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Sleep impact on obesity – literature review

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ABSTRACT

Purpose: The aim of this literature review is to analyze existing studies on the relationship between sleep and obesity, with a focus on how sleep quality, duration, and circadian rhythm disruptions influence obesity development. It also identifies biological mechanisms, such as changes in appetite-regulating hormones, and evaluates the effectiveness of sleep-related interventions in obesity prevention and treatment.

Methodology: A systematic review of scientific literature was conducted, including clinical trials, cohort studies, and meta-analyses from the last 10 years. The review examined various populations and age groups, focusing on the impact of sleep duration, quality, and circadian rhythm disturbances on obesity.

Findings: Results consistently show that poor sleep quality and short sleep duration (<6 hours) are associated with higher BMI, increased waist circumference, and visceral fat. Sleep disturbances impact hormones like ghrelin and leptin, promoting overeating. Circadian disruptions from shift work or social jetlag further contribute to abdominal fat. Sleep improvement interventions have shown promising effects on weight reduction, though long-term impact requires more study.

Conclusions: This review highlights the important role of sleep in obesity development. Poor sleep duration and quality, along with circadian misalignment, are linked to weight gain and metabolic dysfunction. While sleep-targeted interventions show potential for improving metabolic health and aiding weight loss, further research is needed to fully understand underlying mechanisms and lasting effects. Integrating sleep management into obesity prevention and treatment could offer a valuable strategy to address the global obesity epidemic.

Keywords: sleep and obesity, sleep duration, sleep quality, circadian rhythm disruptions, metabolic health, appetite-regulating hormones, obesity prevention.

Introduction

The health issues of modern societies are increasingly focused on the relationships between lifestyle and the occurrence of chronic diseases, including obesity, type 2 diabetes, hypertension, and cardiovascular diseases. One of the essential elements of a healthy lifestyle that impacts the development of these disorders is sleep.

Definitions of Key Terms

Sleep, Its Physiology, and the Relationship with Obesity

Sleep is a physiological, cyclic state of the body characterized by decreased awareness, limited sensitivity to external stimuli, slowing of vital functions, and changes in brain activity. It consists of non-rapid eye movement (NREM) sleep, which is crucial for the body's regeneration, and rapid eye movement (REM) sleep, which plays an important role in memory consolidation and emotional regulation [1]. For adults, the optimal sleep duration is 7 to 9 hours per night, while for children and adolescents, the recommendations include longer sleep: 10-13 hours for children aged 3-5 years, 9-12 hours for children aged 6-12 years, and 8-10 hours for teenagers aged 13-18 years [2].

Obesity – Definition and Classification

Obesity is defined as the pathological or excessive accumulation of body fat, which leads to impaired health and increases the risk of metabolic and cardiovascular diseases [3]. According to the WHO classification, obesity is diagnosed with a body mass index (BMI) of $\geq 30 \text{ kg/m}^2$ [4]. BMI is calculated by dividing body weight (in kg) by height (in m) squared – kg/m^2 . The BMI categories are: underweight ($< 18.5 \text{ kg/m}^2$), normal weight ($18.5\text{-}24.9 \text{ kg/m}^2$), overweight ($25\text{-}29.9 \text{ kg/m}^2$), and obesity ($\geq 30 \text{ kg/m}^2$). For children and adolescents, percentile charts are used, considering age and sex [2].

Abdominal (Central) Obesity is associated with excessive fat accumulation in the abdominal area and around internal organs. It is a significant risk factor for type 2 diabetes and cardiovascular diseases [1]. The diagnosis of abdominal obesity involves measuring waist circumference, with cut-off values of $\geq 88 \text{ cm}$ for women and $\geq 102 \text{ cm}$ for men, while in Asian populations, these values are lower: $\geq 80 \text{ cm}$ for women and $\geq 90 \text{ cm}$ for men [3].

