. DepS and DefS are contributing factors for disorders and disrupt regulatory hormonal mechanisms in sleep. Thus, maintaining sleep duration recommendations is important for reducing the risk of developing and worsening obesity and associated diseases. Sleeping less than the recommendation increases the risks of developing obesity and associated diseases by hindering the proper functioning of the physiological mechanisms of deep sleep.

Ruiz et al. (2014)[36] demonstrated that overweight adolescents had significantly higher DefS and DepS rates than eutrophic ones. DepS was associated with a lower serum HDL-C and IR, with a 3.9-fold higher risk of developing abdominal obesity. Among adolescents with DefS, the risk of being overweight was 2.7 times higher. Sleep duration of less than 9 hours per night, which is considered inadequate, results in metabolic and endocrine changes that increase the risk of obesity, type 2 diabetes, metabolic syndrome, and inflammation. Additionally, these changes increase %BF and possibly raise subcutaneous and visceral fat[37], which are all related to higher metabolic risks, poorer sleep quality, and reduced level of physical activity.

The reduction of sleep duration can lead to metabolic and hormonal dysregulation, favoring the ingestion of foods with high caloric value[38], obesity, and cardiometabolic risk. A short sleep time alters the secretion profiles of hypothalamic hormones resulting in changes in leptin and ghrelin concentrations, thus decreasing satiety responses[39]. Increased opportunities for intermittent eating and staying awake longer, associated with a sedentary lifestyle and fatigue due to poor sleep, also contribute to the obesity epidemic[40,41].

Evidence shows the relationship between short sleep duration, low food quality, and appetite-related hormonal changes in children[32]. Hormonal changes can induce the intake of high-calorie foods and consequently generate weight gain[42,43]. In addition, sleep disorders cause a reduction of physical activity during the day, taking into account the fatigue caused by prolonged wakefulness, thus reducing daily energy expenditure, which together with unhealthy

eating habits[38,42] contribute to the emergence and maintenance of obesity and sleep problems.

4.3 PHYSICAL ACTIVITY, SLEEP, AND OBESITY

The effects of physical activity on sleep are not yet fully understood, as many theories are postulated in the literature. The main theories proposed include thermoregulation and body restoration after physical activity[17].

The increase in body temperature caused by physical activity favors optimal performance, since the muscular system requires adequate temperature for certain functions and local metabolism. Simultaneous to circadian regulation, a corporal cooling occurs, which is initiated after physical activity[44,45]. The sleep-wake cycle that is controlled by the central clock located in the hypothalamus[46] is directed by circadian cooling, reducing nocturnal body temperature and releasing melatonin, a sleep-regulating hormone. Thus, the adaptations due to physical activity contribute to the deepening of sleep and its restorative processes.

Deep sleep is the period where metabolic regulation and homeostatic restoration are performed[18,19,47]. Physical activity promotes physiological fatigue through metabolic and endocrine adaptations during stress, causing reserves of substrates to be diminished or exhausted, thus necessitating restoration that occurs during sleep[44,45]. Carson et al. (2016)[48] demonstrated that longer sleep periods and moderate or intense physical activity levels are associated with the reduction of obesity-related risk markers, with high-intensity physical activity associated with cardiometabolic risk reduction. Despite this, a direct relationship between physical activity and sleep has not been established. In another study, Lin et al. (2018)[49] found that low-intensity physical activities do not improve sleep components in children. Thus, the intensity of physical activity can be an important factor for better sleep patterns.

On the other hand, sleep quality and duration affect daily levels of physical activity. Recovery, restoration, and metabolic regulation during sleep are related to disposition and well-being. Less sleep is associated with a lower level of physical activity[50], resulting in increased discomfort and sedentarism.

Clarification about how physical activity may relate to sleep quality and the relationship of each with reduced risk of obesity is needed. It is known that thermal regulation in response to recovery from physical activity followed by cooling associated with fatigue are mechanisms that improve the quality of sleep [44,45]. Furthermore, the health of children with obesity will get better as their physical activity level increases and their hormonal and metabolic responses during sleep improve.

Studies show that consistent long-term physical activity reduces adipose tissue and improves cognitive performance in obese children. Cognitive improvement was directly related to gains in inhibitory control, while staying sedentary was associated with an increase in adipose tissue, especially visceral[37].

