

MAKUCH, Rafał, KUCHARSKI, Adam, PILARSKI, Konrad, CHROŚCICKA, Alicja, GAŁA, Kamil, CZAJKA, Andrzej, LENARD, Paweł, MICHALSKA, Sara, DEWICKA, Martyna and WAWRZYNIAK, Alicja Maria. Impact of Obesity on Respiratory Health: The Role of Sport and Physical Activity in Prevention and Management. *Quality in Sport*. 2024;15:52231. eISSN 2450-3118.  
<https://dx.doi.org/10.12775/QS.2024.15.52231>  
<https://apcz.umk.pl/QS/article/view/52231>

The journal has had 20 points in Ministry of Higher Education and Science of Poland parametric evaluation. Annex to the announcement of the Minister of Higher Education and Science of 05.01.2024. No. 32553.

Has a Journal's Unique Identifier: 201398. Scientific disciplines assigned: Economics and finance (Field of social sciences); Management and Quality Sciences (Field of social sciences).

Punkty Ministerialne z 2019 - aktualny rok 20 punktów. Załącznik do komunikatu Ministra Szkolnictwa Wyższego i Nauki z dnia 05.01.2024 r. Lp. 32553. Posiada Unikatowy Identyfikator Czasopisma: 201398.

Przypisane dyscypliny naukowe: Ekonomia i finanse (Dziedzina nauk społecznych); Nauki o zarządzaniu i jakości (Dziedzina nauk społecznych).

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The authors declare that there is no conflict of interests regarding the publication of this paper.

Received: 01.06.2024. Revised: 20.06.2024. Accepted: 01.07.2024. Published: 07.07.2024.

## **Impact of Obesity on Respiratory Health: The Role of Sport and Physical Activity in Prevention and Management**

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## **Abstract**

**Introduction and Objective:** Obesity is a major social and economic problem. Since the 1980s, the number of overweight and obese people has doubled. Treatment of such patients is 30% more expensive than for people of normal weight. The main aim of the article is to present the impact of the occurrence of respiratory diseases and the deterioration of the system function. In addition, the article reviews the impact of physical activity and diet on weight loss and the prevention of further weight gain.

**Review and Methods:** Review and summary of studies and meta-analysis of studies available in open-source format on Google Scholar and PubMed.

**Abbreviated Description of the State of Knowledge:** One of the most common diseases in the population is bronchial asthma. People with excess body weight have a more severe course, and numerous studies have been carried out to explain the cause. The main mechanism that worsens respiratory function is mechanical compression by fatty tissue. It has also been observed that the inflammatory response, underlying the etiopathogenesis of asthma, may be caused by the secretion of inflammatory mediators by adipocytes. This is currently being investigated.

Obstructive sleep apnea is a disorder that results in a fragmentation of sleep, which has serious health consequences and leads to a reduction in quality of life. Obesity is a major risk factor. The severity of the condition is strongly related to excess body weight.

Obese hypoventilation syndrome is a condition in which obesity impairs alveolar ventilation and CO<sub>2</sub> removal, resulting in hypercapnia, which can lead to respiratory failure.

**Summary:** Weight loss and interventions to prevent weight gain in people with a baseline BMI >25 kg/m<sup>2</sup> are fundamental to good health. For best results, a calorie-restricted diet should be combined with physical activity. The WHO has created recommendations for the amount of physical activity needed each week.

**Keywords:** Obesity; Respiratory Diseases; Physical Activity; Obstructive Sleep Apnea; Obesity Hypoventilation Syndrome;

## Introduction

Obesity is one of the most important medical issues of the present time. Over the last 50 years, there has been a significant increase in the incidence of obesity. It is promoted by increasingly widespread unhealthy lifestyles, including low levels of physical activity and unhealthy diets [6]. Since the 1980s, the incidence of people with excess body weight has doubled [9], as evidenced by a systematic review and meta-analysis of studies conducted to assess the prevalence of central obesity in populations around the world, taking into account age, sex, race, place of residence, and income level. The results of the meta-analysis of 288 studies involving a total of 13 million people indicate that 41.5% of the world's population aged 15 years and older have a body weight above the normal range. The prevalence of overweight is higher in people over 40 years of age, women, people of Caucasian race, people living in cities, and people with higher income per capita [3].

