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Authors' Affiliation:
Collegium Medicum at Jan Kochanowski University in Kielce, Poland

***Corresponding author:**
Agnieszka Kowalska,
ul. Jana Chryzostoma Paska 10/30, 25-108 Kielce, Poland; Phone: +48881583252; e-mail: aga_kowalska1@onet.eu

Contact list:

Agnieszka Kowalska	aga_kowalska1@onet.eu
Milena Kędzierska	kedzierska.milena19@gmail.com
Michał Biernacki	michalbiernacki01@op.pl
Michał Wójcicki	michal.w953@gmail.com

Orcid list:

Agnieszka Kowalska	0009-0007-1298-7788
Milena Kędzierska	0009-0008-0980-3543
Michał Biernacki	0009-0009-8083-7129
Michał Wójcicki	0009-0008-1649-5024

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The effect of sleep quality on the development of obesity and insulin resistance - a review of current data

Agnieszka Kowalska*, Milena Kędzierska, Michał Biernacki, Michał Wójcicki

ABSTRACT

Complex neuroendocrine mechanisms underlie the relationship between sleep disturbances and an increased risk of obesity and type 2 diabetes. For example, glucose metabolism is regulated differently depending on the sleep phase (NREM vs. REM), and an imbalance between them results in impaired glucose tolerance. Sleep deprivation also triggers a cascade of hormonal changes that are detrimental to body shape and metabolism. Cortisol levels rise, which is a factor that promotes the development of insulin resistance, but on the other hand, appetite hormones become deregulated: leptin (responsible for satiety) levels decrease, and ghrelin (which stimulates hunger) levels increase. As a result, even short-term sleep deprivation intensifies appetite, especially for high-calorie foods, which facilitates weight gain. Insufficient sleep acts as a stressor, activating the HPA axis, which also impairs glucose tolerance. The relationship between obesity and sleep is bidirectional. Obese individuals report poor sleep quality much more frequently (79% of subjects) than the control group (36%), and sleep duration of less than 7 hours has been linked to weight gain. Shift work is a significant risk factor, as it disrupts circadian rhythms, increasing the risk of obesity and metabolic syndrome.

Keywords: obesity, sleep quality, insulin resistance, glucose metabolism, sleep hygiene

1. INTRODUCTION

One of the chronic and complex diseases associated with excessive accumulation of body fat and leading to adverse health complications is obesity. It is a risk factor for many diseases, including type 2 diabetes, cardiovascular diseases, cancers, and problems with the skeletal system and fertility. Obesity also negatively impacts quality of life, for example, by causing sleep disturbances or mobility difficulties. The most commonly used diagnostic tool is the body mass index (BMI), which takes into account body weight and height. However, this is a surrogate measure of body fat percentage, so additional measurements, such as waist circumference, are also used for a more complete diagnosis. The problem of obesity is growing on a global scale. Since 1990, the obesity rate among adults worldwide has more than doubled, while among adolescents, a fourfold increase has been observed. Projections from the World Obesity Atlas 2025 suggest that by 2030, the number of adults struggling with obesity will reach 1.13 billion.

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Data from the Central Statistical Office (GUS) indicate that this trend is also present in Poland. Data from the Central Statistical Office (2021) indicate a worsening epidemiological situation in Poland in terms of excess weight. In just five years (2014-2019), the percentage of adults who were overweight or obese increased from just over 53% to 57%. This increase was evident in both groups analyzed. In the male population, the percentage rose by 3 percentage points (to 65% in 2019), while in women, the increase exceeded 3 percentage points (reaching 49% in 2019).

