

Review Article

## Evaluation and Management of Obstructive Sleep Apnoea in Children with Obesity

Haya Alsubie, MD\*, Faleh Alsubaie<sup>1</sup>, Hamdi Alsufiani, MD<sup>2</sup>

1. College of Medicine, Al-Imam Mohammad Ibn Saud Islamic University, 13317, Riyadh, Saudi Arabia.
2. Paediatric Respiratory Unit, Madina Maternity and Children's Hospital, Madina, Saudi Arabia.

\***Corresponding Author: Dr. Haya Alsubie, MD**, Sleep Disorder Centre, Respiratory Unit, Ad Diriyah Hospital, Ar Rihab 13717, Riyadh, Saudi Arabia.

**Received Date:** March 27, 2021

**Publication Date:** April 01, 2021

### Abstract

Obstructive sleep apnoea (OSA)<sup>2</sup> occurs commonly and more severely in children and adolescents with obesity. The increasing prevalence of obesity is associated with an increasing prevalence of OSA. The clinical approach to OSA in children with obesity has not been standardized. This review focused on the updated evidence available regarding clinical approaches to OSA in children with common obesity.

**Keywords:** Pediatric, obstructive sleep apnea, polysomnography, positive airway pressure, sleep, sleep-disordered breathing.

### Abbreviations:

AHI: Apnoea-Hypopnoea index

ATH: Adenotonsillar Hypertrophy,

BMI: Body mass index,

DISE: Drug-Induced Sleep Endoscopy,

EDS: Excessive Daytime Sleepiness,



INCS: Intranasal Corticosteroids,  
MRI: Magnetic Resonance Imaging,  
OSA: Obstructive Sleep Apnoea,  
PAP: Positive Airway Pressure,  
PSG: Polysomnography,  
SCR: Sleep Clinical Record,  
SDB: Sleep Disordered Breathing,  
T&A: Adenotonsillectomy,  
UPPP: Uvulo Palate Pharyngo Plasty

## Introduction

The prevalence of childhood obesity has increased at an alarming rate over the past decades, with major short- and long-term negative health implications. Simultaneously, the prevalence of obstructive sleep apnoea (OSA) has also increased. From 1975 to 2016, the mean body mass index (BMI) trend in children and adolescents increased globally [1]. For every increment in BMI of 1 kg/m<sup>2</sup> above the 50th percentile, the risk of OSA increases by 12% [2]; therefore, the severity of OSA is proportional to the degree of obesity [3]. Approximately 25–45% of children with obesity have OSA [4] compared with 2–4% of their counterparts with a normal weight [5]. In children, the typical OSA presentation of Aden tonsillar hypertrophy (ATH) and failure to thrive has been predominantly replaced by that of poor weight control and obesity [6]. The presence of obesity in children with OSA increases OSA-related morbidity. Thus, recognizing residual OSA in children with obesity should become a priority to prevent subsequent long-term complications.

## Childhood Obesity

Obesity and overweight in children are defined based on the BMI, a simple tool used in the clinical setting. However, this definition slightly differs between different organizations based on the BMI and percentile cut-off point used (Table 1) [7]. Obesity is diagnosed based on the calculation of the BMI, which is then plotted on a special growth chart concerning the child's age and sex. Several other methods have been used to define obesity, such as waist-to-hip ratio, and dual-energy X-ray absorptiometry, which are more accurate for evaluating obesity than BMI; however, its application in the clinical setting is difficult and more complex. BMI is the standard diagnostic tool to assess obesity and overweight in children and adolescents. Because children's BMI varies with age, a single definition cannot be used. Using BMI percentile charts in children can improve its diagnostic accuracy and utility ([https://www.cdc.gov/growthcharts/clinical\\_charts.htm](https://www.cdc.gov/growthcharts/clinical_charts.htm)).

**Table 1.** BMI and percentiles

Definitions of childhood obesity	CDC	WHO	IOTF
Overweight	85 <sup>th</sup> -95 <sup>th</sup>	85 <sup>th</sup> -97 <sup>th</sup>	91 <sup>st</sup>
Obesity	>95 <sup>th</sup>	>97 <sup>th</sup>	99 <sup>th</sup>

**CDC:** Center for Disease Control and Prevention, **WHO:** World Health Organisation: International Obesity Taskforce.

## Obstructive Sleep Apnoea

Sleep-disordered breathing (SDB) is not a distinct disease, but rather a broad spectrum of sleep-related breathing disorders. At the low end of the severity spectrum of SDB is primary snoring, defined as observed snoring without apnoea, abnormalities in gas exchange, or excessive arousal. This disorder is further classified into frank OSA, of varying severity, and upper airway resistance syndrome. The latter includes fragmented sleep due to increased respiratory effort related to arousal but is distinguished by the fact that oxygenation is not affected. OSA is an important clinical entity within the spectrum of SDB, characterized by repeated episodes of prolonged partial or complete upper airway obstruction during sleep, resulting in gas exchange abnormalities and sleep fragmentation. OSA is common among adults, and is becoming increasingly common among children, and is currently recognized as a major public health concern [8] [5] [9] [10].

## Diagnosis

### History and physical examination

OSA causes both nocturnal and diurnal symptoms. The most common nocturnal symptom is snoring. However, symptoms alone have a high negative predictive value and are not reliable to detect OSA in snoring children [11]. The extent to which OSA affects a child's daytime function in school or their quality of life (QOL) can be assessed clinically via pediatric sleep questionnaires [12]. Neurobehavioral sequelae often exist and are thought to be a result of the effects of chronic intermittent hypoxemia and fragmented sleep on the prefrontal cortex (the area of the brain associated with the ability to plan and organize) [13], [14]. Cognitive dysfunction in children with OSA is dose-dependent; the effect appears even in children with mild OSA and is marked in those with moderate-to-severe OSA [15]. A comprehensive assessment of sleep hygiene should be performed. The physical examination should include both evaluations of risk factors, such as tonsillar hypertrophy and obesity, and examination for potential consequences of OSA, such as systemic hypertension and signs of pulmonary hypertension (Table 2).