Obesity is a disease with a complex etiology caused by excessive accumulation of fat in the body, which has a negative impact on the functioning of organ systems and increases the risk of disease and mortality. In addition, studies have shown that treating obese patients is 30% more expensive for the healthcare system than treating people with a normal body weight [5]. The underlying cause of the disease is a long-term imbalance between the amount of energy taken in with food and the amount of energy used by the organism. Excess energy is stored in fat tissue, whose cells become abnormally enlarged. Recent studies have shown that the origin of foods containing high-quality nutrients is particularly important in preventing obesity and the development of many diseases [8]. The genetic predisposition to metabolic and hormonal disorders and the tendency to gain weight in some people cannot be ignored [7].

This article will focus on the harmful effects of obesity on the respiratory system. In many cases, this common disease leads to an earlier onset of health problems, a more difficult course and treatment, and a worse prognosis for patients [2].

The basic index used to control the nutritional status of adults is BMI ( $\text{kg/m}^2$ ). A resulting index value of 30  $\text{kg/m}^2$  or more allows the diagnosis of obesity. BMI values of 25.0–29.9  $\text{kg/m}^2$  are considered overweight, and obesity is further classified into stage I (BMI 30.0–34.9  $\text{kg/m}^2$ ), stage II (BMI 35.0–39.9  $\text{kg/m}^2$ ), and stage III (BMI 40  $\text{kg/m}^2$  or more) [1]. For children, this is done using centile charts, which are specifically designed for gender, age, and population. Obesity can also be classified according to the area where fat is stored in the body. Abdominal obesity, the excessive accumulation in the abdominal area, is defined by the WHO

as a waist circumference over 94 cm in men and over 80 cm in women. This type of obesity is associated with a greater health risk due to an unfavorable lipid profile, hypertension, and atherosclerosis; it is more common in men [4]. Gluteo-femoral obesity is associated with a lower health risk and is more common in women.

### **Connection Between Obesity and Bronchial Asthma**

Bronchial asthma is a respiratory disease characterized by hyperactivity of the bronchial tubes leading to their excessive constriction, triggered spontaneously or in response to a wide range of external and internal factors that do not cause such a reaction in healthy people. Excessive bronchial reactivity is accompanied by chronic inflammation of the airways, which is the basis for the pathogenesis of bronchial asthma. The influence of obesity on respiratory diseases has been observed for a long time, and many studies confirm the increased frequency and worse course of this disease in people with chronic excess body weight. Based on research conducted in the United States from 2001-2009, the incidence of bronchial asthma increased from 7.3% in 2001 to 8.2% in 2009. The incidence in children aged <18 years was 9.4% [11]. During this time period, the prevalence of self-reported obesity in the US population increased by 7%, reaching 26.7% in 2009 [10]. This suggests that although bronchial asthma is less common than obesity, we are seeing an increasing incidence of both disorders. Many studies have collected data indicating that obesity increases the risk of asthma, and the increased immunological activity in both diseases allows us to look for connections between pathological mechanisms in both diseases [12][13].

Excessive adipose tissue, which worsens the mechanical function of the respiratory system, and chronically elevated inflammatory factors play a particularly important role. These are the main risk factors for the course of asthma, therapeutic response, and prognosis. Excessively developed soft tissues accumulate around the thoracic cage, abdominal cavity, and diaphragm, causing a decrease in the compliance of the chest walls and additionally increasing the energy required for the work of the respiratory muscles. This results in a reduction of air flow in the respiratory tract, causing a deterioration in parameters obtained by spirometry, such as forced vital capacity (FVC) and forced expiratory volume in one second (FEV1). Functional residual capacity (FRC) and expiratory reserve volume (ERV) are significantly reduced [14][15][10]. Characteristics of obese individuals include shallow and rapid breathing and increasing dyspnea.

Many researchers have investigated the relation between inflammation and obesity [20]. For this purpose, it is necessary to closely examine the role of adipocytes, which, in addition to their storage function, play an endocrine role consisting of the production of pro-inflammatory factors such as leptin, TNF-alpha, and IL-6, which stimulate inflammatory processes (chronic but with low CRP levels) in the body. In addition, reduced secretion of adiponectin, an anti-inflammatory factor, is observed in obese patients, and its secretion is inhibited by IL-6. Chronically elevated levels of TNF-alpha have a negative effect on the function of T lymphocytes [21].