Epidemiology of Obesity and Sleep Disorders

Epidemiological data indicate that both obesity and sleep disorders have reached the level of a global epidemic. In South Korea, within just 3.5 years, 826 out of 9,474 adult participants in the study developed obesity [5]. In the international PURE study, involving over 136,000 people from 26 countries, 27% had abdominal obesity [5]. At the same time, a reduction in sleep duration is observed. In the USA, more than 30% of adults sleep less than the recommended 7 hours [4]. In the international PURE study, it was noted that individuals who slept <6 hours and went to bed after midnight had significantly higher obesity risk, especially women [5]. In China, as many as 95% of high school students reported insufficient sleep [5]. Sleep disturbances are exacerbated by lifestyle changes, urbanization, exposure to artificial light, and shift work, which further increases metabolic risk [1].

The Relationship Between Sleep and Obesity

Studies indicate a strong relationship between sleep and obesity. Both shorter sleep duration (<6 hours per day) and poor sleep quality are associated with a higher risk of general and abdominal obesity – regardless of age and gender [3]. In the PURE study, individuals who slept less than 6 hours and went to bed after midnight had a 20-40% higher risk of obesity [5]. Mechanisms underlying this relationship include increased ghrelin levels, decreased leptin levels, increased appetite, decreased physical activity, and sleep fragmentation [3]. Sleep deprivation at a young age can cause lasting metabolic changes that promote obesity in adulthood [6].

Methodology

The research utilized a comprehensive review of existing literature, sourcing studies from databases including PubMed, Scopus, and Google Scholar. The selection criteria focused on clinical trials, cohort studies, and systematic reviews that examined the impact of sleep on obesity, with particular emphasis on sleep duration, quality, and circadian rhythm disruptions. The review included studies across various populations, including adults, children, and adolescents, from different geographical regions and cultural backgrounds. Factors such as age, sex, health status, and lifestyle behaviors were also taken into account. The findings were critically analyzed to determine the relationship between sleep disturbances and obesity, the

effectiveness of sleep-related interventions, and the potential for incorporating sleep management in obesity prevention and treatment strategies. The relevance of the findings was then evaluated for their practical applications in addressing obesity and improving metabolic health.

Impact of Sleep Duration on the Risk of Obesity Development

The Relationship Between Short and Long Sleep and General and Abdominal Obesity

Based on the analysis of available files, short sleep (≤ 6 hours per day) is consistently associated with an increased risk of both general and abdominal obesity. In the China Kadoorie Biobank study involving over 21,000 adults, it was observed that individuals sleeping ≤ 6 hours had a higher risk of significant weight gain (≥ 5 kg) and abdominal obesity, but not general obesity (BMI ≥ 30) [7]. Similar results were obtained in the multinational PURE study, involving 136,652 participants from 26 countries – sleep of less than 6 hours and late bedtimes were associated with both general and abdominal obesity, especially in individuals going to bed after 2:00 AM [5].

On the other hand, long sleep (>9 hours) shows less clear associations with obesity. In the Korean cohort study KoGES, individuals sleeping ≥ 8 hours had a lower risk of developing obesity (HR: 0.67; 95% CI: 0.53–0.85) [8]. However, other analyses do not confirm this relationship and suggest that excessive sleep may coexist with other health and lifestyle problems that also contribute to obesity [3].

Table 1. Summary of studies on sleep impact on obesity

Study name	Methodology	Sample size	Findings	Bibliography
China Kadoorie Biobank	Prospective cohort study in adults aged 30–79 years in China, assessing sleep duration and its association with general and	21,958	Short sleep (≤ 6 h) was associated with increased risk of ≥ 5 kg weight gain and abdominal obesity, especially among those with low	[7]

	abdominal obesity.		physical activity. No significant association with general obesity.	
KoGES (Korean Genome and Epidemiology Study)	Longitudinal cohort study in Korean adults investigating the association between sleep patterns, night-time snacking, and obesity development.	9,474	≥8 h sleep duration associated with reduced obesity risk. Night-time snacking and high night-time caloric intake linked with increased obesity risk.	[8]
PURE Study	Multi-country prospective study examining effects of sleep duration and timing on obesity.	136,652	Short sleep (<6 h) and late bedtime (>2 a.m.) were associated with increased general and abdominal obesity. Daytime naps ≥1 h linked to abdominal obesity.	[6]
Nurses' Health Study	Longitudinal study among U.S. nurses followed for 16 years to evaluate sleep duration and weight gain.	68,183	<5 h sleep was associated with 15% higher obesity risk and greater weight gain over time.	[9]
Kaar et al. (2020)	Prospective study tracking sleep patterns in preschool children and their effect on BMI development	301	Longer sleep at age 3 predicted lower BMI trajectory during early childhood.	[2]