**Table 2.** Common symptoms and signs of obstructive sleep apnoea

Nocturnal symptoms	Snoring Increased breathing effort Witnessed apnoea Restless sleep Oral breathing Sleeping in unusual positions/hyperextended neck Secondary nocturnal enuresis
Diurnal symptoms upon waking	Difficulty waking in the morning Irritability and anger upon waking Early morning headache
Diurnal symptoms	Learning difficulties Excessive daytime sleepiness Behavioural disturbances Unusual aggressiveness, and even social withdrawal
Physical examination	Failure to thrive or obesity Craniofacial abnormalities, adenoidal face Underlying syndromes: e.g. Down syndrome, Pierre Robin syndrome Nasal and oral cavity: high arched palate, crowded oropharynx, adenotonsillar hypertrophy, lingual tonsil enlargement, reduced peritonsillar space, large tongue Signs of long-standing obstruction: pectus excavatum, Harrison's sulcus Signs of pulmonary hypertension such as loud P2 Systemic hypertension Nervous system examination: hypotonia

To differentiate between primary snoring and OSA, the Sleep Clinical Record (SCR), a simple PSG-validated tool based on three elements, can be used: physical examination, subjective symptoms, and clinical history; the clinical history analyses behavioral and cognitive problems. Patients with a total SCR score of 6.5 or more are considered to be positive for OSA, with an 80% sensitivity [16].



### Polysomnography

Polysomnography (PSG) is the gold standard to use for the diagnosis and assessment of the severity of OSA. The criteria for a PSG diagnosis require either  $\geq 1$  obstructive sleep events or obstructive hypoventilation. The latter is identified by the partial pressure of end-tidal CO<sub>2</sub> (PETCO<sub>2</sub>) of  $>50$  mm Hg for  $>25\%$  of sleep time and/or arterial oxygen desaturation coupled with snoring, paradoxical thoracoabdominal movement, or flattening of the nasal airway pressure waveform [17]. Obstructive apnoea is defined by the absence of nasal airflow, despite the presence of chest and abdominal wall movement for a duration of at least two breaths, whereas obstructive hypopnoea refers to a decrease in nasal airflow of 50% from the baseline, accompanied by a fall in oxygen saturation of 3% and/or arousal from sleep.

The severity of OSA is expressed as the mean number of apnoea and hypopnoea episodes per hour of sleep (apnoea-hypopnoea index, AHI); severity is ranked as mild (AHI 1–5), moderate (AHI 5–10), or severe OSA (AHI  $>10$ ) [18]. There is controversy regarding the indication for PSG in children. The American Academy of Sleep Medicine [19], American Academy of Pediatrics [9], and European Respiratory Society guidelines [20] recommend PSG for all children when the clinical assessment is suggestive of OSA. However, the American Academy of Otolaryngology-Head and Neck Surgery [21] favors the limited use of PSG in high-risk groups, including children  $<2$  years of age; those with obesity, Down syndrome, craniofacial abnormalities, neuromuscular disorders, sickle cell disease, or mucopolysaccharidoses; or when there is discordance between the history of OSA and tonsillar size upon physical examination.

### Cervical ultrasonography

The calculated tonsil volume on a cervical ultrasound has been demonstrated to match the actual tonsil volume in children. Without the risk of radiation or need for sedation, ultrasonography is a safe diagnostic tool for tonsil diseases. As a novel approach for assessing obstructive tonsillar hypertrophy in children, ultrasonic assessment has recently been recognized for assessing the tonsillar size. It is a new, safe, and easily applicable modality used in children, which may provide value and guidance as a decision-making tool for tonsillectomy. The degree of airway obstruction due to tonsillar hypertrophy can be objectively determined by ultrasonography in children. An inter tonsillar distance /transverse length of the tongue base ratio of 0.3 or less is compatible with obstructive tonsillar hypertrophy [22] [23].



### Upper airway visualization

Multiple radiological images are currently available to visualize upper airway abnormalities in children with OSA. In clinical practice, it is reserved for diagnosis and management in children with comorbid conditions and complex upper airway abnormalities. Lateral neck radiography (cephalometry), fluoroscopy, and computed tomography are performed while the patient is awake and in an upright position; thus, these procedures may not represent the collapse of the upper airway during sleep. Furthermore, they provide a certain amount of radiation exposure. Given these limitations, novel techniques, including dynamic magnetic resonance imaging (MRI) and drug-induced sleep endoscopy (DISE), have emerged as alternatives for better direct visualization of the upper airway, pinpointing areas causing obstruction and guiding further surgical intervention and tailored personalized treatment strategies [24]. It is important to note that these modalities are useful as an adjunct to PSG to identify levels of obstruction and treatment selection, but are not meant to replace the gold standard of PSG [25].

### MRI sleep study

MRI is a useful tool for identifying and localizing isolated or multiple anatomic and dynamic causes of residual OSA, which can then be targeted surgically [26]. MRI studies affect treatment decisions in >50% of complex OSA cases [27]. They are usually ordered by an otolaryngologist familiar with the use of the MRI sleep study to direct surgical procedures, who can then offer a suitable surgical option following standard protocols [28]. An anesthesiologist is also required to induce general anesthesia in children with critical or difficult airways. Studies concerning MRI-directed surgical outcomes are rare; however, a meta-analysis of 68 patients revealed that all studies reported a reduction in AHI. The mean change in AHI was significant at -7.37 (95% confidence interval (CI), -10.42 to -4.32;  $p < 0.001$ ,  $I^2 = 0\%$ ), as was the change in minimum arterial oxygen saturation (SaO<sub>2</sub>) at 3.53% [29]. Currently, the majority of institutions do not use MRI studies routinely in their clinical approach to residual OSA [28].

### Drug-induced sleep endoscopy

DISE consists of a flexible endoscopic exploration of the upper airway under conscious sedation. Propofol causes dose-dependent excessive muscle relaxation and airway collapse, which presents as steady narrowing of the pharyngeal airway in infants, or the epiglottis in older children [30]. The dexmedetomidine and ketamine protocol, which induces sedation that mimics non-rapid eye movement sleep, is safe and often used for pediatric DISE [31]. The main aim of DISE is to create an upper airway condition analogous to that during sleep. DISE enables the dynamic evaluation of upper airway changes, identification of the area of obstruction, and subsequent surgical treatment planning [32]. A DISE-directed surgical procedure is a sensible step in children with residual OSA post adenotonsillectomy



(T&A) who are unable to tolerate, or unwilling to adopt, positive airway pressure (PAP) therapy [33]. The most common DISE-directed surgical procedures in children include lingual tonsillectomy, supraglottoplasty, nasal surgery, revision adenoidectomy, uvulopalatopharyngoplasty (UPPP), and posterior midline glossectomy [34].

