Laryngoscope
Investigative Otolaryngology

ORIGINAL RESEARCH OPEN ACCESS

Obstructive Sleep Apnea in Overweight and Obese Children: Factors Influencing Quality of Life

Montaha Al-Iede[1,2] | Rahaf Rahal[2] | Sondos Al-Mashaqbeh[2] | Mohannad Alajmi[2] | Hadeel Al-Rawi[2] | Mohammed Alshrouf[2] | Fareed Khdair Ahmad[2,3] | Abeer Alassaf[2,4] | Rasha Odeh[2,4] | Iyad Almofleh[5] | Nihad Almasri[6]

[1]Division of Pediatric Pulmonology and Sleep Medicine, Department of Pediatrics, Jordan University Hospital, Amman, Jordan | [2]The School of Medicine, The University of Jordan, Amman, Jordan | [3]Division of Pediatric Gastroenterology, Department of Pediatrics, Jordan University Hospital, Amman, Jordan | [4]Division of Pediatric Endocrinology, Department of Pediatrics, Jordan University Hospital, Amman, Jordan | [5]Department of Medicine, Jordan University Hospital, Amman, Jordan | [6]Department of Physiotherapy, School of Rehabilitation, The University of Jordan, Amman, Jordan

Correspondence: Montaha Al-Iede (montaha95@yahoo.com; m.alde@ju.edu.jo)

Received: 28 November 2023 | **Revised:** 9 January 2025 | **Accepted:** 19 March 2025

Funding: The authors received no specific funding for this work.

Keywords: biological sex | obesity | obstructive sleep apnea | polysomnography | quality of life | sleep-disordered breathing

ABSTRACT

Introduction: There is a paucity of data regarding biological sex influence and the impact of obstructive sleep apnea (OSA) on the quality of life (QoL) of obese children with OSA. Thus, we aimed to assess the influence of biological sex on polysomnography (PSG) and evaluate the impact of OSA on obese children's QoL.

Methods: Records of overweight or obese pediatric patients referred for sleep studies at the Jordan University Hospital between 2018 and 2022 were retrospectively reviewed. Children underwent PSG and anthropometric measurements. OSA diagnosis and severity were determined per the Apnea-Hypopnea Index (AHI). QoL was determined by the OSA-18 tool.

Results: Across a sample of 136 children, biological sex did not influence PSG indices, but there were significant differences across the sleep disorder ($p = 0.023$) and daily functioning ($p = 0.007$) QoL domains. Age affected the non-REM sleep percentages and NADIR of O2 saturation (all $p < 0.01$). There were significant differences across the emotional distress and daytime function domains across age groups (all $p < 0.05$). Body mass index (BMI) did not significantly influence AHI strata, but was associated with worse daytime function ($p < 0.05$). Additionally, OSA severity was associated with poorer sleep disorder and concerns about caregivers' scores (all $p < 0.05$). On multivariate analysis, gender predicted OSA-18 total score, but not age, BMI, or AHI.

Conclusion: It appears that biological sex has no clinical impact on OSA among obese children. However, it appears that age significantly influences both OSA and its associated QoL.

1 | Introduction

The American Thoracic Society defines obstructive sleep apnea (OSA) as a sleep-breathing disease characterized by persistent partial upper airway blockage and/or intermittent total obstruction that affects normal sleep ventilation and sleep patterns [1]. OSA has been reported to occur in 1%–6% of the pediatric age group [2]. It is related to several comorbidities that disrupt many organ systems, causing acute or long-term consequences and resulting in significant societal and financial expenses [3]. Children with severe OSA can have adverse cardiovascular [4], behavioral, and neurocognitive complications [5]. Advanced age, male sex, and high body mass index (BMI) contribute to an increase in the prevalence of OSA, which varies from 9%–38% in the general population [6].