Physiological adaptations occurring during physical activity promote the increase of energy expenditure and metabolism, thus reducing the deposition of body fat[51,52]. A physically active lifestyle in childhood reduces the risks of developing obesity[53] and cardiovascular and endocrine diseases, besides being related to the best quality of sleep. Thus, the frequency and intensity of physical activity can be determining factors in the process of health maintenance and disease prevention.

The intensity of physical activity plays an important role in acute and long-term physiological adaptations. Tan et al. (2017)[54] observed that children engaged in physical activity programs at 50% of heart rate reserve, five times a week for 10 weeks improved physical fitness, reduced anthropometric and cardiovascular parameters, and attenuated the rate of increase in body mass. Similarly, Patsopoulou et al. (2017)[55] observed that three weekly 45-minute sessions of physical activity for 12 weeks resulted in significant improvements in anthropometric and cardiovascular parameters besides increased physical fitness.

In addition to these benefits, metabolic changes caused by physical activity contribute to the secretion of substances related to adipose tissue. Among these, omentin-1, an adipokine secreted by visceral adipose vascular stroma cells, paneth cells from the intestine, and endothelial cells[56,57], increase the insulin transduction signal, improving transport and uptake of glucose stimulated by insulin, contributing to the regulation of carbohydrate and lipid metabolism[57]. In a study of the effects of physical activity on serum omentin-1 levels, Zehsaz, Farhangi, and Ghahramani (2016)[58] demonstrated significant reductions in body mass, BMI, WC, %BF, fasting insulin, total cholesterol, low-density lipoprotein (LDL), and TG compared to the control group without physical activity. The physical activity program consisted of moderate aerobic activities (55% to 75% HRmax lasting 30 minutes, twice per week) and resistance exercise (two times per week for 55 minutes). At the end of the study, serum concentrations of omentin-1 significantly increased and fasting insulin significantly reduced. Changes in omentin-1 correlated with changes in BMI, WC, %BF, basal insulin, HOMA-IR, TG, and LDL in the intervention group. BMI and fasting insulin independently predicted changes in omentin-1.

Physical activity, in addition to providing improvement in alternative non-insulin-dependent mechanisms of glucose uptake, improves the insulin signaling pathway, thus

reducing serum glucose concentrations[59]. Through physical activity, the substrates that participate in the processes of fat deposition, inflammation, and inhibition of membrane receptors are reduced, resulting in less IR and greater cellular glucose uptake, thus improving metabolism[60].

5 CONCLUSIONS

The relationships between obesity and sleep have been explored, especially in adult individuals, with their mechanisms partially known, but much still needs to be clarified for all phases of life, particularly in children and adolescents. Physical activity through hormonal secretion, thermoregulation, and fatigue is considered a regulating factor of mechanisms involved in the development of obesity and the process of sleep.

Physical activity is an important resynchronizer of the circadian clock and can also contribute to a better quality and quantity of sleep, favoring better metabolic regulation during deep sleep, which is related to healthier eating behaviors and a higher level of physical activity. In this way, physical activity, in addition to providing beneficial effects on sleep, also promotes greater daily energy expenditure, metabolic balance, less fat deposition, greater uptake of circulating substrates, and reduction of inflammatory processes, thus reducing the possibility of obesity and related diseases.

How physical activity affects sleep is not widely studied, especially in childhood and adolescence. Few studies have investigated infant sleep, either in quality or quantity, and most interventions are multifactorial, making it impossible to analyze the impact of physical activity on sleep in childhood and adolescence. Sleep disorders are related to poor quality of life, and knowing that children and adolescents with poor sleep habits are more likely to develop obesity, it is very important to develop strategies to prevent this. A sedentary lifestyle reduces daily and resting energy expenditure, and when associated with an increased intake of high-calorie foods, adiposity is increased, which causes metabolic alterations and damage. However, adequate physical activity, sleeping routinely, and reducing the intake of high-calorie foods may favor better quality sleep and physiological restoration. Poor sleep quality is associated with higher BMI and lower levels of physical activity, contributing to increased caloric intake through hormonal deregulation related to eating behavior. Thus, poor sleep contributes to higher intake of energy and lower daily caloric expenditure through a sedentary lifestyle.

Despite these considerations, few studies have established the relationships among physical activity, sleep, and obesity. Increasing the awareness of the benefits of sleep, physical activity, and healthy eating for good health should be part of a multidisciplinary approach for

the prevention and treatment of childhood obesity. Further studies are needed to better understand the physiological mechanisms that connect poor sleep, sedentary behavior, and body fat mass and how physical activity can be used to improve childhood health and decrease childhood obesity.

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