

# Impact of Weight Loss on Sleep Apnea

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## ABSTRACT

The act of sleeping comprises about one-third of our life. It is not just about time, but also the fact that sleeping affects nearly everything that we need to do. Quality of sleep affects quality of life. Obstructive sleep apnea (OSA) is a most common sleep-related breathing disorder, and snoring is the most important symptom. A 30–50% of snorers suffer from OSA, which can further lead to its comorbidities such as arterial hypertension, myocardial infarction, cerebral stroke, depression and anxiety disorders, or impotency disorders. Total body weight, body mass index (BMI), and fat distribution all correlate with odds of having OSA. Obesity causes deposition of adipose in and around the throat, which might lead to snoring and manifest into OSA if left untreated. Every 10 kg increase in weight increases risk by two times. An increase in BMI by six increases risk by four times. In obese patients, even minimal weight loss can be beneficial since it is related to preferential loss of visceral fat first as opposed to subcutaneous fat which has metabolic advantages. Therefore, one of the major modifiable risk factors for developing OSA is obesity. Lifestyle changes in the form of dietary modifications, imparting right knowledge about required nutrition for body, and implementation of the same monitored in a way that can manifest into a permanent behavioral change can go a long way in preventing and curing sleep-related breathing disorders and improving the quality of life of the patient as well as the bed partner.

**Keywords:** Bariatric surgery, Obesity, Obstructive sleep apnea, Weight loss.

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## INTRODUCTION

There is an evolution in understanding about mechanism of upper airway obstruction during sleep in the last two decades. Episodes of obstructive sleep apnea (OSA) and hypopnea along with daytime sleepiness remain undiagnosed in majority of population largely due to unawareness both on patients and physicians part. Many factors are leading to this OSA. Soft tissues and skeletal factors determine the upper airway size of the person. Extent of the capacity of upper airway to collapse during sleep determines the susceptibility of OSA. It is well documented by now that metabolic factors coexist with OSA on a common platform. Out of this metabolic syndrome environment, obesity comes to fore as a strongest predictor of OSA. Almost half of obese persons suffer from OSA.<sup>1</sup> There is documented evidence through magnetic resonance imaging that obese people have increased adipose tissue infiltration in neck (peripharyngeal fat pads), which increases mechanical load on surrounding tissues leading to collapse of pharynx during sleep predisposing the airway to narrowing and hence obstruction while breathing during sleep or complete closure.<sup>2</sup> Therefore, obesity comes to fore as one of the leading causes of OSA. Central obesity (around the neck, trunk, and abdominal viscera) is one of the strongest predictors of OSA.<sup>3</sup> Adipose tissue is also a favorable base for secretion of proinflammatory cytokines<sup>4</sup> that may depress neuromuscular control over upper airway. These cytokines also catalyze the production of reactive oxygen species (ROS), which weaken the force-generating capacity of skeletal muscles that adversely affect the upper airway function.<sup>5</sup> Another adverse effect of obesity is that it encourages a leptin-resistant environment. Leptin is a hormone of energy expenditure and inhibits hunger. Along with this function, it also acts as a powerful neurohumoral ventilator stimulant<sup>6</sup> but due to the deficiency of leptin in central nervous system, it might prompt disturbance of the respiratory control of central nervous system leading to weakened hypercapnic ventilatory response, especially during sleep. Behavioral treatment, which includes lifestyle and dietary modifications, is strongly

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recommended by American Academy of Sleep Medicine for improvement in apnea–hypopnea index (AHI) in obese OSA patients.<sup>7</sup> These strong recommendations are a result of significant reduction in apnea and hypopnea in overweight OSA patients and have been documented in large randomized controlled trials after successful implementation of lifestyle modification programs that included diet-induced weight loss and increased physical activity levels.<sup>8</sup> The favorable results thus obtained via lifestyle modifications were sustained for a period of 12–36 months even after the intervention programs had ended despite regaining 30% to 50% of the original weight.<sup>9,10</sup> It gives a lot of positivity, hope, and confidence on practising lifestyle changes as a primary treatment mode for addressing OSA.<sup>11</sup> A complex relation exists between weight change and level of severity of OSA as reviewed in obese diabetic patients with OSA in whom significant reduction in AHI was achieved after a year of weight loss therapy and this reduction in AHI persisted even after 4 years of intervention despite significant weight gain.<sup>12</sup> A very few studies have examined the type of changes in diet and lifestyle and impact on OSA through weight loss. This chapter will talk about weight loss and lifestyle as foundation of OSA therapy.

