

Effect of Sleep Duration on Obesity Treatment and Adipocytokine Levels: A Review

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Abstract: Obesity is receiving increasing attention as a global human health problem. Some studies have indicated that sleep duration may have an intervention effect on the occurrence and development of obesity as a lifestyle. The secretion of several adipocytokines in people with insufficient sleep tends to be disrupted, which can induce the development of obesity and even other chronic diseases. This paper presents a review of the current domestic and international approaches to adipocytokines and the use of sleep to interfere with adipocytokines and thus treat obesity, using sleep duration as a disturbing factor.

Keywords: Adipocytokines, Sleep, Obesity.

1. Introduction

The topic of this paper is to investigate the effect of sleep duration on obesity treatment and adipocytokine levels. Nowadays, obesity is a public health problem that should be taken seriously worldwide. Existing studies have found a correlation between sleep quality and obesity, and improvement in sleep quality can be a good way to reduce the development of obesity. However, we do know that adipocytokines are significant mediators of the relationship between sleep deprivation and obesity, which is an useful entry point for our research in this field. The precise involvement of adipocytokines in the development of Research on the mechanism behind the link between inadequate sleep and obesity is still in its infancy. In the future, we need to comprehend how adipocytokines contribute to the onset of obesity, identify the main link between sleep deprivation and obesity, and offer scientific justifications in order to offer recommendations for an all-encompassing approach to the prevention and treatment of obesity.

2. Obesity

2.1. Formation of Obesity

The human body fat is divided into three kinds of fat, brown fat, white fat and beige fat. Obesity does not represent weight gain, but is a state in which the main expression in medicine is the increase in the volume of fat cells and the increase in the number of fat cells, which is usually a disease caused by too much white fat in the body.

2.2. The Harm of Obesity

Obesity (obesity) as an independent risk factor threatens human health. When the caloric intake is greater than the consumption, the excess energy will be stored in the body in the form of fat, when the excess fat storage exceeds the physiological normal value, that is to form obesity[1] . Obesity can interfere with the body's metabolism as an independent influencing factor, posing a threat to human health, and it is also a high risk factor for the development of many common chronic non-communicable diseases (NCDs) such as type 2 diabetes, hypertension, and certain tumors. Obesity has become a global public health topic affecting

human health, and the form of obesity in our population is more serious. In summary, reduce the growth rate of overweight and obesity rate in China, not only to prevent and control obesity, but also to reduce the incidence of other diseases caused by obesity.

2.3. Obesity Treatment

A study [1] speaks of obesity treatment in roughly two aspects:one is lifestyle intervention means, the other is medical means. This paper will explore the treatment of obesity by lifestyle interventions - sleep interventions. Adipose secretion of a biologically active substance called adipocytokines, such as leptin and adiponectin, can maintain energy metabolic homeostasis, such as lipid metabolism, through various secretory pathways. In contrast, insufficient sleep duration or sleep problems may lead to abnormal alterations in the secretion of multiple adipocytokines, which negatively affect the lipid metabolism of the body and thus participate in the development and progression of obesity. [2] can play some complementary role in obesity treatment by reducing the risk of abnormal secretion of factors through abnormal intervention of sleep reduction, which in turn allows the lipid metabolism of the organism to proceed normally. In summary, it is clear that reducing sleep is an important but modifiable risk factor for obesity. The purpose of this paper is to discuss the correlation study between several adipokines and sleep to do a review, and discuss how to regulate adipocytokines through a lifestyle, i.e. sleep, and then regulate lipid metabolism to achieve the purpose of reducing the development of obesity.

3. Correlation between Sleep and Cytokines

3.1. Leptin

The main physiological function of leptin is to regulate eating, energy consumption and body weight. Leptin mainly acts on hypothalamic cells by which anorexigenic factors are induced to act on appetitive neuropeptides, thus affecting the cerebral cortex as well as limbic areas to inhibit eating behavior[3]. A study [4] has indicated [5] that insufficient sleep duration correlates with low levels of leptin. Leptin levels drop with insufficient sleep, and the body produces

more of the hunger hormone gastric hormone. Because leptin suppresses eating and gastric hunger hormone stimulates it, the difference between the two causes an increase in appetite, which in turn stimulates the growth of fat. As a result, getting less sleep increases the likelihood of becoming obese. However, the relationship between excessive sleep duration and obesity is still controversial. In a Canadian study from 2007 [6], it was shown that the rise in body mass brought on by less sleep was linked to a drop in leptin levels, and that the prevalence of obesity was 1.38 and 1.69 times higher in those who slept 9–10 hours per day than in those who slept 2–4 hours less. In a study conducted in 2004 by an American researcher [7], the average blood leptin level was found to be 18% lower in those who slept for 4 h than those who slept for 10 h, while the blood gastric hunger level was 28% higher and hunger and appetite were relatively increased in 6 volunteers who were sleep restricted for 2 nights. The above study shows that there is indeed a significant correlation between leptin and sleep duration.