Despite the increasing popularity in using the DISE technique, there are few studies evaluating outcomes after DISE-directed surgical procedures. A small retrospective study found that DISE, using dexmedetomidine/ketamine, was useful to predict at least one site of obstruction, even in patients with rapid eye movement–predominant OSA. DISE-directed outcomes resulted in improvements in the mean AHI, total sleep time, sleep efficiency, saturation nadir, and proportion of patients with AHI <5 after surgery. However, in this study, more children with moderate or severe OSA were included than those with mild OSA [35]. A small prospective study of 20 healthy children with residual OSA after T&A reported a decreased mean AHI, from 6.1 to 1.9, following DISE-directed surgical procedures [36]. DISE should only be performed after expert evaluation. Any procedures that require sedation in children with OSA should be carried out with caution since they can affect both the upper airway muscle tone and ventilatory response, which could lead to respiratory decompensation [37].

## Management

### Multidisciplinary approach

OSA management in children with obesity is challenging and requires a multidisciplinary approach with comprehensive longitudinal assessments from an expert team, including specialists in sleep, pulmonology, otolaryngology, endocrinology, nutrition, and behavioral science. Current evidence supports the connection between OSA and cardiometabolic morbidity in the pediatric population. Correspondingly, children with severe OSA are often referred to a cardiologist before surgery, as are children with explicit cardiac manifestations (elevated blood pressure or signs of pulmonary hypertension). Fig. 1 demonstrates the suggested management approach in children with obesity and snoring. The evaluation of residual OSA should focus on identifying the causes of upper airway obstruction using physical examination, DISE, or MRI. Intervention should be tailored according to the needs of the patient and should address the symptoms, sites of obstruction, and any preference for surgical versus medical management. Early identification can prevent short- and long-term complications, not only in childhood but also in adulthood.

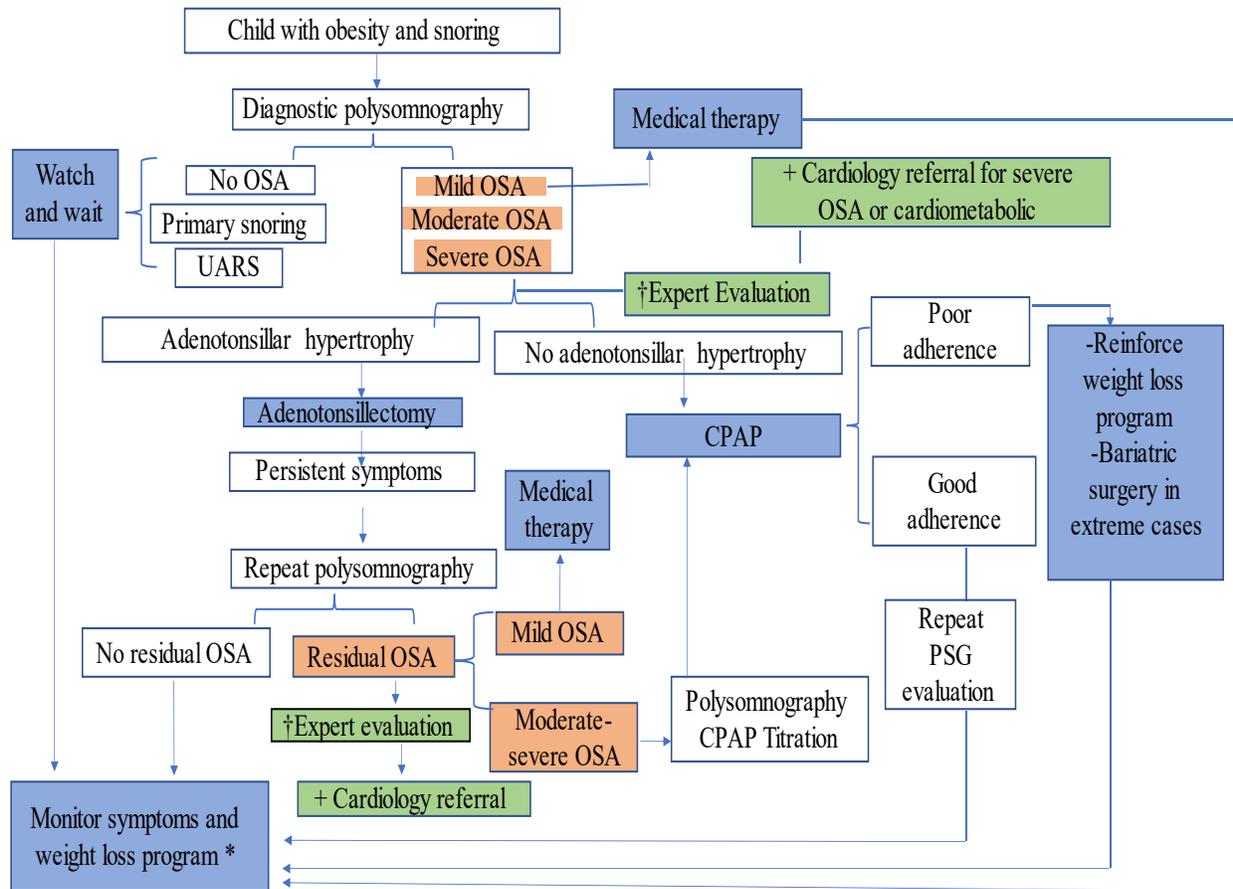


Fig. 1. Suggested approach to the management of children with obesity and snoring

Orange: OSA severity; blue: treatment portions; green: expert evaluation

†Expert Evaluation: Pulmonology, otolaryngology, sleep medicine, endocrinology, dietetic. OSA: obstructive sleep apnoea, UARS: upper airway resistance syndrome, CPAP: continuous positive airway pressure, PSG: polysomnography.

\*Dietary modification, exercise, and lifestyle changes are necessary for all children with obesity.

**Management:**

**Surgical management**

**Adenotonsillectomy**

There is little doubt that hypertrophy of the upper airway lymphoid tissue is the primary contributor to OSA in children, as 50% of children with obesity and OSA have ATH. The American Academy of Pediatrics clinical practice guidelines recommend T&A as the first line of management [9]. Many studies



have delineated that children with obesity and OSA benefit less from T&A compared to children with a normal weight [38], [39], suggesting that obesity may play a decisive role in the pathogenesis of OSA. A cross-sectional, multicentre, prospective study showed a reduction in the severity of OSA experienced by the majority of children with obesity and moderate-to-severe OSA and ATH who underwent T&A; however, >40% had residual OSA [40]. The reason for the high failure rates is unclear; however, some studies have suggested some possibilities.