Childhood Sleep and Obesity Risk (ABIS Study)	Prospective cohort study in Sweden, tracking sleep habits and BMI z-scores from age 1 to 8.	10,840	Shorter sleep duration at age 1 was linked with higher BMI z-scores later in childhood. Bedtime and sleep quality were not independently associated.	[10]
Tasali et al., 2022	Randomized controlled trial in overweight adults testing the effect of sleep extension on caloric intake and body weight.	80	1.2 h of additional sleep reduced energy intake and led to moderate weight loss.	[11]

Summary of Findings in Children and Adult Populations

In the population of children and adolescents, shorter sleep, poorer sleep quality, and irregular bedtimes were consistently associated with higher BMI, greater body fat percentage, and abdominal obesity [2,6]. Prospective studies indicate that sleep deprivation in early childhood (e.g., at age 3) may predict greater BMI gain in subsequent years [6]. In adolescents, a late chronotype and greater variability in sleep timing are also correlated with higher body fat levels [12].

In the adult population, shorter sleep duration (<6 hours) and poorer sleep quality were significantly associated with a higher risk of both general and abdominal obesity. Data from cohort studies in China and Korea confirm that individuals sleeping less have a higher risk of weight gain and abdominal obesity compared to those sleeping 7-8 hours [7,8]. Additionally, shift work and late bedtimes were independently associated with a higher risk of obesity, especially in women [5,13].

Role of Sleep Quality

The Relationship Between Sleep Quality (PSQI) and Obesity

Sleep quality, assessed using the Pittsburgh Sleep Quality Index (PSQI), is a significant factor associated with the risk of obesity, independent of sleep duration. A study conducted among Korean adults showed that women with poor sleep quality (PSQI > 5) had more than twice the risk of obesity compared to women with good sleep quality (PSQI ≤ 5), especially in the group with a poorer diet quality (RFS ≤ 21). The odds ratio (OR) for women with poor diet quality and high PSQI was 2.198 (95% CI: 1.027–4.704) after adjusting for confounding factors such as age, marital status, smoking, physical activity, and menopause [14].

In men, no significant association between sleep quality and obesity risk was observed, which may suggest gender differences in the metabolic mechanisms related to sleep. It is possible that differences in hormone levels such as leptin and ghrelin, or the varying response of the nervous system to stress and sleep deprivation, affect metabolism differently in women and men [14].

Factors That Impair Sleep Quality (Stress, Environmental Factors, Eating Habits)

Sleep quality is influenced by a variety of interacting environmental, psychological, and behavioral factors. A significant disruption to sleep is evening exposure to light emitted by electronic devices. This delays melatonin secretion, shifts the sleep phase, and disrupts the circadian rhythm, leading to shorter sleep and its fragmentation [15]. This phenomenon is particularly observed in children and adolescents, who spend more than two hours a day in front of screens, especially in the evening, which significantly worsens both the quality and duration of their sleep [1].

Environmental factors such as noise, light pollution, high bedroom temperatures, and a lack of feelings of safety also reduce sleep efficiency, increasing the risk of nocturnal awakenings and subjective feelings of insufficient sleep [4].

Psychological factors, such as chronic stress, anxiety, and depression, are strongly linked with insomnia and poor sleep quality. People experiencing emotional stress often have difficulty falling asleep, experience fragmented sleep, and report lower satisfaction with their sleep [4].

This phenomenon is particularly common in women, whose neurohormonal response to stress is more pronounced and has a stronger effect on both sleep and metabolism [4].