An important factor that has been studied for many years is leptin (a product of the Obese gene), a protein secreted by adipocytes. It acts as a satiety regulator by acting on receptors in the hypothalamus. As a result of chronically elevated leptin levels, obese people develop resistance to this hormone acting on brain receptors. In addition, leptin is the major pro-inflammatory mediator secreted by adipose tissue cells; it regulates the proliferation and activation of T lymphocytes, monocytes, and macrophages [16].

Most of the available research suggests an association between obesity and inflammatory diseases of the respiratory tract. However, studies in humans do not allow us to draw clear conclusions that obesity worsens the course of eosinophilic airway inflammation. In a study of 727 obese patients with and without asthma, induced sputum was examined. The number of eosinophils in the sputum was increased in patients with asthma, which is characteristic of the disease. However, no significant association was found between increased BMI and sputum eosinophil count [17]. On the other hand, an association between obesity and asthma and reduced airflow efficiency through the airways has been demonstrated [18]. An increased concentration of IL-5 in sputum has also been shown [19]. This suggests that there may be a link between obesity and an inflammatory airway disease such as asthma, although the mechanisms involved are similar to those known, they are to some extent different and not yet well understood.

### **Obstructive Sleep Apnea**

Obstructive sleep apnea (OSA) is a disease characterized by the occurrence of apnea, defined as a 90% reduction in airflow through the airways or a 50% reduction in breathing with a 3% drop in oxygen hemoglobin saturation lasting >10 seconds, caused by obstruction of airflow through the upper airways, with normal respiratory muscle function. Men are more likely to be affected, accounting for about 24% of the adult population aged 30-60, compared to

women, who make up 9% of this group [22]. It is estimated that about 60% of moderate and severe cases of this disease are correlated with obesity. A BMI  $>25 \text{ kg/m}^2$  is the main risk factor for OSA, and the risk of developing the disease increases as the index rises [23]. The accumulation of adipose tissue around the neck and the restriction of respiratory movements due to the accumulation of adipose tissue in the abdomen and chest causes reduced ventilation and reduced vital capacity of the lungs, leading to hypoxia and hypercapnia. Other causes may include enlargement of the palatine tonsils, uvula, and tongue; anatomical abnormalities in the bone structure of the maxilla and mandible; nasal septum deviation; conditions forcing prolonged mouth breathing such as infectious or allergic rhinitis; swelling caused, for example, by hypothyroidism; consumption of alcohol or muscle-relaxing drugs; and GERD (gastroesophageal reflux disease) [23].

OSA manifests as loud snoring and frequent awakenings at night, leading to daytime sleepiness and decreased concentration. Long-term, the disease significantly reduces the quality of life. Periodic drops in saturation cause increased sympathetic nervous system stimulation. The severity of the disease is determined by the apnea and hypopnea index (AHI) per hour of sleep. A score of 5-15 is classified as mild, 15-30 as moderate, and  $>30$  as severe [22]. The severity of the condition has been shown to be related to body weight, with an estimated 10% increase in body weight causing an increase in AHI of up to 32% [23]. Untreated OSA increases the risk of cardiovascular diseases, metabolic diseases including type 2 diabetes mellitus, neurological disorders, and depression.

CPAP remains the gold standard for the treatment of OSA; however, all patients are advised to reduce their body weight, as both patients with severe and mild forms of the disease benefit from this approach. A 10% weight loss results in a 26% reduction in AHI [25]. In a randomized clinical trial, lifestyle changes and weight loss over six months were shown to reduce disease severity in 57% of subjects, achieve complete remission in 29%, and eliminate the need for CPAP therapy in 61% of subjects [24].

### **Obesity Hypoventilation Syndrome and Obesity-Related Respiratory Failure**

OHS (Obesity Hypoventilation Syndrome) is a condition in which effective ventilation is limited by excess body weight. In this case, a triad of symptoms is observed: BMI  $>30 \text{ kg/m}^2$ , hypoventilation leading to daytime hypercapnia, and nocturnal hypoventilation not explained by the presence of other diseases. Increased body weight causes restriction of diaphragmatic movement and decreased chest compliance, resulting in short and shallow

breaths in patients. OHS is found in about 90% of patients with OSA and in about 50% of patients hospitalized for obesity with a BMI over 50 kg/m<sup>2</sup> [26,27]. Similar to patients with chronic obstructive pulmonary disease (COPD), central chemoreceptors, which respond to the partial pressure of carbon dioxide in the blood, have a reduced stimulatory response to respiratory drive. Their role is partly taken over by peripheral chemoreceptors, which are sensitive to the partial pressure of oxygen. Therefore, oxygen therapy should be used cautiously in these patients because of the risk of inducing respiratory arrest [26, 28].