In a study by Nandalike et al., 21 children with obesity and OSA were evaluated before and after T&A via MRI of the head, obtained while they were awake, and volumetric analysis of the upper airway and surrounding tissues. Their results revealed significant residual adenoid tissue and an increase in the volume of the tongue and soft palate post-T&A [41]. Similarly, a randomized, childhood T&A trial was designed to evaluate the efficacy of early T&A versus monitoring with supportive care in a cohort of 397 children with OSA (AHI >2) or an obstructive apnoea index of >1/h. Among these, 135 children were obese (BMI >95th percentile). In those undergoing early T&A (n=194), the residual OSA was significantly greater in children with obesity compared to those who were not obese—32% vs 15%, respectively (odds ratio: 2.69; 95% CI: 1.33–5.45) [38]. Thus, pre-and post-T&A sleep studies should be conducted among children with obesity and OSA.

### **Other airway surgeries**

#### **Uvulopalatopharyngoplasty**

UPPP aims to enlarge the retropalatal airways by resecting the posterior and anterior lateral pharyngeal pillars and eradicating the uvula and posterior portion of the palate. UPPP has no role in pediatric OSA and is not widely performed in children as it may be associated with velopharyngeal insufficiency, dysphagia, and nasopharyngeal stenosis [42]. Studies on UPPP are sparse and so far demonstrate poor efficacy [43]. The available case series that showed some beneficial use of UPPP when combined with adenotonsillectomy was in children with a neurological impairment who had significant obstructive apnoea localized to the posterior oropharynx, however that improvement was temporary [44].

### **Other surgical management options**

#### **Bariatric surgery**

Bariatric surgery in children is more effective than conventional weight management strategies and is plausible when children with OSA are morbidly obese, more skeletally mature, and have failed organized trials of behavioral weight loss [45]. The benefits of bariatric surgery in maintaining QOL after 10 years are unclear. Kalra et al. [46] enrolled 34 children with obesity (mean BMI, 57±10.1 kg/m<sup>2</sup>; range, 48–87 kg/m<sup>2</sup>) who underwent gastric bypass surgery. Before surgery, 19 children (56%) were diagnosed



with OSA (AHI >5). After surgery, 10 children with OSA returned for follow-up sleep studies after an average of 5.1 months. After a mean weight loss of 58 kg, the median AHI decreased from 9.10 to 0.65 ( $p < 0.01$ ). OSA persisted in 10% of the children after weight loss.

A recent controlled prospective study was performed on the clinical course of OSA obese adolescent and young adults after vertical sleeve gastrectomy or gastric bypass using PSG before and at 3 and 5 weeks after bariatric surgery. The baseline mean (range) age of participants was 17.8 (15.4–20.7) years, with a body mass index of 55.2 (41.3–61.6) kg/m<sup>2</sup> and had a median apnoea hypopnea index (AHI) of 15.8 (7.1–23.8) events/hour. AHI declined postoperatively from baseline by 9.2 events/hour (95% confidence interval: 3.8 to 14.5) at 3 weeks ( $P = .002$ ) and 9.1 events/hour (95% confidence interval: 3.8–14.5) at 5 weeks ( $P = .002$ ); there was no significant change from 3 to 5 weeks in AHI.

Leptin decreased and orexin levels increased significantly by 3 weeks postoperatively. This study concluded that adolescents and young adults with OSA respond early and out of proportion to weight loss after metabolic and or bariatric surgery. Thus, weight-independent factors may at least in part be responsible for the early improvement of OSA postoperatively [47]. There is an obvious need for prospective well-controlled studies to evaluate the long- and short-term efficiency of different management modalities as well as the degree of weight loss and their effect on OSA in children with obesity. Additionally, the effect of weight loss in comparison to continuous PAP (CPAP) should be evaluated.

### **Non-surgical management**

#### **Positive airway pressure therapy**

PAP therapy is commonly used to treat children with moderate-to-severe residual OSA or as a primary intervention in children with no evidence of upper airway lymphoid tissue hypertrophy. PAP is used to mechanically stent the upper airway and maintain its patency throughout the respiratory cycle during sleep. While PAP is a highly effective therapy, adherence is a major challenge at all ages. A multicentre study of children randomly assigned to 6 months of CPAP or bi-level PAP revealed that 28% dropped out before the end of the study, with no difference in adherence between CPAP and bi-level PAP (mean nightly use:  $3.8 \pm 3.3$  hours) [48]. Excellent adherence is possible if PAP therapy is initiated in a specialized pediatric non-invasive ventilation inpatient unit, staffed with experts, using desensitization and behavioral interventions, frequent home visits, and periodic follow-up sleep studies [49].

In a retrospective study, written questionnaires determined the safety and efficacy of nasal CPAP in 9 pediatric sleep centers, including 94 children who were <19 years old (25 who were obese). The



effectiveness of CPAP (defined as the resolution of clinical symptoms, normoxia during sleep, and a marked improvement in PSG parameters) was reported in 81 children [50]. Few studies have investigated the impact of PAP therapy on daytime functioning in children with OSA. A study of 13 adolescents with obesity and OSA showed an improvement in school performance, vigilance, and school-related QOL in those who were adherent to PAP therapy [51]. It has been hypothesized that PAP therapy may induce weight loss in children with obesity by improving excessive daytime sleepiness (EDS) and favoring physical activity. Marcus et al. examined this relationship in a prospective, multicentre study that included 20 children who were randomly assigned to receive either CPAP or bi-level PAP therapy; no difference in BMI was found before and after 6 months of PAP therapy [48].

Although there are clear benefits to PAP therapy, suboptimal adherence is not the only issue, with others frequently including nasal bridge pressure sores from the masks, abdominal distension, oronasal dryness, eye irritation, and overall discomfort from air leaks. When PAP is used at a very young age, flattening of the midface or maxillary retrusion due to longstanding pressure from the mask may occur and requires monitoring with digital photography.

### **Weight loss**

Since OSA is caused by pharyngeal collapse or dysfunction, the deposition of fat in the pharynx of children with obesity may explain why obesity is a risk factor for OSA. Few studies have explored the efficacy of weight loss as treatment—all these studies [46], [52]–[54] showed that weight loss improved OSA significantly. However, it is unknown how much weight loss is needed to relieve OSA [52]. The prevalence of residual OSA is reported as 33–38% after behavioral weight loss intervention, whereas 10–18% is reported after surgical weight loss intervention in some studies [46], [53].