Evening eating habits also play a role. Consuming caffeine, alcohol, nicotine, or heavy meals before sleep leads to longer sleep latency, shallower deep sleep, and more frequent night-time awakenings [4].

Gender and Age Differences

The relationship between sleep and obesity is strongly influenced by both gender and age. Women more frequently than men experience sleep disorders such as insomnia, poor sleep quality, and frequent awakenings, which are attributed in part to cyclical hormonal changes (menstruation, pregnancy, menopause) and a higher susceptibility to stress [8]. In contrast, men more frequently show poorer metabolic parameters with comparable sleep disturbances, including a higher risk of abdominal obesity [14]. Neuroimaging studies also suggest differences in brain structure and function between genders, which may influence sleep and appetite control [8].

Gender differences are also observed at the hormonal level – women exhibit higher levels of leptin, independent of fat content, which may modulate their metabolic response to sleep disturbances [14]. In contrast, in men, poor sleep quality shows a stronger relationship with increased BMI and abdominal obesity [14].

Age also significantly affects sleep and its relationship with obesity. In children and adolescents, shorter sleep duration and irregular bedtimes, especially on school days, are strongly correlated with higher BMI, poorer diet quality, and increased consumption of processed foods [16,17]. In adolescents, a phenomenon known as "social jetlag" – a mismatch between the biological clock and the school schedule – further exacerbates circadian rhythm disruptions and negatively impacts the body's energy balance [18].

In the elderly (≥ 65 years), both sleep quantity and quality worsen with age. The amount of deep sleep decreases, the number of nocturnal awakenings increases, and sleep-related breathing disorders become more common, all of which may lead to weight gain and a higher risk of developing metabolic syndrome [4,5]. Interestingly, in older women, delayed sleep onset was particularly strongly associated with higher BMI, suggesting the role of circadian rhythm as a mediator in obesity development [5].

Physiological Mechanisms Linking Sleep to Obesity

Hormonal Mechanisms Regulating Appetite (Leptin, Ghrelin, Cortisol)

Sleep plays a key role in the hormonal regulation of appetite, and its deficiency can lead to disturbances in energy balance, which promote obesity. The three most important hormones involved in this process are leptin, ghrelin, and cortisol.

Leptin, known as the satiety hormone, is primarily secreted by adipose tissue and works by inhibiting appetite and increasing energy expenditure. Under conditions of shortened sleep, leptin levels significantly decrease, leading to a reduced satiety signal and promoting overeating [1,17].

Ghrelin, conversely, as the hunger hormone, stimulates appetite. Its levels increase during sleep deprivation, which intensifies the feeling of hunger, especially for high-calorie and sweet foods [1,3,17]. Experimental studies have shown that even a single night of sleep deprivation can significantly increase ghrelin levels and hunger in healthy individuals [19].

Additionally, sleep disturbances affect the rhythm of cortisol secretion, the stress hormone, which increases in response to the activation of the hypothalamic-pituitary-adrenal (HPA) axis. In conditions of chronic sleep restriction, elevated cortisol levels, particularly in the evening, are observed, along with a flattening of its daily rhythm, which contributes to insulin resistance, hyperglycemia, and the accumulation of visceral fat [3,17].

The interactions between leptin, ghrelin, and cortisol are complex but together lead to increased hunger, a preference for high-calorie foods, and a decrease in control over eating. In the long term, this can lead to a positive energy balance and the development of obesity [1,3,19].

Circadian Rhythms and Sleep-Related Metabolic Disorders

Circadian rhythms are endogenous, approximately 24-hour cycles regulating various physiological processes such as sleep, hormone secretion, appetite, and energy metabolism. The main biological clock is the suprachiasmatic nucleus (SCN) in the hypothalamus, which synchronizes peripheral clocks in various tissues of the body. Light is a key factor in

synchronizing the circadian rhythm [17]. Under normal conditions, metabolism, physical activity, and food intake are synchronized with the daytime phase, while sleep, fasting, and regeneration occur at night. Disruptions to the circadian rhythm—such as through shift work, irregular sleep hours, or evening eating—lead to the dysregulation of metabolic processes [1]. Studies show that circadian misalignment, i.e., performing biologically daytime activities (eating, exercising) at night, leads to increased levels of ghrelin, reduced leptin levels, and impaired insulin sensitivity [1]. Additionally, phase shifts in cortisol and melatonin secretion contribute to disturbances in blood glucose and lipid rhythms [17].