One-third of patients with OHS may develop ORRF (Obesity-Related Respiratory Failure) through a mechanism of acute hypercapnic respiratory failure, most commonly in association with COPD. These patients require non-invasive ventilation (NIV) [25]. Treatment should be multifaceted, including positive airway pressure (PAP) therapy, lifestyle changes, weight loss, and, for eligible patients, bariatric surgery [27,28].

### **Role of Physical Activity in Weight Loss and Preventing Obesity**

Physical activity plays a very important role in a healthy human life. The WHO recommends that adults should perform at least 150–300 minutes per week of moderate-intensity aerobic physical activity, or at least 75–150 minutes per week of vigorous-intensity aerobic physical activity, or an equivalent amount of mixed-intensity physical activity during the week. At the same time, it is recommended to limit the time spent sitting [30]. This will help to prevent many systemic diseases and obesity, and to maintain previously reduced body weight.

Physical activity plays an important role in reducing body weight and preventing obesity. Many trials have been carried out to compare the most effective ways of losing weight. Physical activity alone has been compared with a combination of exercise and a calorie-restricted diet. Based on a review of the research, the best solution is to combine a calorie-restricted diet with physical activity. This strategy resulted in the most effective reduction in the BMI index.

Based on the systematic review by Washburn et al., results were obtained confirming the higher efficacy of weight loss through exercise and dietary modification. The median weight loss was 8.8%, compared with 6.9% for a restricted diet alone [33][34]. A study, which included 130 adults with class III obesity and a BMI of around 44 kg/m<sup>2</sup>, showed that physical activity added to dietary weight loss could reduce body weight by an additional 2.7 kg, achieving a 10.9 kg lower body weight versus 8.2 kg lower body weight after 6 months in



patients using diet alone [35]. A systematic review by Thorogood et al. examined the effects of exercise alone, without diet, on weight loss. During 6 months of exercise programs, the average weight loss was only 1.6 kg [36].

A low-calorie diet has been shown to be effective in reducing body weight. An even better effect can be achieved by adding physical activity of at least moderate intensity to create a greater difference between the amount of energy consumed and used by the body. However, the role of physical activity is particularly important for long-term weight loss and preventing weight gain. Performing 150–300 minutes of at least moderate-intensity physical activity per week, as recommended by the WHO, can help achieve this [32][37]. Another positive aspect of physical activity is the reduction of abdominal adipose tissue. This is beneficial even without weight loss, as abdominal obesity significantly increases the risk of cardiovascular disease [29][31][38][39].

## **Conclusions**

A review of articles and scientific research leads to the conclusion that excess body weight has a negative effect on the functioning of the respiratory system. The influence of excess adipose tissue on the mechanical function of the organs of this system is best documented. The flow of air through the respiratory tract deteriorates during exercise and sleep, and the effort required to breathe increases. The influence of fat cells in the development of inflammatory diseases is also very likely. The problem of obesity in the world is serious and growing, so both treatment and prevention are important. Physical activity combined with a healthy, low-calorie diet are proven methods for a healthy lifestyle.

## **Disclosure**

### **Author's contribution**

Conceptualization: Rafał Makuch and Adam Kucharski; Methodology: Alicja Wawrzyniak; Software: Alicja Chrościcka; Check: Andrzej Czajka and Kamil Gała; Formal analysis: Konrad Pilarski and Martyna Dewicka; Investigation: Paweł Lenard and Sara Michalska; Resources: Kamil Gała; Data curation: Alicja Chrościcka; Writing - rough preparation: Adam Kucharski and Rafał Makuch; Writing - review and editing: Alicja Wawrzyniak and Konrad Pilarski; Visualization: Martyna Dewicka; Supervision: Sara Michalska; Project administration: Rafał Makuch and Paweł Lenard; Receiving funding - no specific funding.

All authors have read and agreed with the published version of the manuscript.

**Financing statement**

This research received no external funding.