## EPIDEMIOLOGY

Epidemiological studies have unfurled the cause of morbidity as to even the mildest form of sleep apnea. There is an independent relation of sleep apnea (with or without symptoms) with cardiovascular disease (CVD), hypertension, stroke, daytime sleepiness, motor vehicular accidents, and poor quality of life. Approximately 25% of adults having a BMI in the range of 25 to 28 kg/m<sup>2</sup> suffer from mild OSA indicated by AHI score of greater than or equal to five. The prevalence of OSA syndrome (OSAS) in the population of Delhi, India, was found to be 13.74% and 3.57%, respectively, for OSA and OSAS<sup>13</sup> In Western countries, the prevalence of OSA has been reported to vary according to gender (~30% in men and ~15% in women), age, and body weight. Men's risk for OSA is twofold higher than that of women.<sup>14</sup>

Premenopausal women are at a lower risk than postmenopausal women. Age also affects the prevalence of OSA, which shows an upward trend until 65 years of age when it reaches its zenith.<sup>15</sup> Most interactive strategy to combat sleep apnea supported by strong research evidence is weight loss. This strategy needs special focus seeing the rampant weight gain in the developed and developing countries leading to lifestyle diseases, of which OSA is one of them.<sup>16</sup>

## OBESITY AND OSA

Obesity and OSA are both casually yet powerfully related.<sup>17</sup> Mechanisms and conditions typically observed in obesity have been proved to be linked mystically with OSA.<sup>18</sup> Approximately 1% of adult population suffers from OSA and it is more common in obese persons. Mechanical obstruction due to obesity is one of the main reasons for developing OSA. Body mass index, circumference of neck, and visceral mass are the strong predictors of OSA due to obesity. Even with a 10% increase in weight in mild OSA patients, the disease progresses to its severe form and with equivalent weight loss more than 20% improvement has been reported in OSA severity.<sup>19</sup> Adolescent sleep apnea has its roots in childhood obesity, which is a result of parental obesity, poor eating habits, and dearth of physical activity.<sup>20</sup>

Children and adolescents having OSA have more than six times risk of having metabolic syndrome when compared with the same age groups without OSA.<sup>21</sup> This scenario calls for an early screening as a preventive measure for the same.

Visceral obesity is common in OSA subjects, which worsens OSA due to fat deposition at particular areas around upper airway. It results in smaller lumen and increased susceptibility of upper airway to collapse leading to apnoea.<sup>22</sup> Adipose tissue deposition around thorax, called truncal obesity, may increase the demand for oxygen by reducing chest compliance and functional residual capacity.<sup>23</sup> Genetic polymorphisms may influence both sleep apnea and obesity, since studies have shown a significant correlation between anthropomorphic adiposity measures and apnea hypoxia index as well as correlation between leptin (involved in energy homeostasis and weight regulation) and OSA and obesity when compared with healthy controls.<sup>24,25</sup>

Surprisingly, awareness about relationship of obesity with sleep apnea was almost lacking in OSA patients with only 5% being aware about it, whereas the awareness of consequences of being obese with other comorbidities like CVD and diabetes were much better.<sup>26</sup>

## WEIGHT LOSS AND OSA

Gain or loss of body weight significantly affects the stage of OSA. Weight management is a scientifically creative strategy to reduce weight through modifying dietary habits. A 40–90% of people having severe obesity report sleep apnea, and 70% of OSA patients present themselves with obesity (sleep apnea and sleep disruptions in obese patients).<sup>27</sup>

Fortunately, obesity is the most modifiable risk factor of OSA. Even a short-term weight reduction leads to a better metabolic regulation in OSA patients.<sup>28</sup> This may lead to the initiation of a weight reduction cycle since research has proved that even a weight loss of as low as 10% leads to more than double the reduction (measured via AHI) in sleep apnea and reduced sleep apnea follows weight loss.<sup>19</sup> Chemoreflex function is regulated via obesity through neurohormonal mediators such as leptin, which shows a downward trend with weight loss in sleep apnea patients.<sup>28</sup>

Studies done past 2–3 decades vouch for a weight loss therapy and maintenance of the same as an improvement in severity of OSA without surgery.<sup>29,30</sup> Behavioral programs with and without continuous positive airway pressure (CPAP) therapy to reduce weight show promising results in weight reduction at an average of 13.5% till a year significantly reducing oxygen de-saturation index, which remained significantly reduced from baseline level even after two years of weight loss therapy despite the fact that patients had regained lost weight.<sup>8</sup> Interestingly, the patients with CPAP therapy did not differ in the oxygen de-saturation index from the ones without CPAP therapy. It is also indicated from the studies that the length of weight loss interventions matters as a motivational factor to reduce weight or maintain the reduced weight.<sup>8</sup> It has been documented that weight loss has a different effect on different gender. Men were more prone to worsening of OSA as their body weight increased as compared to women. Women's risk was half of men's risk of increase in OSA severity with increase in equal amount of body weight but interestingly the same amount of weight loss improved OSA symptoms more in men than in women<sup>31</sup> Obese patients undergoing weight loss therapy and achieving a mean reduction in BMI by 15 kg/m<sup>2</sup> experience an AHI reduction by 36 events per hour indicating that every 1 unit reduction in BMI equals to 2.3 events reduction in AHI per hour.<sup>32</sup>

### Weight Loss as First-line Treatment for Snoring and Mild Sleep Apnea

For a simple snorer or even a mild sleep apneic, most of the medical and surgical modalities would neither be acceptable nor warranted. So, weight management by dietary consultation and lifestyle modification remains the mainstay of treatment. Snoring and mild sleep apnea when ignored may progress into moderate and severe OSA with a progressive weight gain.<sup>33,34</sup> For patients of simple snoring and mild sleep apnea, it is the quality of life of bed partner also is affected. In many studies in which lifestyle modification including primarily diet and exercise programs targeting weight loss was the primary therapeutic intervention for correcting snoring and mild sleep apnea, it was observed that the more the patients lost weight, the more was the elimination of snoring with a significant reduction in BMI and AHI scores.<sup>35–37</sup>

### Diet and OSA

Food is a fuel for our body to survive and work in a healthy way. Good nutrition provides all the essential nutrients through our daily diet, which supports healthy body and survival.