One commonly described phenomenon among adolescents is "social jetlag," which refers to the misalignment between the biological sleep rhythm and social obligations (e.g., school). Individuals with a high level of social jetlag tend to have higher BMI and consume more calories in the evening and nighttime hours [1,18]. Similar mechanisms are observed in shift workers, who sleep during the day and eat at night, leading to increased risks of obesity, type 2 diabetes, and metabolic syndrome [13,17]. Importantly, the timing of caloric intake also has a metabolic effect—those who eat mainly in the evening or at night tend to gain weight, even with a similar caloric intake, suggesting the importance of the circadian rhythm in energy metabolism [1].

Chronic disruption of the circadian rhythm leads to internal desynchronization between the central and peripheral clocks, which is considered one of the mechanisms that leads to obesity and metabolic diseases [1,17].

Role of Sleep in the Regulation of Energy Balance and Metabolism

Sleep plays a crucial role in maintaining the balance between energy intake and expenditure. Sleep restriction leads to increased appetite, particularly for high-calorie foods, which is associated with the disturbance of leptin and ghrelin levels [1,3].

Experimental studies have shown that just a few nights of shortened sleep can lead to increased caloric intake without compensatory increases in energy expenditure, resulting in a positive energy balance and weight gain [1,3].

Sleep also affects glucose metabolism—its deficiency causes reduced insulin sensitivity and glucose tolerance disorders, increasing the risk of type 2 diabetes [17]. Furthermore, disrupted

sleep is associated with elevated cortisol levels, which support the accumulation of visceral fat and insulin resistance [1,17].

The Impact of Social Jetlag on the Risk of Abdominal Obesity

Social jetlag, defined as the discrepancy between sleep patterns on weekdays and weekends, is increasingly recognized as an independent risk factor for obesity, especially abdominal obesity. Disruption of circadian rhythms affects appetite regulation and metabolism, which can lead to excessive accumulation of fat around the abdomen [1].

Population studies have shown that each additional hour of social jetlag is associated with a higher waist circumference and fat mass index in adolescents, regardless of overall sleep duration and lifestyle [18]. These mechanisms may involve altered hormonal profiles, including higher ghrelin levels and lower leptin levels, which increase hunger and the tendency to consume high-calorie meals [1].

In a study conducted among shift workers, it was shown that individuals with higher levels of social jetlag were more likely to have overweight and abdominal obesity, even after considering sleep duration and gender [13]. While some statistical models indicated a protective effect of jetlag, other analyses pointed to its significant contribution to increased metabolic risk, especially when combined with short sleep and night shifts [13].

The long-term effects of social jetlag include impaired glucose tolerance, insulin resistance, and the accumulation of visceral fat—these changes are more pronounced in adolescents and individuals with an evening chronotype. Irregular sleep and activity rhythms also lead to poorer diet quality and lower physical activity, further exacerbating the risk of abdominal obesity [18].

Interventions Aimed at Improving Sleep as a Strategy for Obesity Prevention and Treatment

Review of Interventions to Extend Sleep Duration

In response to the growing prevalence of sleep deprivation in the general population, there is increasing interest in interventions aimed at extending sleep duration as a strategy for preventing and treating obesity and metabolic disorders.

One of the most commonly used methods is behavioral educational interventions, which include elements of sleep hygiene, such as establishing a regular sleep-wake rhythm, avoiding caffeine and screen time before sleep, improving environmental conditions in the bedroom, or individual consultations with sleep specialists. In a study by Tasali et al., individuals with overweight who slept less than 6.5 hours per night experienced a two-week intervention focused on sleep hygiene, resulting in a 1.5-hour increase in sleep duration, a 4% reduction in appetite, and a 62% decrease in cravings for salty and sweet foods [3].