**Institutional Review Board Statement**

Not applicable.

**Informed Consent Statement**

Not applicable.

**Data Availability Statement**

Not applicable.

**Conflict of interest**

The authors deny any conflict of interest.

## References

1. Kendall, B. J., Rubenstein, J. H., Cook, M. B., Vaughan, T. L., Anderson, L. A., Murray, L. J., Shaheen, N. J., Corley, D. A., Chandar, A. K., Li, L., Greer, K. B., Chak, A., El-Serag, H. B., Whiteman, D. C., & Thrift, A. P. (2016). Inverse association between gluteofemoral obesity and risk of Barrett's esophagus in a pooled analysis. *Clinical Gastroenterology and Hepatology: The Official Clinical Practice Journal of the American Gastroenterological Association*, 14(10), 1412-1419.e3. <https://doi.org/10.1016/j.cgh.2016.05.032>
2. Lin, X., & Li, H. (2021). Obesity: Epidemiology, pathophysiology, and therapeutics. *Frontiers in Endocrinology*, 12. <https://doi.org/10.3389/fendo.2021.706978>
3. Wong, M. C. S., Huang, J., Wang, J., Chan, P. S. F., Lok, V., Chen, X., Leung, C., Wang, H. H. X., Lao, X. Q., & Zheng, Z.-J. (2020). Global, regional and time-trend prevalence of central obesity: a systematic review and meta-analysis of 13.2 million subjects. *European Journal of Epidemiology*, 35(7), 673–683. <https://doi.org/10.1007/s10654-020-00650-3>
4. WHO. Waist Circumference and Waist–Hip Ratio: Report of a WHO Expert Consultation 8–11 December ed. Geneva; 2008. p. 47. <https://www.who.int/publications/i/item/9789241501491>
5. Carey DGP. Abdominal obesity. *Curr Opin Lipidol*. 1998;9(1):35–40. DOI: [10.1097/00041433-199802000-00008](https://doi.org/10.1097/00041433-199802000-00008)
6. WHO Consultation on Obesity (1999: Geneva, Switzerland) & World Health Organization. Obesity: Preventing and Managing the Global Epidemic. Report of a WHO Consultation. *World Health Organ Tech Rep Ser* (2000) 894:i–xii, 1-253
7. Singh RK, Kumar P, Mahalingam K. Molecular Genetics of Human Obesity: A Comprehensive Review. *C R Biol* (2017) 340(2):87–108. doi: 10.1016/j.crv.2016.11.007 DOI: [10.1016/j.crv.2016.11.007](https://doi.org/10.1016/j.crv.2016.11.007)
8. Sacks FM, Bray GA, Carey VJ, Smith SR, Ryan DH, Anton SD, et al. Comparison of Weight-Loss Diets With Different Compositions of Fat, Protein, and Carbohydrates. *N Engl J Med* (2009) 360(9):859–73. doi: 10.1056/NEJMoa0804748 DOI: [10.1056/NEJMoa0804748](https://doi.org/10.1056/NEJMoa0804748)