The significant rise in the prevalence of pediatric obesity, not only in developed countries but also in developing countries, has

caused serious concerns [7]. In the literature, it is well reported that OSA associated with obesity is highly prevalent in children and adolescents, with a prevalence ranging between 36% and 60% [8–10]. Obesity-related OSA is expected to affect 25% of overweight children, and in a community-based cohort, obese children had a 4- to 5-fold higher incidence of OSA than lean controls [8]. In fact, in children and adolescents with OSA, the severity of OSA parallels the severity of obesity [11]. OSA and obesity have a complicated association that is most likely the result of a combination of biological factors, including biological sex, race, genetic, neurohormonal, and lifestyle factors [12].

Studies in adults have confirmed that OSA is more common in males than in females, with an estimated male-to-female ratio of 3:1 to 5:1 in the general population [13, 14]. Moreover, research has shown that severe OSA is associated with the male sex and a higher BMI than the female sex and a lower BMI [15, 16]. Fewer studies in children have examined the biological sex influence on OSA severity and its consequences [17]. Studies have reported a similar prevalence in children during puberty but a higher prevalence in boys than in girls after puberty [1, 18]. In addition, Inoshita et al. have reported more severe OSA among adolescent males compared to females [19].

The gold standard test available for diagnosing obstructive sleep apnea in children is polysomnography. However, polysomnography testing is costly and relatively inaccessible [20]. Pediatric OSA has been linked to a reduction in health-related quality of life (QoL) [21]. The OSA-18 QoL questionnaire, developed by Franco et al., is now the most extensively used QoL survey for pediatric OSA in Western countries [22]. A few studies have assessed the impact of biological sex on OSA severity and the impact of OSA on the lives of children with obesity [23], but none have looked at the Arab population in the Middle East [24]. This study aimed to identify the characteristics of obese children with sleep-disordered breathing (SDB), the influence of biological sex on the polysomnography (PSG) result, and the impact of OSA on obese children's QoL.

2 | Materials and Methods

2.1 | Study Design and Data Collection

This retrospective study was conducted at Jordan University Hospital between November 2018 and August 2023. The anthropometric variables, including weight and height, of 434 patients who were referred to the sleep laboratory for polysomnography within the included period were reviewed. Weight and height were recorded without shoes using a scale DC 7.5 V/IA (NAGATA digital with measuring height rod: Model BW-1222H, Taiwan) (error +/− 0.1 kg) present in the sleep laboratory on the same night of the sleep study. BMI and the BMI Z-score were calculated using an online calculator from Children's Hospital of Philadelphia [25]. This tool allows BMI calculation of patients between the ages of 2 and 20 years, as well as the exact BMI percentile and Z-score based on the Center for Disease Control (CDC) growth charts. Based on the BMI Z-score, patients were classified as underweight/normal weight with Z-scores between −2 and + 0.99, overweight with Z-scores between 1 and + 1.99, and obese from 2 to 2.99, and very obese ≥ 3 [26]. A total of 136 children (62 overweight and 74 obese) aged 3–16 years, were included. Children with neuromuscular diseases, chronic lung disease, tonsillar hypertrophy, and any inherited or craniofacial syndromes leading to OSA were excluded.

2.2 | Polysomnography

All patients underwent overnight PSG in the sleep unit at Jordan University Hospital using the Phillips Alex 6 system during a normal sleep period. The PSG included sleep staging, respiratory events, and ventilation. Sleep staging was monitored using four electroencephalogram (EEG) leads (central and occipital, two each): C3–A2, C4–A1, O1–A2, and O1–A1. Bilateral electrooculogram (EOG) channels (LOC–A1, ROC–A1) and submental electromyogram (EMG) channels were used. Respiratory channels included a nasal cannula, a pressure sensor to monitor airflow, and plethysmography to monitor respiratory movements of the chest and abdomen. Oxygen saturation (SaO2) and pulse were recorded using a pulse oximeter. Cardiac rhythm was monitored by ECG leads.