3.2. Adiponectin

Adiponectin regulates glucose and fatty acid metabolism via the endocrine cycle and has biological effects that are anti-diabetic, anti-inflammatory, and anti-atherosclerotic. Plasma lipocalin levels tend to be somewhat negatively correlated with obesity. A 2013 study by [4] Chinese scholars indicated that blood lipocalin levels decreased by 21.716 ng/mL for every 1-hour increase in sleep duration. [8] Healthy men's fasting serum lipocalin levels were assessed by Kotani et al [4] while age and other lifestyle characteristics were taken into account. Serum lipocalin levels showed a significant inverse relationship with body mass index and a significant inverse relationship with sleep duration. These findings imply that, due to the low serum lipocalin levels linked with increasing BMI and decreased sleep duration, these two factors may be risk factors for obesity. Although most studies have indicated that lipocalin is correlated with sleep duration and the development of obesity, respectively. However, no relevant studies have yet clearly pointed out the relationship between the three, which shows the complex role. But still can speculate, improve the sleep time to improve the sleep quality can in a certain degree reduce the chance of obesity development.

3.3. Perilipin

Perilipin, a crucial phosphoprotein that is encapsulated on the surface of lipid droplets, can be specifically expressed in adipocytes and steroid-producing cells. It is crucial for the regulation of triglyceride export and lipolysis rate in adipose tissue. Perilipin inhibits lipolysis in its baseline state, but it stimulates it when stimulatory effects are being received and energy demands are rising. [9] According to one study, highly obese people had much lower levels of perilipin in their subcutaneous adipose tissue than people who were not obese. [10] Although the effect of perilipin on obesity is gradually unfolding, the correlation between its effect and sleep duration has not been carried out in depth.

3.4. Interleukin-6 (IL-6)

IL-6: is a pro-inflammatory cytokine who maintains lipid metabolism homeostasis and glucose homeostasis. Some studies have indicated that knockdown of IL-6 gene inhibits white fat browning and reduced sensitivity to insulin in mice. [2] According to one study, sleep deprivation leads to

overproduction of IL-6 during the day and underproduction at night. Total sleep deprivation was linked to higher IL-6 secretion, as demonstrated by Vgontzas et al [11]. The average amount of IL-6 in the body increased significantly over the course of a 24-hour period after modest sleep loss in volunteers [0.80.3 pg/mL; P 0.05]. Contrary to Vgontzas' findings, Frey et al. [12] found that acute total sleep deprivation caused a significant drop in IL-6 levels in subjects with total sleep deprivation. When taken as a whole, these results imply that various sleep deprivation techniques with various degrees of deprivation have varying impacts on the changed IL-6 secretion and may even exhibit opposite variations.

3.5. TNF- α

A cytokine called TNF- α is produced by macrophages and other types of cells. TNF- α in adipose tissue may support a number of insulin resistance pathways, including the release of free fatty acids from adipocytes, decreased lipocalin production, and disrupted insulin signaling. Lack of sleep has an impact on the TNF- α system in people. Sleep restriction was linked to a significant increase in the 24-hour TNF- α secretion cycle in men [minus post-sleep pre-sleep restriction] in a study of healthy men [N = 12] and women [N = 13] with mild sleep deprivation [2 hours per night for 7 nights] but no sign of a significant increase in women [13]. Therefore, it can be concluded that elevated TNF- α after sleep deprivation may raise insulin resistance and decrease glucose metabolism, increasing the risk of developing obesity and diabetes, although there are no obvious changes in women and more research is required.

3.6. SFRP5

Secretory frizzled-related protein-5 (Sfrp5) is a newly identified anti-inflammatory adipokine. It has been indicated [14] that deficiency of sfrp5 exacerbates adipose inflammation and insulin resistance in obese conditions by enhancing JNK1 activation in adipose tissue, and is a potential contributor to metabolic diseases and chronic conditions such as obesity and type 2 diabetes. A recent study [15] showed that both Sfrp5 and Wnt5a are recognized as "adipokine duo", which interact with each other in obesity and glucose homeostasis and are among the novel mediators. However, there are few studies on the relationship between sfrp5 and sleep, and there are more studies on the correlation and role of sfrp5 with obstructive sleep apnea, but the relationship between sfrp5 and normal sleep duration is still inconclusive.

4. Conclusion

As a result, it is clear that current research has demonstrated that the secretion of different adipocytokines, such as leptin and adiponectin, is a critical mediator between insufficient sleep and the emergence of obesity and overweight, but the relationship between other adipocytokines, such as periplasmic, IL-6, and sfrp5, and sleep is still unclear and requires further clarification and in-depth investigation. The mechanism of action of adipocytokines was discovered late, and there are still many unknowns about their action, which still need to be explored and studied in depth. The influence mechanism of sleep time on obesity is somewhat vague, but we can still confirm that adipocytokines are as an important medium between sleep and obesity.

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