Sleep duration appears to be a modifying factor that determines how strongly genetic predisposition to obesity and the amount of adipose tissue actually translate into BMI (Masood & Moorthy, 2023). This provides further confirmation of the thesis that sleep is essential for maintaining neuroendocrine homeostasis and ensuring the proper functioning of processes related to glucose metabolism. There are also strong reasons to believe that the relationship between sleep duration, obesity, and type 2 diabetes is bidirectional. On one hand, obesity and diabetes can lead to sleep disturbances, for example, through a high frequency of obstructive sleep apnea, the occurrence of nocturnal hypoglycemia, nocturia, or painful neuropathy. On the other hand, changes in body weight affect sleep; it has been observed that a clinically significant weight loss in individuals who previously slept for short durations (≤7 h) was associated with an extension of sleep time and a reduction in daytime sleepiness (Antza et al., 2021). This paper aims to review current research analyzing the impact of sleep quality on the development of obesity and insulin resistance.

The paper also aims to present the biological mechanisms underlying this relationship and to indicate potential preventive and therapeutic strategies.

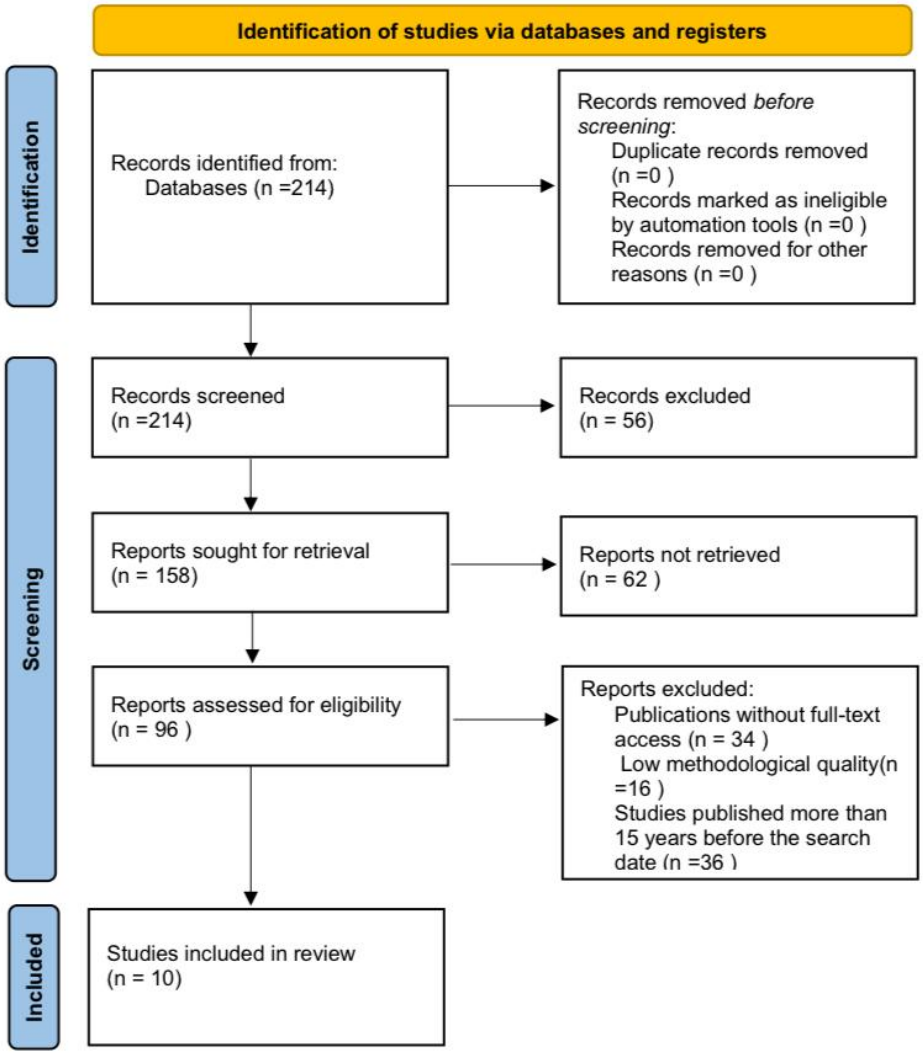

Figure 1: PRISMA Chart

2. REVIEW METHODS

This review was prepared based on a systematic literature search in major scientific databases, including PubMed. The search process employed combinations of keywords, including obesity, sleep quality, insulin resistance, glucose metabolism, and sleep hygiene. Publications from the last 15 years, published in English or Polish, were included. Additionally, the reference lists of the reviewed publications were analyzed to identify additional items that met the thematic criteria.