A registered PSG technician manually scored the studies, which were then reviewed and interpreted by a pediatric sleep physician. The American Academy of Sleep Medicine (AASM) criteria were applied to analyze the sleep stages of 1–3 for non-rapid eye movement (NREM) sleep and rapid eye movement (REM) sleep [27] Studies were scored according to the ASTA/ASA Addendum to the AASM Guidelines for recording and scoring pediatric sleep (April 2011) and the AASM manual for the scoring of sleep [27]. Respiratory events were scored as obstructive, central apnea, or hypopnea. Obstructive apnea was defined as ongoing respiratory efforts despite cessation of flow for at least two respiratory cycles. Central apnea was defined as cessation of both airflow and respiratory effort for at least 20 s, or less than 20 s if associated with desaturation of > 3% or arousal. The severity of SDB was assessed by the total apnea-hypopnea index (AHI) and abnormalities of nocturnal gas exchange. OSA severity was classified as mild (1 ≤ AH I < 5), moderate (5 ≥ AHI < 10), and severe (10 ≥ AHI) [28].

2.3 | Diagnostic Sleep Study Results

Key metrics examined in the diagnostic sleep study results included baseline and minimal oxygen saturation (SaO2), AHI, obstructive apnea-hypopnea index (OAHI), and respiratory events in REM sleep (REM index), the arousal index (the number of arousals per hour of sleep), and sleep efficiency. The AHI reflects the number of apnea or hypopnea events recorded per hour of sleep, while the OAHI refers to the number of obstructive events per hour of sleep.

2.4 | The OSA-18 Quality of Life Questionnaire

The OSA-18 survey is a valid and reliable QoL measurement tool, which is sufficient to discriminate between children with SDB. It focuses on physical, functional, and emotional problems resulting from SDB. The questionnaire consists of 18 items grouped into five domains: sleep disturbance, physical

symptoms, emotional distress, daytime function, and caregiver concerns. Items were scored on an ordinal 7-point scale (1- none of the time, 2- hardly any of the time, 3- a little of the time, 4- some of the time, 5- a good bit of the time, 6- most of the time, 7- all of the time). OSA-18 domains yield the following scores: [A] sleep disturbances (4 items with scores between 4 and 28), [B] physical suffering (4 items with scores between 4 and 28), [C] emotional distress (3 items with scores between 3 and 21), [D] daytime problems (3 items with scores between 3 and 21), [E] parent or caretaker concern (4 items with scores between 4 and 28). The total OSA-18 score is between 18 (no impact on QoL) and 126 (significant impact on QoL).

A total score was calculated by adding the points from each domain to define the impact of OSA on health-related QoL. The impact on health-related QoL was categorized according to Franco et al.'s prior validation into three groups: minor (< 60), moderate (60–80), and major (> 80) [29]. Partial scores were also calculated for each domain by summing the items within each domain.

The questionnaire was expertly translated into Arabic in accordance with World Health Organization (WHO) guidelines. This translation process took place within an accredited translation office in Amman and was carried out by a certified translator along with a bilingual healthcare professional with relevant clinical and research experience. To ensure accuracy, a rigorous back translation was then conducted by English-speaking healthcare staff, verifying the precise alignment of terms with those found in the original document. In addition, the questionnaire's face and content validity were reviewed by a panel of specialist pediatricians from Jordan University Hospital for proof that the questions and translation were coherent.

Parents of recruited children were approached by members of the research team prior to or during the sleep study. Should the parents agree to the premise of the study, they were instructed to complete a printed, paperback version of the translated OSA-18. Research members would ensure full completion of the questionnaire and consult parents on any missing responses.

2.5 | Ethics Committee Approval

This study was conducted in accordance with the Declaration of Helsinki. The study was approved by the Institutional Review Board of the Jordan University Hospital (approval number: 2021/371). Written informed consent was obtained from all children's parents after explaining the aim of the study and the nature of their children's participation on the night of the polysomnography.

2.6 | Statistical Analysis

All data was analyzed on SPSS version 23. Continuous variables were presented as means ± standard deviations while categorical variables were presented as frequencies [*n*(%)]. Differences among continuous variables between categorical variables composed of two groups (e.g., gender) were examined using *t*-test. Differences among categorical variables composed of more than two groups (e.g., Age groups) were examined using ANOVA. Mean differences were visualized using the 'dabester' package on R using Gardner-Altman plots. A multivariate regression model was utilized to explore predictors of OSA-18 scores and sub-scores. A *p*-value of less than 0.05 was considered statistically significant.