Chemical compounds such as carbon, hydrogen, oxygen, nitrogen, and phosphorus are the elements that are the part of compounds such as water, carbohydrates, amino acids (in proteins), fatty acids (in lipids), and nucleic acids (DNA and RNA). This is what our body is basically composed of. The fuel for the body, that is, the nutrition consists essentially of five classes of nutrients, viz., carbohydrates, fats, proteins, vitamins, and minerals. The first three classes are termed as macronutrients, which are majorly required by the body for energy and as building blocks and provide structural material for the compounds of the body, whereas the latter two classes are termed as micronutrients required as protective and defense mechanism against various diseases as well as support macronutrients for their proper functioning and utilization.

The body gets unwell due to many reasons, but one of the major reasons in today's world is unhealthy lifestyle, in which diet forms a major part. We are what we eat held true since time immemorial and will hold true till infinity. To understand this fact, let us discuss the macronutrients in brief.

### *Carbohydrates*

These are classified as monosaccharide (glucose, fructose-fruit sugar, and galactose), disaccharides (sucrose-table sugar, lactose-milk sugar, and maltose-malt sugar), and polysaccharides (starch and glycogen) depending upon the number of sugar units (monomer) they contain. Polysaccharides are referred to as complex carbohydrates as it takes longer for them to be digested because their digestion requires breaking of multiple bonds of sugar molecules by which they are made of. Whole grains, pulses, whole fruits, and green vegetables are some of the examples of the same. Monosaccharide and disaccharides are considered simple sugars.

Major source of energy comes from carbohydrates in our food; therefore, they play a very important role in weight management both quantitatively and qualitatively.<sup>38</sup> One gram of carbohydrate gives 4 kilocalories. A 45–55% of energy should come from carbohydrates, preferably the complex sources, in daily diet. The spike in blood glucose levels after ingestion of simple sugars (rapidly absorbed from carbohydrates) has been researched and confirmed as a root cause of today's lifestyle diseases ranging from obesity, dyslipidemia, diabetes, etc., since simple sugars have knowingly or unknowingly become a major part of food in modern times due to so much of processing in every food stuff, which is eaten nowadays.<sup>39</sup> Rampant consumption of free sugar particularly in the form of sugar-sweetened beverages has been linked to weight gain.

### *Fats*

Fats are the most concentrated source of energy with 1 g of fat giving 9 kilocalories. There are two kinds of dietary fats—visible and invisible. Visible fats are animal fats such as butter, cream, and vegetable oils. We can see these fats and use them in measured amounts in our diet. Invisible fats are the fats that are naturally occurring in the food stuffs and not visible to the naked eye. Fat found in grains, legumes, eggs, meat, etc. are some examples of invisible fat. Nuts and oil seeds provide the body with omega-3 and omega-6 fatty acids. An adequate fat intake is required as a concentrated source of energy as well as source of essential fatty acids and to aid absorption of fat soluble vitamins but excess fat in diet converts into triglycerides and cholesterol in blood. Less than 30% of energy should be derived from fat in the diet.

### *Protein*

Proteins are the building blocks of body. They are required for growth, muscle mass, and to repair wear and tear of body. Good quality protein sources are milk and milk products, eggs, and meat. To put it in low-calorie version, it has to be fat free or low fat milk and milk products, egg whites, and lean meat cuts like poultry. Other protein sources in diet are pulses, legumes, nuts, and oil seeds. Indians get most of their protein from mixed sources like cereals and pulses. These sources are also rich in carbohydrates, which might end up in carbohydrate overload and protein-deficit diets. Such subjects despite being overweight are deficient in protein intake and hence have low muscle mass.

### *Thermic Effect of Food (TEF)*

It is also called specific dynamic action (SDA). It is diet-induced thermogenesis. It is the amount of energy required by a particular food to be digested by the body. In other words, body also spends energy (cost of processing a nutrient for its use to the body as well as disposal of waste) to digest the food which is termed as TEF.<sup>40</sup> Different macronutrients have different SDA. Proteins have the greatest SDA at the rate of 20–35%. Therefore, food rich in protein has fewer calories to be absorbed by the body. SDA for carbohydrates varies from 5% to 15% depending upon the complexity of the carbohydrate. Fats have at the most 5% to 15% of SDA.