9. Ataey A, Jafarvand E, Adham D, Moradi-Asl E. The Relationship Between Obesity, Overweight, and the Human Development Index in World Health Organization Eastern Mediterranean Region Countries. *J Prev Med Public Health* (2020) 53(2):98–105. doi: 10.3961/jpmph.19.100  
DOI: [10.3961/jpmph.19.100](https://doi.org/10.3961/jpmph.19.100)
10. Sutherland, E. R. (2014). Linking obesity and asthma. *Annals of the New York Academy of Sciences*, 1311(1), 31–41. <https://doi.org/10.1111/nyas.12357>
11. 2011. Vital signs: asthma prevalence, disease characteristics, and self-management education: United States, 2001–2009. *Morb. Mortal. Wkly. Rep.* 60: 547–552.
12. Beuther, D.A. & E.R. Sutherland. 2007. Overweight, obesity, and incident asthma: a meta-analysis of prospective epidemiologic studies. *Am. J. Respir. Crit. Care Med.* 175: 661–666. DOI: 10.1164/rccm.200611-1717OC. DOI: [10.1164/rccm.200611-1717OC](https://doi.org/10.1164/rccm.200611-1717OC)
13. Nystad, W., H.E. Meyer, P. Nafstad, *et al.* 2004. Body mass index in relation to adult asthma among 135,000 Norwegian men and women. *Am. J. Epidemiol.* 160: 969–976. DOI: [10.1093/aje/kwh303](https://doi.org/10.1093/aje/kwh303)
14. Biring, M.S., M.I. Lewis, J.T. Liu & Z. Mohsenifar. 1999. Pulmonary physiologic changes of morbid obesity. *Am. J. Med. Sci.* 318: 293–297. DOI: [10.1097/00000441-199911000-00002](https://doi.org/10.1097/00000441-199911000-00002)
15. Beuther, D.A. & E.R. Sutherland. Obesity and pulmonary function testing. *J. Allergy Clin. Immunol.* 2005; 115: 1100–1101. DOI: [10.1016/j.jaci.2004.12.1141](https://doi.org/10.1016/j.jaci.2004.12.1141)
16. Sierra-Honigmann, M.R, A.K. Nath, C. Murakami, *et al.* 1998. Biological action of leptin as an angiogenic factor. *Science* 281: 1683–1686. DOI: [10.1126/science.281.5383.1683](https://doi.org/10.1126/science.281.5383.1683)
17. Todd, D.C., S. Armstrong, L. D'Silva, *et al.* 2007. Effect of obesity on airway inflammation: a cross-sectional analysis of body mass index and sputum cell counts. *Clin. Exp. Allergy* 37: 1049–1054. DOI: [10.1111/j.1365-2222.2007.02748.x](https://doi.org/10.1111/j.1365-2222.2007.02748.x)
18. McLachlan, C.R., R. Poulton, G. Car, *et al.* 2007. Adiposity, asthma, and airway inflammation. *J. Allergy Clin. Immunol.* 119: 634–639. DOI: [10.1016/j.jaci.2006.10.029](https://doi.org/10.1016/j.jaci.2006.10.029)

19. Desai, D., C. Newby, F.A. Symon, *et al.* 2013. Elevated sputum interleukin-5 and submucosal eosinophilia in obese individuals with severe asthma. *Am. J. Respir. Crit. Care Med.* 188: 657–663. DOI: [10.1164/rccm.201208-1470OC](https://doi.org/10.1164/rccm.201208-1470OC)
20. Holgate, S. T. (2008). Pathogenesis of asthma. *Clinical and Experimental Allergy: Journal of the British Society for Allergy and Clinical Immunology*, 38(6), 872–897. <https://doi.org/10.1111/j.1365-2222.2008.02971.x>
21. Trojanowski, T. (2019). Infekcje układu oddechowego—czy mają związek z otyłością?. In *Forum Medycyny Rodzinnej* (Vol. 13, No. 6, pp. 292-299).
22. Lv, R., Liu, X., Zhang, Y., Dong, N., Wang, X., He, Y., Yue, H., & Yin, Q. (2023). Pathophysiological mechanisms and therapeutic approaches in obstructive sleep apnea syndrome. *Signal Transduction and Targeted Therapy*, 8(1), 1–46. <https://doi.org/10.1038/s41392-023-01496-3>
23. Wyszomirski, K., Walędziak, M., & Różańska-Walędziak, A. (2023). Obesity, bariatric surgery and obstructive sleep apnea—A narrative literature review. *Medicina* (Kaunas, Lithuania), 59(7), 1266. <https://doi.org/10.3390/medicina59071266>
24. Carneiro-Barrera, A., Amaro-Gahete, F. J., Guillén-Riquelme, A., Jurado-Fasoli, L., Sáez-Roca, G., Martín-Carrasco, C., Buena-Casal, G., & Ruiz, J. R. (2022). Effect of an interdisciplinary weight loss and lifestyle intervention on obstructive sleep apnea severity: The INTERAPNEA randomized clinical trial. *JAMA Network Open*, 5(4), e228212. <https://doi.org/10.1001/jamanetworkopen.2022.8212>
25. Krishnan, A., Ellis, P., Antoine-Pitterson, P., Oakes, A., Jones, B., Turner, A., & Mukherjee, R. (2023). Long-term mortality following acute noninvasive ventilation for obesity-related respiratory failure: A retrospective single-centre study. *Canadian Respiratory Journal: Journal of the Canadian Thoracic Society*, 2023, 1–7. <https://doi.org/10.1155/2023/5370197>
26. Dougherty, M., Lomiguen, C. M., Chin, J., & McElroy, P. K. (2021). Obesity hypoventilation syndrome-related challenges in acute respiratory failure. *Cureus*, 13(9). <https://doi.org/10.7759/cureus.18066>