Inclusion & Exclusion

The review included original, clinical, and observational studies, as well as meta-analyses and systematic literature reviews that analyzed the relationship between sleep quality or duration and the development of obesity, insulin resistance, or metabolic disorders. Experimental and interventional studies concerning the impact of sleep hygiene, physical activity, circadian rhythm, and shift work on glucose metabolism and body weight were also included. The included works covered both adult and adolescent populations, as well as well-designed studies on animal models analyzing the metabolic and hormonal mechanisms of sleep disorders. Excluded from the analysis were: case reports, editorial comments, and review papers not including quantitative data, publications without full-text access, and articles of low methodological quality (e.g., lack of a control group, small sample size <10 participants). All data presented in this paper come from peer-reviewed scientific studies that met the above criteria.

A preliminary search of databases identified 214 records. After removing duplicates and screening titles and abstracts, 96 articles were selected for full-text evaluation. Based on inclusion and exclusion criteria, 10 publications were included in the final synthesis. The entire selection process is illustrated in the PRISMA diagram (Figure 1).

3. RESULTS & DISCUSSION

The role of sleep in metabolic and hormonal regulation

Sleep is a fundamental pillar for maintaining metabolic homeostasis, and its disturbances, both in terms of quantity and quality, are closely linked to an increased risk of developing lifestyle diseases, including obesity and type 2 diabetes. This relationship is mediated through complex neuroendocrine interactions and changes in the secretion of key hormones, whose dynamics are strictly dependent on the different phases of sleep (Leproult & Van Cauter, 2010; Hirotsu et al., 2015). Two main phases, NREM and REM, play different metabolic roles. In the NREM phase, the sympathetic nervous system becomes less active, and blood pressure, heart rate, and brain temperature all decrease. The brain's glucose consumption drops to a minimum in this phase, serving the vital function of energy preservation (Leproult & Van Cauter, 2010). In contrast, the REM phase is characterized by increased brain activity, comparable to the waking state, which is associated with increased glucose metabolism. Consequently, disturbances in the balance between the phases, especially through the reduction of SWS sleep, result in impaired glucose tolerance and reduced insulin sensitivity, even when the nominal sleep time is maintained (Leproult & Van Cauter, 2010). Metabolic regulation during sleep is inextricably linked with the circadian rhythm of the secretion of key hormones. Cortisol, known as the stress hormone, reaches its lowest concentration under physiological conditions in the first hours of sleep. After this, its level increases in the second half of the night, preparing the body for activity during the day. The evening increase in cortisol levels, which is a direct result of reduced sleep time, is considered one of the mechanisms promoting the development of insulin resistance (Hirotsu et al., 2015). However, this is only part of the bigger picture, as the body's energy balance is equally strongly influenced by appetite-regulating hormones, namely leptin and ghrelin. Leptin, which is synthesized in adipose tissue, acts as a satiety hormone; its concentration naturally increases during sleep, thereby inhibiting appetite. On the other hand, ghrelin, secreted mainly in the stomach, is a hunger hormone, and its level decreases during sleep (Shukla & Basheer, 2016). As Leproult and Van Cauter (2010) have shown, the hormonal balance that regulates appetite is extremely sensitive to sleep deprivation. Just two nights of limited sleep are enough to cause leptin levels to drop by 18%, while ghrelin levels increase by 28%.

This rapid hormonal change has a direct impact on subjective feelings – it intensifies hunger and increases appetite. Importantly, this appetite is particularly focused on high-calorie and carbohydrate-rich foods. Chronic sleep deprivation can lead to the consolidation of this hormonal imbalance, promoting excessive calorie consumption and weight gain (Shukla & Basheer, 2016).