### **Food Perceptions of Overweight OSA Persons**

Behavioral factors influenced by individual lifestyle, culture, socioeconomic status, and education and awareness status may lead to faulty and excessive caloric intake.<sup>41</sup>

In one of the studies done by Sahni et al., it was most interesting to find that there were many wrong perceptions about certain food stuffs lead to obesity in these patients as their lack of knowledge and awareness made them consume excess of calories and carbohydrates. These OSA patients considered food stuffs like “high-fiber biscuits” and packed juices with labels of “no added sugar” as healthy to consume because of labels of “high fiber” or “no added sugar” and consumed them in excess amount leading to obesity. Moreover, they did not even consider them important to even mention these items on their own, during their dietary intake survey. It was also found that patients used refined oils liberally thinking them to be of lower calorific value than animal fat unaware of the fact that almost all oils and fats give approximately the same calories.<sup>26</sup>

### **Customized Diet Plans, Weight Loss, and Maintenance**

Customized or tailor-made diet plans mean food intake calculated according to the BMI and nutritional status of the patient, precise in quality and quantity of macronutrients such as carbohydrates, proteins, and fats to be incorporated daily, and taking care of fulfilling the micronutrient content per day according to the recommended dose. This diet plan also means teaching the patient carbohydrate counting per meal or snack since carbohydrates are the basis of our daily diet and make the largest percentage among all macronutrients to provide energy to our body. Eventually, the target is to make the patients aware and self-sufficient that they incorporate healthy foods in their daily diet as a permanent lifestyle measure and maintain their ideal body weight and biochemical parameters leading a healthy life as a result. To understand this fully, here is a case presentation.

A 45-year-old male obese patient with weight 95 kg, height 180 cm, and BMI 29.3 kg/m<sup>2</sup>, suffered from snoring for the past 2 years. He was taken up for weight loss therapy as the only treatment. His ideal body weight was calculated by formula—50 kg + 2.3 kg, for each inch over 5 feet. It comes to 74–75 kg. Target weight loss –20 kg with weight loss per month at the rate of 4 kg. Total time for therapy is 5 to 6 months.

Energy requirement = 20 kcal/kg body ideal weight

20 × 75 = 1500 kilocalories per day

Distribution of calories from carbohydrates: proteins: fats = 55%:20%:25%

Amount of carbohydrates = 205 g/day

Amount of protein = 75 g/day

Amount of fat = 42 g/day out of which not more than 25–30 g visible fat

Carbohydrate distribution in a day

Breakfast and mid morning = 70 g

Lunch = 65 g

Evening and dinner = 70 g

Quality of carbohydrates—prefer complex carbohydrates over simple ones

Quality of proteins—half of proteins in diet should come from high biological value sources.

### Quality of Fats

Visible/added fats should be from variety of plant sources. Type of vegetable oil should be changed after every three months, for example, sunflower oil, corn oil, and cotton seed oil. Animal fat like clarified butter and white butter and mustard oil should also be a part of the daily diet in ratio of 1:1:1 for PUFA:MUFA:SATFAT. Bakery items and packaged food stuffs which contain trans fat should be totally avoided.

Besides the normal customized diet plans, many other customized diets have been experimented and researched upon like Atkins diets, paleo diets, and keto diets. These diets assure weight loss but have mixed response on weight maintenance. The basic principle all these diets have maintained that when body does not get carbohydrates to be used as a source of energy, it burns fats instead as an active source of energy to be utilized by the body, and as a result, there is a quick and dramatic weight loss. Another principle on which this diet is based is that increased intake of proteins and fats reduces appetite and hence food cravings.<sup>42</sup>

Atkins diet is basically a very-low-carbohydrate diet plan (begins with 20% of carbohydrates per day) with high in fats and proteins. It completely avoids grains, legumes, roots, tubers, and simple sugars. It promotes meats, fatty fish, eggs, full-fat milk, and nuts and oil seeds. Earlier, this diet was considered unhealthy due to high-saturated fat content, but latest research shows that saturated fat is harmless.<sup>43</sup> Keto diet is very low in carbohydrate, as low as 5% per day and rich in fats (75%/day) and is similar to Atkins diet in some aspects. Such drastic reduction of carbohydrates changes the metabolic state of body and puts it into ketosis, which triggers rapid fat metabolism for energy production. It also turns fat into ketones in liver, which supplies energy to the brain. Paleo diet eliminates dairy, grains, pulses, legumes, and processed sugars and encourages consumption of organic meats, wild caught fish, eggs, fruits, vegetables, tubers, and nuts and oil seeds. This diet derives its name from paleolithic age human diet, and recommendations are the same as that what humans used to eat in the Stone Age. It is also named as the caveman diet.

Seeing the above customized diet plans, the first noncompliance is dependent on animal products like meats, fish, and eggs, whereas mostly the Indian population is pure vegetarian. The second compliance issue is that staple diet of carbohydrate sources like Indian breads and rice is completely eliminated. With the whole family eating a different diet and arranging a completely different diet for the patient will definitely affect the compliance since Indian families cook and eat together. Thirdly, these diets encourage organic produce and organic meats, which is very difficult to be procured in today's world of pesticides and insecticides. Therefore, the same seems to be almost impossible for these special diets to be followed for a greater period of time since in reality nobody is living in caves like "hunters and gatherers" and is not hunting animals that are grass- and pasture-fed, and gathering fruits from trees.