3 | Results

In total, 136 children with overweight and obesity were included in the analysis. The majority of participants were males 92 (67.6%) and between 6 and 12 years of age 68 (50.0%). Asthma was the most common comorbidity among participants 44 (62.9%), followed by hypothyroidism 9 (12.9%). Across the included sample, the AHI index was dominantly severe 58 (42.6%). Median AHI was 8.0 (IQR, 5–19) ranging from 1 to 77. Approximately 77.9% of participants were obese while 22.1% were overweight. Median BMI for the included sample was 24.5 (IQR, 21.3–29.5) ranging from 17 to 51.4. Among those with obesity, AHI was severe in 45.3%. On the other hand, mild AHI was the most prevalent among overweight patients (43.3%). Age, weight, prevalence of comorbidities, and AHI index were not statistically different among males and females (Refer to Table 1).

QoL scores were computed for all 5 sub-domains. The median scores for sleep disturbance, physical symptoms, emotional distress, daytime function, and caregiver concerns were 10 (IQR, 6.8–15.3), 12 (IQR, 8.0–17.0), 7 (4.0–13.0), 7 (4.0–11.0), and 11 (IQR, 7.0–16.0). The total median OSA score was 50 (IQR, 39.8–67.0).

On univariate analysis, there were no meaningful differences among genders in terms of any polysomnography indices (Refer to Table 2). However, when stratified by age, participants demonstrated a number of meaningful differences in their polysomnography readings. Non-REM sleep percentage stage N1 and N3 ($p = 0.01$ and $p < 0.001$, respectively) and NADIR of O2 saturation were meaningfully different across age groups ($p = 0.01$) (Refer to Table S1).

There was a significant difference between males and females in terms of total OSA-18 score ($p = 0.036$), particularly the sleep disorder and daily functioning sub-domains ($p = 0.023$ and 0.007, respectively). In terms of age, emotional distress and daytime function were significantly different across age groups ($p = 0.02$ and $p < 0.001$, respectively). Finally, when stratified by AHI severity, participants demonstrated significant differences in the sleep disorder and concerns of caregivers domains ($p = 0.015$ and 0.032, respectively). Table 3 and Figure 1 demonstrate mean differences in total OSA-18 score and sub-scores among participants.

On multivariate analysis, female gender was an independent predictor of total OSA-18 score (B: 11.73 (1.36–22.09)), sleep disorder score (B: 3.94 (0.95–6.93)), and daily functioning score (B: 2.52 (0.45–4.59)). Similarly, age at diagnosis was a positive independent predictor of daily functioning score (B: 0.44 (0.13–0.74)). Interestingly, AHI was not a significant predictor of the OSA-18 score or any of its sub-scores. It is noteworthy to showcase that awake arterial oxygen saturation was a positive independent

TABLE 1 | Characteristics of included participants.

Variables	Categories	Total *N* (%)	Males *N* (%)	Females *N* (%)	*p*
Age groups	< 6	30 (22.1)	20 (21.7)	10 (22.7)	0.73
	6–12	68 (50.0)	48 (52.2)	20 (45.5)	
	> 12	38 (27.9)	24 (26.1)	14 (31.8)	
BMI	Z-score	2.17 ± 0.76	2.21 ± 0.80	2.09 ± 0.67	0.42
Comorbidities	Asthma	2 (2.9)	1 (2.0)	1 (5.0)	0.10
	Hypothyroidism	8 (11.4)	6 (12.0)	2 (10.0)	
	Diabetes mellitus	7 (10.0)	3 (6.0)	4 (20.0)	
	Brain tumors	44 (32.4)	31 (33.7)	13 (29.5)	
	Hypertension	34 (25.0)	23 (25.0)	11 (25.0)	
Apnea-hypoapnea index (AHI)	Mild	58 (42.6)	38 (41.3)	20 (45.5)	0.90
	Moderate	30 (22.1)	20 (21.7)	10 (22.7)	
	Severe	68 (50.0)	48 (52.2)	20 (45.5)	

TABLE 2 | Characteristics of polysomnography among included participants.