The impact of sleep quality and duration on glucose metabolism and insulin sensitivity

Disturbances in sleep rhythm, both in terms of its shortened duration and reduced quality, are increasingly being identified as a significant factor for the development of insulin resistance and abnormalities in glucose metabolism. The body's treatment of sleep

deprivation as a stressor triggers excessive activity of the sympathetic nervous system and the HPA axis. The direct consequence of this reaction is an increase in the concentration of cortisol and catecholamines in the blood (Xu et al., 2016). This pathway is identified as one of the fundamental mechanisms by which sleep deprivation leads to glucose homeostasis disorders. These hormones have an action opposite to that of insulin, which directly translates into impaired glucose tolerance. Experiments on animal models have demonstrated that long-term sleep deprivation leads to a meaningful increase in the HOMA-IR index and unfavorable outcomes in glucose tolerance tests (IPGTT) and insulin tolerance tests (ITT), providing clear evidence of the development of insulin resistance and glucose intolerance (Xu et al., 2016). Even short-term sleep restriction has significant consequences. In human studies, it has been demonstrated that a single night of sleep restriction can reduce peripheral insulin sensitivity (Mattos et al., 2020). These studies, conducted in diverse groups, provide consistent evidence for this relationship.

The relationship between sleep quality and obesity

The relationship between obesity and sleep problems is bidirectional. People with obesity more often complain about poor sleep, and at the same time, insufficient sleep promotes further weight gain (Tuna et al., 2022). Clinical studies have confirmed a high relationship between sleep indicators and anthropometric parameters. In one study that compared obese individuals with a control group, it was shown that poor sleep quality (measured by a PSQI score above 5) affected as many as 79% of obese individuals. For comparison, in the control group, this problem occurred in only 36% of the subjects. The same study found a statistically significant correlation between the total PSQI score and body mass index (BMI), percentage of body fat, and waist, hip, and neck circumferences. Moreover, regression analysis showed that BMI is a predictive factor for the PSQI score (Tuna et al., 2022). The authors of studies on healthcare workers came to similar conclusions. In this group, the percentage of people suffering from poor sleep quality was significantly higher among overweight or obese individuals (36.1%) compared to those with a normal body weight (22.5%). It was found that sleeping fewer hours with the presence of sleep disorders independently predicted overweight and obesity in this group. Sleeping less than 7 hours per day, which is classified as insufficient, has been linked to weight gain (Huang et al., 2024).

An important risk factor for obesity, which is directly related to circadian rhythm disorders, is shift work (Huang et al., 2024; Sooriyaarachchi et al., 2022). Shift work schedules disrupt natural sleep rhythms, leading to chronic sleep deficiency (Huang et al., 2024). It has been demonstrated that shift work, sleep deficiency, and exposure to light at night are associated with an increased incidence of obesity (Sooriyaarachchi et al., 2022). It has been reported that every 5 years of rotating night work is associated with a statistically significant increase in the BMI index (Huang et al., 2024). Shift work also increases the risk of developing metabolic syndrome (MetS). A key feature of this syndrome is abdominal obesity (Sooriyaarachchi et al., 2022). A meta-analysis of studies revealed that shift workers in the healthcare sector are at more than twice the risk (OR = 2.17) of developing metabolic syndrome compared to day workers (Sooriyaarachchi et al., 2022). Table 1 presents a comparison of three publications that illustrate the different research approaches to the problem described in this paper. It contrasts the study by Tuna et al., (2022) on clinically obese patients with the 2024 analysis on healthcare professionals. Also, a meta-analysis by Sooriyaarachchi et al., (2022) examined the metabolic risk associated with shift work. Taken together, these studies from diverse cohorts provide a consistent body of evidence for this relationship (Table 1).

Table 1: Comparative summary of studies analyzing the relationship between sleep quality and obesity

Compared Feature	**Tuna et al., 2022**	**Huang et al. , 2024**	**Sooriyaarachchi et al., 2022**
Main Objective	Determining the impact of obesity on sleep and the relationship between sleep and anthropometric and metabolic parameters.	Study of the sleep quality of healthcare workers and its correlation with overweight and obesity	A review and meta-analysis of the association between shift work (affecting sleep) and the risk of metabolic syndrome (MetS), a key feature of which is abdominal obesity
Study population	76 obese patients (BMI >30 kg/m^2) and 27 control subjects (BMI <30 kg/m^2)	402 healthcare workers (doctors and nurses) in China	Meta-analysis of 12 studies (totalling 42 to 738 participants per study) involving healthcare professionals.