Technological interventions, such as mobile applications supporting changes in sleep habits, have also proven effective. In a pilot study involving individuals with hypertension, the use of digital behavioral therapy led to clinically significant increases in sleep duration and a reduction in blood pressure [20].

Studies on extending sleep in obese individuals have shown that such an approach may improve insulin sensitivity and reduce the consumption of simple sugars. For example, in a randomized study by Al Khatib et al., behavioral consultations with individuals sleeping less than 7 hours resulted in increased sleep duration and a reduction in sugar and fat intake [17].

In children and adolescents, educational and physical activity programs, such as CEMHaVi – a one-year program improving physical activity and health habits – contributed to improved sleep quality and a reduction in BMI in the intervention group [17].

Effectiveness of Interventional Programs (Health Education, Behavioral Therapy)

The growing interest in the role of sleep in regulating body weight has led to the integration of health education and behavioral therapy elements in obesity prevention and treatment programs. One example of an effective intervention is the program by Logue et al., which used a 12-week cognitive-behavioral therapy (CBT) combined with sleep education. The group that received additional support in sleep hygiene achieved a greater weight loss (average 5%) compared to the group receiving only the standard CBT components (2%) [3].

Similar results were obtained in the study by Sawamoto et al., in which overweight participants underwent a 7-month weight loss program incorporating CBT for insomnia. Although the increase in sleep duration was small (+14 minutes), participants experienced a significant weight loss (15% of initial weight), improved body composition, and an increase in adiponectin levels, suggesting metabolic benefits associated with improved sleep [20].

In another interventional study in adolescents with obesity, a four-week strategy combining caloric restriction and an additional hour of sleep on workdays was applied. This group not only lost more weight but also showed a greater reduction in waist circumference, insulin levels, and inflammatory markers compared to the control group [20].

The effectiveness of educational interventions was also confirmed in the context of children and adolescents. The CEMHaVi program, based on health education and physical activity, led to significant improvements in both sleep quality and duration, as well as obesity parameters among participants [17].

The Potential of Sleep-Improving Therapies in Weight Loss Reduction

In the study by Sawamoto et al., a 7-month lifestyle program involving overweight women included a caloric deficit, increased physical activity, and cognitive-behavioral therapy (CBT) for insomnia. Although the increase in sleep duration was modest (+14 minutes), participants experienced a 15% reduction in body weight, improved body composition, and an increase in adiponectin levels – an anti-inflammatory hormone, indicating favorable metabolic changes resulting from improved sleep quality [20].

Similarly, in the study by Logue et al., standard CBT for weight loss was compared with an extended version that included a sleep component (education and sleep hygiene). The group receiving additional sleep elements achieved greater weight loss (5%) compared to the control group (2%) [20].

In the study by Demos et al., participants who were assigned to the sleep intervention before starting the weight loss diet were instructed to improve meal regularity and extend sleep duration. After 6 weeks, participants in this group had less weight loss than the control group, but after 18 weeks, the differences equalized, suggesting the need for a long-term approach to sleep therapy in the context of weight loss reduction [20].

Similarly, data from a randomized study by Al Khatib et al. show that extending sleep through health psychologist consultations improves sleep parameters and leads to reduced consumption of sugars, fats, and carbohydrates, which may indirectly support weight control [3].

Discussion

Summary of the Relationship Between Sleep and Obesity

Sleep plays a key role in maintaining proper body weight and the functioning of the metabolic system. Sleep disturbances—both in terms of duration and quality—are significantly associated with an increased risk of developing overweight and obesity. Individuals who sleep less than the recommended 7–9 hours per day are more likely to have higher BMI, increased waist circumference, and higher levels of visceral fat [3].

The mechanisms underlying these associations are complex. Insufficient sleep affects hormonal imbalances, including decreased leptin levels and increased ghrelin levels, leading to increased appetite and food preferences for high-calorie products [3,17]. Additionally, sleep influences the activity of brain structures responsible for impulse control, which can make it more difficult to follow healthy eating habits [3].