Variables	Total (*n* = 136) mean ± SD	Males (*n* = 92) mean ± SD	Females (*n* = 44) mean ± SD	*p*
Non-REM sleep (% TST)				
Stage N1	9.83 ± 7.09	10.09 ± 5.88	9.29 ± 9.16	0.543
Stage N2	48.31 ± 9.57	47.32 ± 10.08	50.39 ± 8.10	0.080
Stage N3	33.51 ± 10.18	34.25 ± 10.28	31.98 ± 9.90	0.224
REM sleep (% TST)	8.81 ± 8.01	8.10 ± 7.45	10.30 ± 8.96	0.133
Arousal index (events/h)	19.90 ± 12.03	19.70 ± 12.10	20.34 ± 12.01	0.771
AHI (events/h)	14.27 ± 15.89	13.80 ± 16.01	15.23 ± 15.76	0.626
Respiratory event				
Central apnea	14.69 ± 29.68	11.39 ± 14.80	21.43 ± 46.92	0.066
Obstructive apnea	6.92 ± 16.85	5.39 ± 13.40	10.12 ± 22.28	0.131
Mixed apnea	2.27 ± 7.60	2.16 ± 7.99	2.50 ± 6.78	0.812
Hypoapnea	54.13 ± 67.22	51.14 ± 66.84	60.25 ± 68.36	0.464
Gas exchange				
Awake arterial O2 saturation (%)	94.97 ± 2.56	95.01 ± 2.54	94.89 ± 2.63	0.792
Average arterial O2 desaturation at sleep (%)	93.64 ± 2.76	93.70 ± 2.62	93.52 ± 3.08	0.734
NADIR O2 saturation (%)	83.15 ± 8.24	83.52 ± 7.70	82.39 ± 9.31	0.454
Snoring time (minutes)	54.39 ± 68.68	48.46 ± 67.30	66.65 ± 70.66	0.150
Percentage of sleep (%)	16.15 ± 19.93	14.14 ± 19.10	20.30 ± 21.17	0.092

predictor of physical distress score (B: 1.96 (0.64–3.28)). On the other hand, average arterial oxygen desaturation at sleep was a negative predictor of physical distress score (B: −1.73 (−3.09 to −0.37)). Table 4 demonstrates the multivariate linear regression models for the OSA-18 score and sub-scores.

4 | Discussion

To the best of our knowledge, this study presents itself as the first work from the Middle East to explore the clinical characteristics of overweight children with SDB. Moreover, this

TABLE 3 | Differences in QoL stratified per gender, age, and AHI strata.

		Sleep disorder	Physical distress	Emotional distress	Daily functioning	Concerns of caregivers
Gender	Male	10.43 ± 5.36*	12.47 ± 6.14	8.12 ± 4.79	7.25 ± 3.99*	11.31 ± 5.73
	Female	13.67 ± 6.59*	13.17 ± 6.46	9.58 ± 5.02	10.17 ± 5.24*	13.67 ± 7.68
Age	< 6	11.24 ± 6.81	11.43 ± 5.59	6.48 ± 3.61*	5.14 ± 2.67*	11.43 ± 6.17
	6–12	11.71 ± 5.58	14.29 ± 6.25	10.11 ± 5.10*	8.86 ± 4.20*	12.43 ± 5.96
	> 12	11.04 ± 5.73	11.56 ± 6.33	8.11 ± 4.88*	9.41 ± 5.21*	11.85 ± 7.27
AHI	Mild	8.93 ± 3.50*	11.39 ± 4.96	7.79 ± 4.29	7.57 ± 3.89	10.86 ± 4.87*
	Moderate	11.70 ± 6.30*	11.90 ± 7.11	10.35 ± 5.55	7.85 ± 4.92	9.90 ± 5.62*
	Severe	13.21 ± 6.62*	14.14 ± 6.40	8.11 ± 4.78	8.66 ± 4.88	14.09 ± 7.35*

*Indicates significant mean differences between subgroups at *p*.