The scientifically based customized diet plan according to what a normal human being consumes nowadays is based on its easy availability, eliminating only lack of knowledge about healthy and unhealthy foods and making the patient sensitized and aware of his nutritional status and actual requirement of the right nutrients for his body to eliminate the disease seems to be more practical and can be followed for a lifetime to achieve permanent health goals.

### Surgical Weight Loss vs Lifestyle Management and Weight Loss

There have been guidelines developed in the past as recommendations for obese OSA patients emphasizing the role of specific weight management techniques as an important treatment strategy. The official American Thoracic Society Clinical practice guidelines provide evidence-based recommendations for the management of overweight/obesity as a treatment strategy for OSA. According to the guidelines, behavioral, pharmacological, and surgical treatments promote weight loss and can reduce OSA severity and its comorbidities, thereby improving the life in terms of its quality. These treatment strategies are based on a detailed analysis of the impact of the type of weight loss therapy [depending on the stage of obesity (overweight/obese grade—1, obese grade—2, obese grade—3, and morbid obese) defined by the body mass index (BMI)] on OSA and its co morbidities. These recommendations and guidelines are the result of discussion and consensus between sleep and pulmonary physicians, weight management experts, and behavioral scientists.<sup>43</sup>

For patients with OSA who are overweight or obese (i.e., BMI ≥25 kg/m<sup>2</sup>): a reduced-calorie diet, exercise/increased physical activity, and behavioral counseling in the form of a comprehensive lifestyle intervention program are recommended rather than no lifestyle intervention program.<sup>44–46</sup>

For patients with OSA with a BMI greater than or equal to 27 kg/m<sup>2</sup>, not benefitting from a comprehensive weight-loss lifestyle program, with no contraindications including no active cardiovascular disease, an evaluation for anti-obesity pharmacotherapy was suggested.<sup>47–49</sup>

For patients with OSA with a BMI greater than or equal to 35 kg/m<sup>2</sup>, not benefitting from a comprehensive weight-loss lifestyle intervention program, and with no contraindications, should be referred to bariatric surgery evaluation.<sup>50,51</sup>

There is enough evidence that any program including the three components—reduced calorie diet, exercise/increased physical activity, and behavioral guidance, offers the greatest potential for a successful behavioral approach to the treatment

of overweight/obesity, and hence, a comprehensive lifestyle intervention has been recommended for obesity management guidelines.<sup>52-54</sup>

On the contrary, The American College of Physicians recommended that OSA patients who are obese or overweight should be given at least three trials of the comprehensive lifestyle intervention, but pharmacological therapy or bariatric surgery was not included in the recommendations.

The American Academy of Sleep Medicine and the Canadian Thoracic Society lay emphasis on educating the obese/overweight OSA about the relationship between excess weight and OSA, recommending them to lose weight but there are no recommendations regarding specific strategies to achieve weight loss beyond consideration of bariatric surgery.<sup>55-57</sup>

Evidence suggests that physicians fail to recommend the most effective interventions when counseling on weight-loss strategies.<sup>58</sup> According to a study, the OSA patients responded much better to a specific weight-loss program rather than when simply advised to lose weight. And also, the individuals who were referred to a weight-loss program lost more than twice as much weight as those who were simply advised to lose weight.<sup>59</sup> Thus, an unbiased communication between the clinician and the OSA patient about being overweight or obese, and the remedial measures in the form of a specific weight management program, has the potential not only to improve OSA severity but also to reduce cardio metabolic and other weight-related risks and diseases and improve quality of life.<sup>60</sup>

## CONCLUSION

Weight loss is consistently associated with the improvement in OSA severity, regardless of how the weight loss is achieved. Weight loss may also additionally help in actual resolution of OSA, improvement or prevention of type 2 diabetes mellitus, decreased blood pressure, and improved quality of life. Pharmacological and surgical therapies have mild to moderate risks, whereas behavioral modifications and interventions to weight loss have practically no risks.

Permanent behavioral change in the lifestyle of the patient should be aimed at rather than an experimental change for a few months. Weight loss needs to be a parallel strategy in OSA patients even with other medical and surgical interventions. Eating right at the right time is an important lifestyle change, which should be stressed upon by medical and paramedical teams to the patient as well as his family. One of the most important tools for an effective method of prevention of obesity induced OSA is patient education. By educating the patient and his family about healthy food habits, the nutritionists have an important role to play in nutrition education along with the physicians and paramedical staff so as to provide a better, consistent, and rationale multidisciplinary approach to promote healthy eating among the patients, thus helping in a persistent and sustained weight management of such patients over long term.