Circadian rhythms also play a significant role in these processes. Shifts in the sleep-wake cycle—such as those caused by shift work or weekend "social jetlag"—lead to desynchronization of the circadian rhythm, which impairs glucose tolerance, reduces insulin sensitivity, and promotes fat accumulation [1,17].

The accumulated data indicate that sleep should be considered an important, yet often overlooked, component of obesity prevention and treatment strategies. Improving sleep quality and regularity—through educational, behavioral, and environmental measures—can positively affect not only body weight but also overall metabolic health [17,20].

The Role of Sleep in Obesity Prevention and Treatment

Contemporary epidemiological and clinical data indicate that both the duration and quality of sleep play a significant role in preventing and treating obesity. Reduced sleep time, irregular circadian rhythms, and poor sleep quality are factors that contribute to increased appetite, calorie intake, and metabolic changes leading to weight gain [17].

Health recommendations increasingly include sleep as an integral part of weight loss strategies. Randomized clinical trials have shown that interventions aimed at extending

sleep—such as sleep hygiene education—can lead to reduced energy intake, decreased consumption of simple sugars and fats, and increased feelings of fullness [3,19].

Similarly, among children and adolescents, educational programs that combine promoting physical activity with improving sleep habits (e.g., CEMHaVi) have shown a positive impact on obesity indicators as well as sleep duration and quality [17].

From a public health perspective, increasing awareness about the importance of sleep—both among patients and healthcare professionals—is crucial. There is a need to integrate sleep-related issues into preventive programs and daily clinical practice, especially for at-risk groups such as children, adolescents, and individuals with overweight [4,17].

Identifying Gaps in Current Knowledge and the Need for Further Research

Despite the growing interest in the effects of sleep on metabolic health and the development of obesity, there are still significant gaps in the existing knowledge that limit the ability to implement effective interventions. One of the main problems is the lack of long-term prospective studies assessing the lasting effects of sleep restriction and circadian rhythm disruptions on metabolic parameters. As noted, most existing data come from short-term studies, often experimental, with a limited number of participants and insufficient statistical power[1].

Another significant gap is the limited number of interventional studies whose primary goal is to extend sleep as a therapeutic strategy. Most existing interventions treat sleep as an auxiliary component in weight loss programs, which makes it difficult to assess its independent impact on weight reduction [17].

There is also a lack of consensus regarding optimal sleep measurement methods—most studies rely on self-reports of sleep duration and quality, which limits their reliability. More objective methods of assessment, such as polysomnography or integrated sleep monitoring devices, need to be introduced, particularly in population-based studies [9].

Additionally, there is a need for a better understanding of the biological mechanisms underlying the relationship between sleep and obesity. In particular, further research is needed on the role of circadian rhythms, gut hormones (e.g., ghrelin, leptin), gene-environment

interactions, and differences between genders and ethnic groups in their responses to sleep disturbances [1,21].

Conclusions and Recommendations

Based on available studies, it can be concluded that optimal sleep duration and quality are crucial for public health, particularly in the context of obesity prevention. It is recommended that preschool-aged children (3–5 years) sleep between 10 and 13 hours, school-aged children (6–12 years) sleep between 9 and 12 hours, adolescents (13–18 years) sleep between 8 and 10 hours, and adults over 18 years of age sleep 7 to 9 hours per day. Adequate sleep duration reduces the risk of excessive weight gain and the development of general and abdominal obesity.

Authors' contribution

All authors contributed to the article. Conceptualization-Kacper Buczek, Patrycja Zwierzchlewska; methodology-Kamil Dziekoński; software-Julia Głowacz; check-Maksymilian Wiśniowski, Klaudia Kulig; formal analysis-Dominik Stanibuła; investigation-Jakub Rybowski; resources-Kacper Buczek; data curation-Aneta Redner; writing - rough preparation-Kacper Buczek; writing - review and editing-Maksymilian Wiśniowski; visualization-Klaudia Kulig; supervision-Kacper Buczek; project administration-Michał Popiel. All authors have read and agreed with the published version of the manuscript.

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