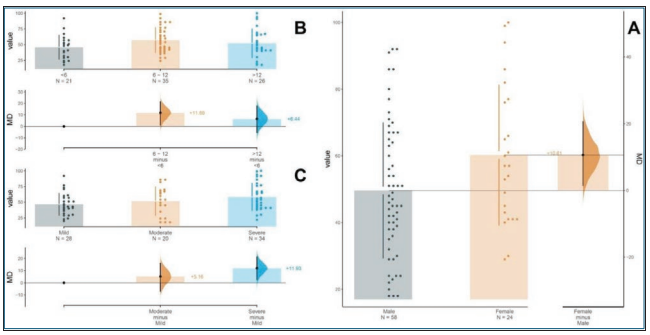

FIGURE 1 | Comparison of total OSA-18 scores across gender, age groups, and AHI. Severity levels. (A) Differences in total OSA-18 score among genders (male vs. female; mean difference (MD): 10.6; *p*-value: 0.0414). (B) Differences in total OSA-18 score among age groups (< 6 vs. 6–12; MD: 11.7; *p*-value: 0.034), (< 6 vs. > 12; MD: 6.4; *p*-value: 0.296). (C) Differences in total OSA-18 score among AHI strata (mild vs. moderate; MD: 5.1; *p*-value: 0.401), (mild vs. severe; MD: 11.9; *p*-value: 0.018).

work examines the influence of age and biological sex on their clinical characteristics and QoL metrics. The first striking finding in our report is the percentage of pediatric patients with moderate and/or severe OSA. We show that 72% of total participants had moderate and/or severe OSA. Compared with similar literature, moderate-to-severe OSA comprised only a minority of the sample, particularly severe OSA. Such differences are mainly attributed to differences in methodology—sampling frame to be exact. While Horne et al. studies patients referred for sleep studies in comparison with a community-based sample, Bixler et al., sampled the general population in a similar 'case vs. control' design [23, 30]. In contrast, we included only patients undergoing sleep studies at our institution.

The current body of literature shows significant variability in observations between adult and pediatric cohorts. Heinzer et al. demonstrated that the prevalence of SDB, expressed in AHI, is 2–3 times higher in adult men compared to older and younger adult women, respectively [31]. On the other hand, Bixler et al. showed that biological sex does not influence the prevalence of SDB among pediatrics [30]. Similarly, Horne et al. found no association between biological sex and the severity and consequences of SDB [23]. Our results concur with the latter, as there were no differences in AHI or polysomnography per biological sex of included participants. Such findings are attributed to the multifactorial and multifaceted nature of SDB development.

Among children, nutritional disorders—mainly obesity—are implicated to be the most relevant risk of SDB during early development [32]. Within the context of our study, all included children had high BMI levels that were not significantly different between males and females. Interestingly, our results show that BMI did not significantly differ across different AHI strata (i.e., different SDB severities) but did significantly correlate with poor daytime function. This observation has a number of possible implications. Firstly, the impact of BMI on SDB may have a certain threshold, after which SDB becomes influenced by other physiologic or pathologic fluctuations. Secondly, BMI could be a mediator of behavioral disorders, which are independently worsened by or worsen SDB. Nonetheless, it should be noted that a biological sex difference in SDB characteristics could be present for patients with genetic syndromes (e.g., Down syndrome) or congenital malformations (e.g., Chiari malformation) as those patients exhibit extreme yet wide prevalence rates of SDB [33].

Almost 32% of our cohort were brain tumor survivors, primarily with craniopharyngiomas and medulloblastomas. These patients had been treated for their primary brain tumors and were referred for sleep studies due to symptoms suggestive of OSA, likely associated with their obesity. The high proportion of brain tumor survivors in our cohort reflects the specialized patient population at our institution, which includes referrals for children with complex medical conditions. Craniopharyngiomas, which are benign tumors arising near the hypothalamic–pituitary axis, are known to cause hypothalamic dysfunction that can lead to severe obesity and increase the risk of OSA [34]. Similarly, medulloblastomas, though malignant, are often treated successfully, but survivors may experience long-term complications such as obesity and respiratory control issues due to tumor location, treatment effects, or both.

It is worth mentioning that there do exist studies which did demonstrate biological sex-based differences in SBD characteristics, particularly the rate of AHI [19, 35]. However, the conclusions of the referenced studies are limited by sample size.