27. Ramírez Molina, V. R., Masa Jiménez, J. F., Gómez de Terreros Caro, F. J., & Corral Peñafiel, J. (2020). Effectiveness of different treatments in obesity hypoventilation syndrome. *Pulmonology*, 26(6), 370–377. <https://doi.org/10.1016/j.pulmoe.2020.05.012>
28. Obesity-Hypoventilation Syndrome Marsha H. Antoine, Abdulghani Sankari 1 , Pradeep C. Bollu 2 <https://www.ncbi.nlm.nih.gov/books/NBK482300/>
29. Hill, J. O., & Wyatt, H. R. (2005). Role of physical activity in preventing and treating obesity. *Journal of Applied Physiology* (Bethesda, Md.: 1985), 99(2), 765–770. <https://doi.org/10.1152/japplphysiol.00137.2005>
30. <https://www.who.int/news-room/fact-sheets/detail/physical-activity>
31. Oktay, A. A., Lavie, C. J., Kokkinos, P. F., Parto, P., Pandey, A., & Ventura, H. O. (2017). The interaction of cardiorespiratory fitness with obesity and the obesity paradox in cardiovascular disease. *Progress in Cardiovascular Diseases*, 60(1), 30–44. <https://doi.org/10.1016/j.pcad.2017.05.005>
32. Swift, D. L., McGee, J. E., Earnest, C. P., Carlisle, E., Nygard, M., & Johannsen, N. M. (2018). The effects of exercise and physical activity on weight loss and maintenance. *Progress in Cardiovascular Diseases*, 61(2), 206–213. <https://doi.org/10.1016/j.pcad.2018.07.014>
33. Jakicic, J. M., Rogers, R. J., Davis, K. K., & Collins, K. A. (2018). Role of physical activity and exercise in treating patients with Overweight and obesity. *Clinical Chemistry*, 64(1), 99–107. <https://doi.org/10.1373/clinchem.2017.272443>
34. Washburn, R. A., Szabo, A. N., Lambourne, K., Willis, E. A., Ptomey, L. T., Honas, J. J., Herrmann, S. D., & Donnelly, J. E. (2014). Does the method of weight loss effect long-term changes in weight, body composition or chronic disease risk factors in overweight or obese adults? A systematic review. *PloS One*, 9(10), e109849. <https://doi.org/10.1371/journal.pone.0109849>
35. Goodpaster, B. H., DeLany, J. P., Otto, A. D., Kuller, L., Vockley, J., South-Paul, J. E., Thomas, S. B., Brown, J., McTigue, K., Hames, K. C., Lang, W., & Jakicic, J. M. (2010). Effects of diet and physical activity interventions on weight loss and cardiometabolic risk factors in severely obese adults: A randomized trial. *JAMA: The Journal of the American Medical Association*, 304(16), 1795. <https://doi.org/10.1001/jama.2010.1505>

36. Chin, S.-H., Kahathuduwa, C. N., & Binks, M. (2016). Physical activity and obesity: what we know and what we need to know. *Obesity Reviews: An Official Journal of the International Association for the Study of Obesity*, 17(12), 1226–1244.  
<https://doi.org/10.1111/obr.12460>
37. Donnelly, J. E., Blair, S. N., Jakicic, J. M., Manore, M. M., Rankin, J. W., & Smith, B. K. (2009). Appropriate physical activity intervention strategies for weight loss and prevention of weight regain for adults. *Medicine and Science in Sports and Exercise*, 41(2), 459–471.  
<https://doi.org/10.1249/mss.0b013e3181949333>
38. Ross, R. (2000). Reduction in obesity and related comorbid conditions after diet-induced weight loss or exercise-induced weight loss in men: A randomized, controlled trial. *Annals of Internal Medicine*, 133(2), 92. <https://doi.org/10.7326/0003-4819-133-2-200007180-00008>
39. Després, J. P. (2006). Intra-abdominal obesity: an untreated risk factor for Type 2 diabetes and cardiovascular disease. *Journal of Endocrinological Investigation*, 29(3 Suppl).  
<https://pubmed.ncbi.nlm.nih.gov/16751711/>