Main Relationship (Sleep vs. Obesity)	Poor sleep leads to weight gain, which in turn can cause further sleep disturbances. BMI has been identified as a predictor of PSQI (sleep quality index).	Healthcare workers who are overweight or obese have poorer sleep quality compared to those of a healthy weight.	Shift work, which often results in sleep disturbances, is associated with chronic diseases, including obesity. Sleep deprivation is an individual risk factor for developing obesity.
Prevalence of Poor Sleep Quality	Poor sleep quality (PSQI>5) was observed in 79% of the obese group and 36% of the control group.	The percentage of people with poor sleep quality (PSQI>10) was significantly higher in the overweight/obese group (36.1%) than in the normal weight group (22.5%).	10 of the 12 studies analyzed showed a higher prevalence of MetS (strongly associated with obesity) among shift workers (exposed to sleep disorders)
Sleep Factors Affecting Weight	A significant correlation was found between BMI and: sleep quality, sleep latency, sleep duration, sleep efficiency and sleep disorders	Shorter sleep duration and sleep disturbances have been identified as independent risk factors for overweight and obesity.	Shift work leads to sleep disturbances and reduced sleep duration. Sleep deprivation is a risk factor for obesity.
Mechanisms	Poor sleep quality leads to endocrine changes: lower leptin levels and higher ghrelin levels, which result in excessive food intake and weight gain.	Sleep restriction causes increased ghrelin levels and decreased leptin levels. Sleep deprivation leads to increased energy intake and weight gain (particularly visceral fat).	Disruption of the circadian rhythm (chronodisk) can lead to symptoms of MetS. Sleep deprivation is associated with hormonal changes (e.g., leptin).
Diseases associated with sleep disorders	Obstructive sleep apnea (OSA) and restless legs syndrome (RLS) are more common in obese individuals. Obese individuals had an 88% risk of OSA and a 45% risk of RLS.	(The study did not focus on diagnosing specific disorders, but on overall sleep quality measured by the PSQI questionnaire.)	Shift work disorder is the most common work disorder among healthcare shift workers.

4. CONCLUSION

Sleep plays a fundamental role in maintaining metabolic balance. Our review shows that lack of sleep acts as a stressor for the body. It triggers cortisol release and deregulates appetite hormones, which drives insulin resistance. We also observed a clear bidirectional link. Patients with obesity report poorer sleep, and insufficient sleep fuels further weight gain. Shift work aggravates this by disrupting natural rhythms. Irrespective of the group studied, sleep disorders are consistently linked to a higher body mass index and a worse metabolic profile.

List of abbreviations:

BMI – Body Mass Index
GUS – Central Statistical Office (Główny Urząd Statystyczny)
HOMA-IR – Homeostatic Model Assessment for Insulin Resistance
HPA – Hypothalamic-Pituitary-Adrenal axis
IPGTT – Intraperitoneal Glucose Tolerance Test
ITT – Insulin Tolerance Test
MetS – Metabolic Syndrome
NREM – Non-Rapid Eye Movement
OSA – Obstructive Sleep Apnea
PSQI – Pittsburgh Sleep Quality Index

REM – Rapid Eye Movement
RLS – Restless Legs Syndrome
SWS – Slow-Wave Sleep
WHO – World Health Organization

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Author's contribution
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Methodology: Milena Kędzierska, Michał Biernacki.
Formal Analysis: Agnieszka Kowalska.
Resources: Milena Kędzierska, Michał Biernacki, Michał Wójcicki.
Writing-original draft: Agnieszka Kowalska, Milena Kędzierska, Michał Biernacki, Michał Wójcicki.
Writing-review and editing: Michał Biernacki, Michał Wójcicki.
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Conflict of interest
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Data and materials availability
All data associated with this study will be available based on reasonable request to the Corresponding Author.

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