## REFERENCES

- Punjabi NM, Sorkin JD, Katzel LI, et al. Sleep-disordered breathing and insulin resistance in middle-aged and overweight men. *Am J Respir Crit Care Med* 2002;165(5):677-682. DOI: 10.1164/ajrccm.165.5.2104087.
- Schwab RJ, Gupta KB, Geftter WB, et al. Upper airway and soft tissue anatomy in normal subjects and patients with sleep-disordered breathing. Significance of the lateral pharyngeal walls. *Am J Respir Crit Care Med* 1995;152(5 Pt 1):1673-1689. DOI: 10.1164/ajrccm.152.5.7582313.
- Soriano-Co M, Vanhecke TE, Franklin BA, et al. Increased central adiposity in morbidly obese patients with obstructive sleep apnoea. *Intern Med J* 2011;41(7):560-566. DOI: 10.1111/j.1445-5994.2010.02283.x.
- Opp MR. Cytokines and sleep. *Sleep Med Rev* 2005;9(5):355-364. DOI: 10.1016/j.smrv.2005.01.002.
- Kimoff RJ, Hamid Q, Divangahi M, et al. Increased upper airway cytokines and oxidative stress in severe obstructive sleep apnoea. *Eur Respir J* 2011;38(1):89-97. DOI: 10.1183/09031936.00048610.
- O'Donnell CP, Schaub CD, Haines AS, et al. Leptin prevents respiratory depression in obesity. *Am J Respir Crit Care Med* 1999;159(5 Pt 1):1477-1484. DOI: 10.1164/ajrccm.159.5.9809025.
- Epstein LJ, Kristo D, Strollo PJ Jr, et al. Clinical guideline for the evaluation, management and long-term care of obstructive sleep apnea in adults. *J Clin Sleep Med* 2009, Jun 15;3(3):263-276.
- Tuomilehto HP, Seppä JM, Partinen MM, et al. Lifestyle intervention with weight reduction: first-line treatment in mild obstructive sleep apnea. *Am J Respir Crit Care Med* 2009;179(4):320-327. DOI: 10.1164/rccm.200805-669OC.
- Johansson K, Hemmingsson E. Longer term effects of very low energy diet on obstructive sleep apnoea in cohort derived from randomised controlled trial: prospective observational follow-up study. *BMJ* 2011;1(342):d3017. DOI: 10.1136/bmj.d3017.
- Tuomilehto H, Gylling H, Peltonen M, et al. Sustained improvement in mild obstructive sleep apnea after a diet- and physical activity-based lifestyle intervention: postinterventional follow-up. *Am J Clin Nutr* 2010;92(4):688-696. DOI: 10.3945/ajcn.2010.29485.
- Tuomilehto H, Seppä J, Uusitupa M. Obesity and obstructive sleep apnea—clinical significance of weight loss. *Sleep Med Rev* 2013;17(5):321-329. DOI: 10.1016/j.smrv.2012.08.002.
- Kuna ST, Reboussin DM, Borradaile KE, et al. Long-term effect of weight loss on obstructive sleep apnea severity in obese patients with type 2 diabetes. *Sleep AHEAD Research Group of the Look AHEAD Research Group. Sleep* 2013;36(5):641A-649A. DOI: 10.5665/sleep.2618.
- Sharma SK, Kumpawat S, Banga A, et al. Prevalence and risk factors of obstructive sleep apnea syndrome in a population of Delhi, India. *Chest* 2006;130(1):149-156. DOI: 10.1378/chest.130.1.149.
- Bixler EO, Vgontzas AN, Ten Have T, et al. Effects of age on sleep apnea in men: I. Prevalence and severity. *Am J Respir Crit Care Med* 1998;157(1):144-148. DOI: 10.1164/ajrccm.157.1.9706079.
- Young T, Shahar E, Nieto FJ, et al. Predictors of sleep-disordered breathing in community-dwelling adults: the Sleep Heart Health Study. *Sleep Heart Health Study Research Group. Arch Intern Med* 2002;162(8):893-900. DOI: 10.1001/archinte.162.8.893.
- Young T, Peppard PE, Gottlieb DJ, et al. Epidemiology of obstructive sleep apnea a population health perspective. *Am J Respir Crit Care Med* 2002;165(9):1217-1239.
- Gami AS, Caples SM, Somers VK, et al. Obesity and obstructive sleep apnea. *Endocrinol Metab Clin North Am* 2003;32(4):869-894. DOI: 10.1016/S0889-8529(03)00069-0.
- Vgontzas AN, Papanicolaou DA, Bixler EO, et al. Sleep apnea and daytime sleepiness and fatigue: relation to visceral obesity, insulin resistance, and hypercytokinemia. *J Clin Endocrinol Metab* 2000;85(3):1151-1158. DOI: 10.1210/jcem.85.3.6484.
- Peppard PE, Young T, Palta M, et al. Longitudinal study of moderate weight change and sleep-disordered breathing. *JAMA* 2000;284(23):3015-3021. DOI: 10.1001/jama.284.23.3015.
- Levers-Landis C, Redline S. Pediatric sleep apnea: implications of the epidemic of childhood overweight. *Am J Respir Crit Care Med* 2007;175(5):436-441. DOI: 10.1164/rccm.200606-790PP.
- Redline S, Storfer-Isser A, Rosen CL, et al. Association between metabolic syndrome and sleep-disordered breathing in adolescents. *Am J Respir Crit Care Med* 2007;176(4):401-408. DOI: 10.1164/rccm.200703-375OC.