Verhulst et al. [53] evaluated the effect of behavioral weight loss intervention in children with OSA using a multidisciplinary approach consisting of dietary restriction, physical activity, and psychological support. Sixty-one children with obesity, mean age 14.8±2.3 years, with a mean BMI of 37.5±5.7 kg/m<sup>2</sup>, were enrolled. Before the intervention, 61% of the children were diagnosed with OSA (AHI >2). Twenty-one of these children underwent a follow-up sleep study after an average of 5.2 months. Following a median weight loss of 24 kg, the AHI declined from a median of 3.8 to 1.9 ( $p = 0.002$ ), although OSA persisted in 38% of the children. This study included a small number of participants and was not a laboratory sleep study; thus, arousal events may be missed. Furthermore, weight loss was caused by a domestic treatment program and was not compared with treatment programs in outpatient clinics.

**Anti-inflammatory medications**

A meta-analysis showed that oral montelukast improves AHI by 55% when used alone, and by 70% when used in combination with intranasal corticosteroids (INCS). Children with obesity and children >7 years were less likely to benefit from anti-inflammatory medication. Younger children (<7 years) were 2.3 times more likely to normalize their sleep studies after combined INCS and oral montelukast than children with obesity (95% CI, 1.43–4.13;  $p < .001$ ), whereas children who were not obese were 6.3 times more likely to normalize their sleep studies after combined INCS and oral montelukast than children with obesity (BMI z-score >1.65; 95% CI: 4.23–11.18;  $p < .000001$ ) [55]. Thus, montelukast plays no role in OSA in children with obesity.

**Prebiotics oral/nasal sprays as an adjunct therapy for recurrent respiratory infections and adenotonsillar hypertrophy**

Using probiotics as oral/nasal sprays as an adjunct therapy for recurrent respiratory infections and adenotonsillar hypertrophy has been recently hypothesized, and its effects have been demonstrated by some studies. In a prospective study of 42 children with SDB, anamnestic and general examination data were collected using the SCR questionnaire during the first inspection and after 3 months of treatment with *Streptococcus salivarius* 24SMBc nasal spray. After 3 months, the enrolled patients showed lower SCR scores than that during the first inspection (6.0 vs 7.5  $p < 0.000$ ), with a significant reduction in nasal obstruction ( $p = 0.001$ ) and oral breathing ( $p = 0.04$ ), and a positive Brouillette Score ( $p = 0.001$ ). The children and parents did not declare any adverse reactions during the 3-month treatment [56][57]; [58].

**Oral appliances**

Oral appliances include tongue devices and mandibular advancement devices that move the tongue and mandible forward and away from the posterior pharynx to expand the upper airway. Oral appliances are effective in adults with mild OSA, who do not tolerate or are not candidates for CPAP [59]. In children, the efficacy of oral appliances has not been confirmed, although they may be appropriate for use in older children with acceptable dentition. In a small study of children with mild OSA and dysgnathia, oral jaw-positioning appliances reduced AHI, improved sleep, and mitigated EDS [60]. However, there have only been limited studies to support these findings, and no studies were performed for OSA in children with obesity.

**Myofunctional therapy**

This is a modified treatment for OSA in adults and children to correct undesirable changes in the upper airway musculature. For structural intervention, patients learn how to perform daily oropharyngeal



exercises to improve labial tone. This practice enhances the use of nasal breathing as the preferred respiratory route, leading to strengthening of the tongue and orofacial muscles, and reinforces the correct intraoral position [61]. Challenges related to this therapy include compliance, the need for continued parental involvement, and long-term outcome uncertainties [62]. So far, there is no evidence of myofunctional therapy improving symptoms of OSA in children with obesity.

### **Positional therapy**

In adults, OSA severity is worse in the supine position because the tongue collapses into the oropharynx, leading to more severe obstructive events. In children, however, studies show mixed results on whether OSA severity is worse in the supine versus lateral position [63]. Although studies in adults show improvements in OSA with positional therapy, there are no published studies in children evaluating its long-term effectiveness and safety or compare it with other modalities to treat OSA in children with obesity. Positional therapy as a potential treatment option for children with obesity with persistent OSA after adenotonsillectomy warrants further investigation.

### **Childhood obesity prevention**

Avoiding food restriction, focusing on a healthy diet with the family, having exercise routines, and preventing bullying can help prevent childhood obesity. Research has shown that rigid restriction or control of a child's diet can trigger eating disorders. Prevention programs that involve the entire family, encourage children to make healthy food choices and increase their physical activity are most likely to lead to a positive and steady change [64]. Prevention programs must address bullying and stigmatization of children with obesity. [65]. Interventions that target entire populations, rather than focusing on individuals who are overweight or obese, are less likely to cause discriminatory and stigmatizing issues [66].

### **Conclusion**

The global prevalence of obesity in children and adolescents has increased alarmingly, paralleling the increase in OSA. The management of OSA in children and adolescents with obesity is challenging and requires a multidisciplinary team. The evaluation of residual OSA should focus on identifying the causes of upper airway obstruction. Intervention should be tailored to address patient symptoms, sites of obstruction, and preference for surgical versus medical management.



## References

1. N. R. F. C. (NCD-RisC), “Worldwide trends in body-mass index, underweight, overweight, and obesity from 1975 to 2016: a pooled analysis of 2416 population-based measurement studies in 128.9 million children, adolescents and adults,” *Yearb. Paediatr. Endocrinol.*, 2018 DOI:10.1530/ey.15.13.20.
2. S. Redline, P. V. Tishler, M. Schluchter, J. Aylor, K. Clark, and G. Graham, “Risk factors for sleep-disordered breathing in children: Associations with obesity, race, and respiratory problems,” *Am. J. Respir. Crit. Care Med.*, vol. 159, no. 5 I, pp. 1527–1532, 1999 DOI:10.1164/ajrccm.159.5.9809079.
3. Z. Xu, A. Jiaqing, L. Yuchuan, and K. Shen, “A case-control study of obstructive sleep apnea-hypopnea syndrome in obese and nonobese Chinese children,” *Chest*, vol. 133, no. 3, pp. 684–689, Mar. 2008 DOI:10.1378/chest.07-1611.
4. R. Arens et al., “Upper airway structure and body fat composition in obese children with obstructive sleep apnea syndrome,” *Am. J. Respir. Crit. Care Med.*, vol. 183, no. 6, pp. 782–787, 2011 DOI:10.1164/rccm.201008-1249OC.
5. J. C. Lumeng and R. D. Chervin, “Epidemiology of pediatric obstructive sleep apnea,” *Proceedings of the American Thoracic Society*, vol. 5, no. 2, pp. 242–252, Feb. 15, 2008 DOI:10.1513/pats.200708-135MG.
6. E. Dayyat, L. Kheirandish-Gozal, and D. Gozal, “Childhood Obstructive Sleep Apnea: One or Two Distinct Disease Entities?,” *Sleep Medicine Clinics*, vol. 2, no. 3, NIH Public Access, pp. 433–444, Sep. 2007 DOI:10.1016/j.jsmc.2007.05.004.
7. “Dinsdale H, Ridler C, Ells L J. A simple guide to classifying body mass index in children. Oxford: National Obesity Observatory, 2011.”
8. P. E. Peppard, T. Young, J. H. Barnet, M. Palta, E. W. Hagen, and K. M. Hla, “Increased prevalence of sleep-disordered breathing in adults,” *Am. J. Epidemiol.*, vol. 177, no. 9, pp. 1006–1014, May 2013 DOI:10.1093/aje/kws342.
9. C. L. Marcus et al., “Diagnosis and management of childhood obstructive sleep apnea syndrome,” *Pediatrics*, vol. 130, no. 3, Sep. 2012 DOI:10.1542/peds.2012-1672.
10. W. T. McNicholas, Y. Luo, and N. Zhong, “Sleep apnoea: A major and under-recognised public health



- concern,” *Journal of Thoracic Disease*, vol. 7, no. 8. Pioneer Bioscience Publishing, pp. 1269–1272, 2015 DOI:10.3978/j.issn.2072-1439.2015.07.29.
11. J. L. Carroll, S. A. McColley, C. L. Marcus, S. Curtis, and G. M. Loughlin, “Inability of clinical history to distinguish primary snoring from obstructive sleep apnea syndrome in children,” *Chest*, vol. 108, no. 3, pp. 610–618, Sep. 1995 DOI:10.1378/chest.108.3.610.
  12. R. D. Chervin, K. Hedger, J. E. Dillon, and K. J. Pituch, “Pediatric sleep questionnaire (PSQ): Validity and reliability of scales for sleep-disordered breathing, snoring, sleepiness, and behavioral problems,” *Sleep Med.*, vol. 1, no. 1, pp. 21–32, Feb. 2000 DOI:10.1016/S1389-9457(99)00009-X.
  13. P. M. Macey, R. Kumar, M. A. Woo, E. M. Valladares, F. L. Yan-Go, and R. M. Harper, “Brain structural changes in obstructive sleep apnea,” *Sleep*, vol. 31, no. 7, pp. 967–977, Jul. 2008 DOI:10.5665/sleep/31.7.967.
  14. Y. J. Eun et al., “Reduced brain gray matter concentration in patients with obstructive sleep apnea syndrome,” *Sleep*, vol. 33, no. 2, pp. 235–241, Feb. 2010 DOI:10.1093/sleep/33.2.235.
  15. S. J. Hunter, D. Gozal, D. L. Smith, M. F. Philby, J. Kaylegian, and L. Kheirandish-Gozal, “Effect of sleep-disordered breathing severity on cognitive performance measures in a large community cohort of young school-aged children,” *Am. J. Respir. Crit. Care Med.*, vol. 194, no. 6, pp. 739–747, Sep. 2016 DOI:10.1164/rccm.201510-2099OC.
  16. M. P. Villa et al., “Sleep clinical record: An aid to rapid and accurate diagnosis of paediatric sleep disordered breathing,” *Eur. Respir. J.*, vol. 41, no. 6, pp. 1355–1361, Jun. 2013 DOI:10.1183/09031936.00215411.
  17. “American Academy of Sleep Medicine (2014) International classification of sleep disorders, 3rd edn. American Academy of Sleep Medicine, Darien.”
  18. Y. Pamula et al., “Australasian Sleep Association clinical practice guidelines for performing sleep studies in children,” *Sleep Medicine*, vol. 36. Elsevier B.V., pp. S23–S42, Aug. 01, 2017 DOI:10.1016/j.sleep.2017.03.020.
  19. R. N. Aurora et al., “Practice parameters for the respiratory indications for polysomnography in children,” *Sleep*, vol. 34, no. 3. Associated Professional Sleep Societies, LLC, pp. 379–388, Mar. 01, 2011 DOI:10.1093/sleep/34.3.379.



20. A. G. Kaditis et al., “Obstructive sleep disordered breathing in 2- to 18-year-old children: Diagnosis and management,” *Eur. Respir. J.*, vol. 47, no. 1, pp. 69–94, Jan. 2016 DOI:10.1183/13993003.00385-2015.
21. R. B. Mitchell et al., “Clinical Practice Guideline: Tonsillectomy in Children (Update),” *Otolaryngol. - Head Neck Surg. (United States)*, vol. 160, no. 1\_suppl, pp. S1–S42, Feb. 2019 DOI:10.1177/0194599818801757.
22. E. Sağtaş, E. Mengi, C. O. Kara, and H. Şenol, “A Novel Assessment Method With Ultrasound for Obstructive Tonsillar Hypertrophy in Children,” *J. Ultrasound Med.*, 2020 DOI:10.1002/jum.15559.
23. E. Kay-Rivest, C. Saint-Martin, and S. J. Daniel, “High-Frequency Ultrasound: A Novel Diagnostic Tool to Measure Pediatric Tonsils in 3 Dimensions,” *Otolaryngol. - Head Neck Surg. (United States)*, vol. 161, no. 5, pp. 856–861, Nov. 2019 DOI:10.1177/0194599819850139.
24. C. M. Quinlan, H. Otero, and I. E. Tapia, “Upper airway visualization in pediatric obstructive sleep apnea,” *Paediatric Respiratory Reviews*, vol. 32. W.B. Saunders Ltd, pp. 48–54, Nov. 01, 2019 DOI:10.1016/j.prrv.2019.03.007.
25. M. A. Slaats et al., “Upper airway imaging in pediatric obstructive sleep apnea syndrome,” *Sleep Medicine Reviews*, vol. 21. W.B. Saunders Ltd, pp. 59–71, Jun. 01, 2015 DOI:10.1016/j.smrv.2014.08.001.
26. L. F. Donnelly, “Obstructive sleep apnea in pediatric patients: Evaluation with cine MR sleep studies,” *Radiology*, vol. 236, no. 3, pp. 768–778, Sep. 2005 DOI:10.1148/radiol.2363040306.
27. S. E. Gibson, J. L. Strife, C. M. Myer, and D. M. O’Connor, “Sleep fluoroscopy for localization of upper airway obstruction in children,” *Ann. Otol. Rhinol. Laryngol.*, vol. 105, no. 9, pp. 678–683, 1996 DOI:10.1177/000348949610500902.
28. R. J. Fleck, S. R. Shott, M. Mahmoud, S. L. Ishman, R. S. Amin, and L. F. Donnelly, “Magnetic resonance imaging of obstructive sleep apnea in children,” *Pediatric Radiology*, vol. 48, no. 9. Springer Verlag, pp. 1223–1233, Aug. 01, 2018 DOI:10.1007/s00247-018-4180-2.
29. M. A. Socarras, B. P. Landau, and M. L. Durr, “Diagnostic techniques and surgical outcomes for persistent pediatric obstructive sleep apnea after adenotonsillectomy: A systematic review and meta-analysis,” *Int. J. Pediatr. Otorhinolaryngol.*, vol. 121, pp. 179–187, Jun. 2019 DOI:10.1016/j.ijporl.2019.02.030.