22. Schwab RJ, Pasirstein M, Pierson R, et al. Identification of upper airway anatomic risk factors for obstructive sleep apnea with volumetric magnetic resonance imaging. *Am J Respir Crit Care Med* 2003; 168(5):522–530. DOI: 10.1164/rccm.200208-866OC.
23. Naimark A, Cherniack RM. Compliance of the respiratory system and its components in health and obesity. *J Appl Physiol* 1960 May;15:377–382. DOI: 10.1152/jappl.1960.15.3.377.
24. Patel SR, Larkin EK, Redline S. Shared genetic basis for obstructive sleep apnea and adiposity measures. *Int J Obes (Lond)* 2008;32(5): 795–800. DOI: 10.1038/sj.ijo.0803803.
25. Popko K, Gorska E, Wasik M, et al. Frequency of distribution of leptin receptor gene polymorphism in obstructive sleep apnea patients. *J Physiol Pharmacol* 2007 Nov;58(Suppl 5(2)):551–561.
26. Sahni N, Bansal S. Food Perceptions of Obstructive Sleep Apnea Patients. *Online J Health Allied Scs* 2018;17(2):3, available at URL: <https://www.ojhas.org/issue66/2018-2-3.html>.
27. Vgontzas AN, Tan TL, Bixler EO, et al. Sleep apnea and sleep disruption in obese patients. *Arch Intern Med* 1994;154(15):1705–1711. DOI: 10.1001/archinte.1994.00420150073007.
28. Pillar G, Shehadeh N. Abdominal fat and sleep apnea: the chicken or the egg? *Diabetes Care* 2008;31(Suppl 2):S303–S309. DOI: 10.2337/dc08-s272.
29. Smith PL, Gold AR, Meyers DA, et al. Weight loss in mildly to moderately obese patients with obstructive sleep apnea. *Ann Intern Med* 1985;103(6(Pt 1)):850–855. DOI: 10.7326/0003-4819-103-6-850.
30. Nosedá A, Kempnaers C, Kerkhofs M, et al. Sleep apnea after 1 year domiciliary nasal-continuous positive airway pressure and attempted weight reduction. Potential for weaning from continuous positive airway pressure. *Chest* 1996;109(1):138–143. DOI: 10.1378/chest.109.1.138.
31. Newman AB, Foster G, Givelber R, et al. Progression and regression of sleep-disordered breathing with changes in weight: the Sleep Heart Health Study. *Arch Intern Med* 2005;165(20):2408–2413. DOI: 10.1001/archinte.165.20.2408.
32. Dixon JB, Schachter LM, O'Brien PE, et al. Polysomnography before and after weight loss in obese patients with severe sleep apnea. *Int J Obes (Lond)* 2005;29(9):1048–1054. DOI: 10.1038/sj.ijo.0802960.
33. Sahlman J, Pukkila M, Seppä J, et al. Evolution of mild obstructive sleep apnoea after different treatments. *Laryngoscope* 2007;117(6):1107. DOI: 10.1097/MLG.0b013e3180514d08.
34. Berger G, Berger R, Oksenberg A, et al. Progression of snoring and obstructive sleep apnoea: the role of increasing weight and time. *Eur Respir J* 2009;33(2):338–345. DOI: 10.1183/09031936.00075408.
35. Suratt PM, McTier RF, Findley LJ, et al. Effect of very-low-calorie diets with weight loss on obstructive sleep apnea. *Am J Clin Nutr* 1992;56(1):182S–184S. DOI: 10.1093/ajcn/56.1.182S.
36. Braver HM, Block AJ, Perri MG, et al. Treatment for snoring: combined weight loss, sleeping on side, and nasal spray. *Chest* 1995;107(5): 1283–1288. DOI: 10.1378/chest.107.5.1283.
37. Barnes M, Goldsworthy UR, Cary BA, et al. A diet and exercise program to improve clinical outcomes in patients with obstructive sleep apnoea—a feasibility study. *J Clin Sleep Med* 2009;5(05):409–415.
38. Jebb S. Carbohydrates and obesity: From evidence to policy in the UK. *Proc Nutr Soc* 2015;74(3):215–220. DOI: 10.1017/S0029665114001645.
39. Simin L, Manson JE. Dietary carbohydrates, physical inactivity, obesity, and the 'metabolic syndrome' as predictors of coronary heart disease. *Curr Opin Lipidol* 2001;12(4):395–404. DOI: 10.1097/00041433-200108000-00005.
40. Denzer CM, Young JC. The effect of resistance exercise on the thermic effect of food. *International Journal of Sport Nutrition and Exercise Metabolism*. 2003;13(3):396–402.
41. Lau DC, Douketis JD, Morrison KM, et al. 2006 Canadian clinical practice guidelines on the management and prevention of obesity in adults and children summary. *CMAJ (Practice Guideline, Review)* 2007;176(8):S1–S13. DOI: 10.1503/cmaj.061409.
42. McClernon FJ, Yancy WS Jr, Eberstein JA, et al. The effects of a low-carbohydrate ketogenic diet and a low-fat diet on mood, hunger, and other self-reported symptoms. *Obesity (Silver Spring)* 2007;15(1): 182–187. DOI: 10.1038/oby.2007.516.