30. Z. Ehsan, M. Mahmoud, S. R. Shott, R. S. Amin, and S. L. Ishman, “The effects of Anesthesia and opioids on the upper airway: A systematic review,” in *Laryngoscope*, Jan. 2016, vol. 126, no. 1, pp. 270–284 DOI:10.1002/lary.25399.
31. A. Kandil et al., “Comparison of the combination of dexmedetomidine and ketamine to propofol or propofol/sevoflurane for drug-induced sleep endoscopy in children,” *Paediatr. Anaesth.*, vol. 26, no. 7, pp. 742–751, Jul. 2016 DOI:10.1111/pan.12931.
32. M. Cavaliere, F. Russo, and M. Iemma, “Awake versus drug-induced sleep endoscopy: Evaluation of airway obstruction in obstructive sleep apnea/hypopnoea syndrome,” *Laryngoscope*, vol. 123, no. 9, pp. 2315–2318, Sep. 2013 DOI:10.1002/lary.23881.
33. C. T. Wootten, S. Chinnadurai, and S. L. Goudy, “Beyond adenotonsillectomy: Outcomes of sleep endoscopy-directed treatments in pediatric obstructive sleep apnea,” *Int. J. Pediatr. Otorhinolaryngol.*, vol. 78, no. 7, pp. 1158–1162, 2014 DOI:10.1016/j.ijporl.2014.04.041.
34. S. He et al., “Outcomes of Drug-Induced Sleep Endoscopy–Directed Surgery for Pediatric Obstructive Sleep Apnea,” *Otolaryngol. - Head Neck Surg. (United States)*, vol. 158, no. 3, pp. 559–565, Mar. 2018 DOI:10.1177/0194599817740332.
35. D. F. Smith et al., “Effectiveness of pediatric drug-induced sleep endoscopy for REM-predominant obstructive sleep apnea,” *Sleep Breath.*, pp. 1–9, Apr. 2020 DOI:10.1007/s11325-020-02056-7.
36. E. Esteller et al., “Outcome of drug-induced sleep endoscopy-directed surgery for persistent obstructive sleep apnea after adenotonsillar surgery,” *Int. J. Pediatr. Otorhinolaryngol.*, vol. 120, pp. 118–122, May 2019 DOI:10.1016/j.ijporl.2019.02.004.
37. A. Francis, K. Eltaki, T. Bash, S. Cortes, K. Mojdehi, and N. A. Goldstein, “The safety of preoperative sedation in children with sleep-disordered breathing,” *Int. J. Pediatr. Otorhinolaryngol.*, vol. 70, no. 9, pp. 1517–1521, Sep. 2006 DOI:10.1016/j.ijporl.2006.02.001.
38. C. L. Marcus et al., “A randomized trial of adenotonsillectomy for childhood sleep apnea,” *N. Engl. J. Med.*, vol. 368, no. 25, pp. 2366–2376, 2013 DOI:10.1056/NEJMoa1215881.
39. M. Friedman, M. N. Wilson, J. Friedman, N. J. Joseph, H. C. Lin, and H. W. Chang, “Intracapsular coblation tonsillectomy and adenoidectomy for the treatment of pediatric obstructive sleep apnea/hypopnea syndrome,” *Otolaryngol. - Head Neck Surg.*, vol. 140, no. 3, pp. 358–362, Mar. 2009 DOI:10.1016/j.otohns.2008.11.031.



40. M. L. Alonso-Álvarez et al., “Treatment outcomes of obstructive sleep apnoea in obese community-dwelling children: The NANOS study,” *Eur. Respir. J.*, vol. 46, no. 3, pp. 717–727, Sep. 2015 DOI:10.1183/09031936.00013815.
41. K. Nandalike et al., “Adenotonsillectomy in obese children with obstructive sleep apnea syndrome: Magnetic resonance imaging findings and considerations,” *Sleep*, vol. 36, no. 6, pp. 841–847, Jun. 2013 DOI:10.5665/sleep.2708.
42. C. Carenfelt and P. O. Haraldsson, “Frequency of complications after uvulopalatopharyngoplasty,” *The Lancet*, vol. 341, no. 8842. Elsevier, p. 437, Feb. 13, 1993 DOI:10.1016/0140-6736(93)93030-5.
43. P. Scheffler et al., “Surgery for Obstructive Sleep Apnea in Obese Children: Literature Review and Meta-analysis,” *Otolaryngology - Head and Neck Surgery (United States)*, vol. 160, no. 6. SAGE Publications Inc., pp. 985–992, Jun. 01, 2019 DOI:10.1177/0194599819829415.
44. J. E. Kerschner, J. B. Lynch, H. Kleiner, V. A. Flanary, and T. B. Rice, “Uvulopalatopharyngoplasty with tonsillectomy and adenoidectomy as a treatment for obstructive sleep apnea in neurologically impaired children,” *Int. J. Pediatr. Otorhinolaryngol.*, vol. 62, no. 3, pp. 229–235, Feb. 2002 DOI:10.1016/S0165-5876(01)00623-1.
45. T. H. Inge et al., “Bariatric surgery for severely overweight adolescents: Concerns and recommendations,” *Pediatrics*, vol. 114, no. 1. Pediatrics, pp. 217–223, Jul. 2004 DOI:10.1542/peds.114.1.217.
46. M. Kalra et al., “Obstructive sleep apnea in extremely overweight adolescents undergoing bariatric surgery,” *Obes. Res.*, vol. 13, no. 7, pp. 1175–1179, 2005 DOI:10.1038/oby.2005.139.
47. R. Amin, N. Simakajornboon, R. Szczesniak, and T. Inge, “Early improvement in obstructive sleep apnea and increase in orexin levels after bariatric surgery in adolescents and young adults,” *Surg. Obes. Relat. Dis.*, vol. 13, no. 1, pp. 95–100, Jan. 2017 DOI:10.1016/j.soard.2016.05.023.
48. C. L. Marcus et al., “Adherence to and effectiveness of positive airway pressure therapy in children with obstructive sleep apnea,” *Pediatrics*, vol. 117, no. 3, Mar. 2006 DOI:10.1542/peds.2005-1634.
49. A. Ramirez et al., “Continuous positive airway pressure and noninvasive ventilation adherence in children,” *Sleep Med.*, vol. 14, no. 12, pp. 1290–1294, Dec. 2013 DOI:10.1016/j.sleep.2013.06.020.



50. C. L. Marcus et al., "Use of nasal continuous positive airway pressure as treatment of childhood obstructive sleep apnea," *J. Pediatr.*, vol. 127, no. 1, pp. 88–94, 1995 DOI:10.1016/S0022-3476(95)70262-8.
51. D. W. Beebe and K. C. Byars, "Adolescents with Obstructive Sleep Apnea Adhere Poorly to Positive Airway Pressure (PAP), but PAP Users Show Improved Attention and School Performance," *PLoS One*, vol. 6, no. 3, p. e16924, Mar. 2011 DOI:10.1371/journal.pone.0016924.
52. S. T. Kuna et al., "Long-term effect of weight loss on obstructive sleep apnea severity in obese patients with type 2 diabetes," *Sleep*, vol. 36, no. 5, pp. 641–649, May 2013 DOI:10.5665/sleep.2618.
53. S. L. Verhulst, H. Franckx, L. Van Gaal, W. De Backer, and K. Desager, "The effect of weight loss on sleep-disordered breathing in obese teenagers," *Obesity*, vol. 17, no. 6, pp. 1178–1183, Jun. 2009 DOI:10.1038/oby.2008.673.
54. C. J. Lettieri, A. H. Eliasson, and D. L. Greenburg, "Persistence of obstructive sleep apnea after surgical weight loss," *J. Clin. Sleep Med.*, vol. 4, no. 4, pp. 333–338, Aug. 2008 DOI:10.5664/jcsm.27233.
55. L. Kheirandish-Gozal, R. Bhattacharjee, H. P. R. Bandla, and D. Gozal, "Antiinflammatory therapy outcomes for mild OSA in children," *Chest*, vol. 146, no. 1, pp. 88–95, 2014 DOI:10.1378/chest.13-2288.
56. M. Santagati, M. Scillato, N. Muscaridola, V. Metoldo, I. La Mantia, and S. Stefani, "Colonization, safety, and tolerability study of the *Streptococcus salivarius* 24SMBc nasal spray for its application in upper respiratory tract infections," *Eur. J. Clin. Microbiol. Infect. Dis.*, vol. 34, no. 10, pp. 2075–2080, Oct. 2015 DOI:10.1007/s10096-015-2454-2.
57. S. Manti et al., "Bacteriotherapy with *Streptococcus salivarius* 24SMB and *Streptococcus oralis* 89a nasal spray for treatment of upper respiratory tract infections in children: A pilot study on short-term efficacy," *Ital. J. Pediatr.*, vol. 46, no. 1, Apr. 2020 DOI:10.1186/s13052-020-0798-4.
58. L. M. Bellussi et al., "Preventive nasal bacteriotherapy for the treatment of upper respiratory tract infections and sleep disordered breathing in children," *Int. J. Pediatr. Otorhinolaryngol.*, vol. 110, pp. 43–47, Jul. 2018 DOI:10.1016/j.ijporl.2018.04.024.
59. C. A. Kushida et al., "Practice parameters for the treatment of snoring and obstructive sleep apnea with oral appliances: An update for 2005," *Sleep*, vol. 29, no. 2, pp. 240–243, Feb. 2006



DOI:10.1093/sleep/29.2.240.

60. M. P. Villa, E. Bernkopf, J. Pagani, V. Broia, M. Montesano, and R. Ronchetti, “Randomized controlled study of an oral jaw-positioning appliance for the treatment of obstructive sleep apnea in children with malocclusion,” *Am. J. Respir. Crit. Care Med.*, vol. 165, no. 1, pp. 123–127, Jan. 2002 DOI:10.1164/ajrccm.165.1.2011031.
61. J. P. Moss, “Orofacial Myology – International Perspectives Second Edition,” *Br. Dent. J.*, vol. 195, no. 6, pp. 355–355, Sep. 2003 DOI:10.1038/sj.bdj.4810539.
62. Y. S. Huang, S. C. Hsu, C. Guilleminault, and L. C. Chuang, “Myofunctional Therapy: Role in Pediatric OSA,” *Sleep Medicine Clinics*, vol. 14, no. 1. W.B. Saunders, pp. 135–142, Mar. 01, 2019 DOI:10.1016/j.jsmc.2018.10.004.
63. L. M. Walter, D. U. N. Dassanayake, A. J. Weichard, M. J. Davey, G. M. Nixon, and R. S. C. Horne, “Back to sleep or not: the effect of the supine position on pediatric OSA: Sleeping position in children with OSA,” *Sleep Med.*, vol. 37, pp. 151–159, Sep. 2017 DOI:10.1016/j.sleep.2017.06.014.
64. R. S. Strauss, “Childhood obesity and self-esteem.,” *Pediatrics*, vol. 105, no. 1, 2000 DOI:10.1542/peds.105.1.e15.
65. R. Puhl and Y. Suh, “Health Consequences of Weight Stigma: Implications for Obesity Prevention and Treatment,” *Current obesity reports*, vol. 4, no. 2. *Curr Obes Rep*, pp. 182–190, Jun. 01, 2015 DOI:10.1007/s13679-015-0153-z.
66. S. M. Azevedo and L. R. Vartanian, “Ethical Issues for Public Health Approaches to Obesity,” *Current obesity reports*, vol. 4, no. 3. *Curr Obes Rep*, pp. 324–329, Sep. 01, 2015 DOI:10.1007/s13679-015-0166-7.

**Volume 2 Issue 4 April 2021**

**©All rights reserved by Haya Alsubie**