43. Billings ME, Krishnan V, Su G, et al. Clinical Practice Guideline Summary for Clinicians: The Role of Weight Management in the Treatment of Adult Obstructive Sleep Apnea. *Ann Am Thorac Soc* 2019;16(4): 405–408. DOI: 10.1513/AnnalsATS.201810-708CME.
44. Habdank K, Paul T. A randomized controlled trial evaluating the effectiveness of a weight loss strategy in overweight and obese patients with obstructive sleep apnea (OSA). *Sleep Med* 2006;7(Suppl 2):S73–S74. DOI: 10.1016/j.sleep.2006.07.176.
45. Wu T, Gao X, Chen M, et al. Long-term effectiveness of diet-plus-exercise interventions vs. diet-only interventions for weight loss: a meta-analysis. *Obes Rev* 2009;10(3):313–323. DOI: 10.1111/j.1467-789X.2008.00547.x.
46. Johns DJ, Hartmann-Boyce J, Jebb SA, et al. Behavioural Weight Management Review Group. Diet or exercise interventions vs combined behavioral weight management programs: a systematic review and meta-analysis of direct comparisons. *J Acad Nutr Diet* 2014;114(10):1557–1568. DOI: 10.1016/j.jand.2014.07.005.
47. Winslow DH, Bowden CH, DiDonato KP, et al. A randomized, double-blind, placebo-controlled study of an oral, extended-release formulation of phentermine/topiramate for the treatment of obstructive sleep apnea in obese adults. *Sleep* 2012;35(11):1529–1539. DOI: 10.5665/sleep.2204.
48. Blackman A, Foster GD, Zammit G, et al. Effect of liraglutide 3.0 mg in individuals with obesity and moderate or severe obstructive sleep apnea: the SCALE Sleep Apnea randomized clinical trial. *Int J Obes* 2016;40(8):1310–1319. DOI: 10.1038/ijo.2016.52.
49. Rössner S, Sjöström L, Noack R, et al. European Orlistat Obesity Study Group. Weight loss, weight maintenance, and improved cardiovascular risk factors after 2 years treatment with orlistat for obesity. *Obes Res* 2000;8(1):49–61. DOI: 10.1038/oby.2000.8.
50. Dixon JB, Schachter LM, O'Brien PE, et al. Surgical vs conventional therapy for weight loss treatment of obstructive sleep apnea: a randomized controlled trial. *JAMA* 2012;308(11):1142–1149. DOI: 10.1001/2012.jama.11580.
51. Feigel-Guiller B, Drui D, Dimet J, et al. Laparoscopic gastric banding in obese patients with sleep apnea: a 3-year controlled study and follow-up after 10 years. *Obes Surg* 2015;25(10):1886–1892. DOI: 10.1007/s11695-015-1627-5.
52. Balshem H, Helfand M, Schünemann HJ, et al. GRADE guidelines: 3. Rating the quality of evidence. *J Clin Epidemiol* 2011;64(4):401–406. DOI: 10.1016/j.jclinepi.2010.07.015.
53. GRADEpro GDT: GRADEpro Guideline Development Tool [software]. McMaster University; 2015 (developed by Evidence Prime, Inc.).
54. Schünemann HJ, Jaeschke R. ATS Documents Development and Implementation Committee. An official ATS statement: grading the quality of evidence and strength of recommendations in ATS guidelines and recommendations. *Am J Respir Crit Care Med* 2006;174(5):605–614. DOI: 10.1164/rccm.200602-197ST.
55. Qaseem A, Holty JE, Owens DK, et al. Management of obstructive sleep apnea in adults: a clinical practice guideline from the American College of Physicians. *Ann Intern Med* 2013;159(7):471–483.
56. Fleetham J, Ayas N, Bradley D, et al. CTS Sleep Disordered Breathing Committee. Canadian Thoracic Society guidelines: diagnosis and treatment of sleep disordered breathing in adults. *Can Respir J* 2006;13(7):387–392. DOI: 10.1155/2006/627096.
57. Morgenthaler TI, Kapen S, Lee-Chiong T, et al. Standards of Practice Committee; American Academy of Sleep Medicine. Practice parameters for the medical therapy of obstructive sleep apnea. *Sleep* 2006;29(8):1031–1035. DOI: 10.1093/sleep/29.8.1031.
58. Phelan S, Nallari M, Darroch FE, et al. What do physicians recommend to their overweight and obese patients? *J Am Board Fam Med* 2009;22(2):115–122. DOI: 10.3122/jabfm.2009.02.080081.
59. Aveyard P, Lewis A. Screening and brief intervention for obesity in primary care: a parallel, two-arm, randomised trial. *Lancet* 2016;388:2492–2500. DOI: 10.1016/S0140-6736(16)31893-1.
60. Rose SA, Poynter PS, Anderson JW, et al. Physician weight loss advice and patient weight loss behavior change: a literature review and meta-analysis of survey data. *Int J Obes* 2013;37(1):118–128. DOI: 10.1038/ijo.2012.24.