TABLE 4 | Predictors of QoL score and sub-scores on multivariate linear regression.

	Total OSA score	Sleep disorder	Physical distress	Emotional distress	Daily functioning	Concerns of caregivers
	B (95% CI)	B (95% CI)	B (95% CI)	B (95% CI)	B (95% CI)	B (95% CI)
Gender	11.73 (1.36–22.09)	3.94 (0.95–6.93)	0.97 (−2.15–4.09)	2.26 (−0.22–4.75)	2.52 (0.45–4.59)	1.99 (−1.33–5.30)
Age at diagnosis	0.54 (−0.99–2.07)	−0.21 (−0.65–0.23)	0.08 (−0.38–0.54)	0.23 (−0.14–0.60)	0.44 (0.13–0.74)	0.01 (−0.48–0.49)
BMI	−0.03 (−0.88–0.82)	0.00 (−0.25–0.24)	−0.15 (−0.40–0.10)	−0.05 (−0.24–0.15)	0.10 (−0.06–0.27)	0.09 (−0.17–0.35)
Sleep efficacy	−0.19 (−0.86–0.48)	−0.09 (−0.28–0.11)	−0.02 (−0.22–0.18)	0.07 (−0.10–0.23)	−0.05 (−0.18–0.09)	−0.10 (−0.31–0.11)
Total sleep time	−0.04 (−0.16–0.09)	−0.01 (−0.04–0.03)	−0.01 (−0.05–0.03)	−0.02 (−0.05–0.01)	0.00 (−0.02–0.03)	0.00 (−0.04–0.04)
Sleep onset	−0.03 (−0.14–0.09)	0.00 (−0.04–0.03)	−0.01 (−0.05–0.02)	0.01 (−0.02–0.04)	−0.01 (−0.03–0.01)	−0.01 (−0.05–0.02)
N1	0.10 (−1.09–1.29)	−0.22 (−0.56–0.13)	0.11 (−0.21–0.43)	−0.05 (−0.30–0.21)	0.13 (−0.09–0.34)	0.07 (−0.27–0.41)
N2	−0.07 (−1.13–0.99)	−0.19 (−0.49–0.12)	0.03 (−0.24–0.30)	−0.11 (−0.32–0.11)	0.08 (−0.10–0.26)	0.05 (−0.24–0.33)
N3	0.36 (−0.65–1.37)	−0.18 (−0.47–0.11)	0.12 (−0.15–0.40)	−0.02 (−0.24–0.20)	0.19 (0.01–0.37)	0.20 (−0.09–0.49)
REM	0.50 (−0.45–1.45)	0.00 (−0.27–0.28)	0.16 (−0.09–0.41)	0.03 (−0.17–0.23)	0.13 (−0.04–0.30)	0.13 (−0.14–0.40)
Arousal index	0.11 (−0.47–0.69)	0.03 (−0.14–0.19)	0.11 (−0.06–0.28)	−0.01 (−0.15–0.13)	−0.01 (−0.13–0.10)	−0.02 (−0.20–0.17)
AHI	−0.03 (−0.51–0.44)	0.01 (−0.13–0.15)	−0.04 (−0.19–0.10)	−0.05 (−0.16–0.07)	0.01 (−0.08–0.11)	0.04 (−0.11–0.19)
Awake arterial oxygen saturation	3.92 (−0.47–8.32)	0.14 (−1.13–1.41)	1.96 (0.64–3.28)	0.82 (−0.23–1.87)	0.26 (−0.62–1.13)	0.72 (−0.68–2.12)
Average arterial oxygen desaturation at sleep	−2.31 (−6.84–2.21)	−0.06 (−1.37–1.24)	−1.73 (−3.09 to −0.37)	−0.27 (−1.36–0.81)	0.10 (−0.80–1.00)	−0.30 (−1.74–1.14)
NADIR of O2 saturation	−0.77 (−1.61–0.08)	−0.21 (−0.46–0.03)	−0.09 (−0.35–0.16)	−0.19 (−0.40–0.01)	−0.14 (−0.31–0.03)	−0.13 (−0.40–0.14)
Snoring time	−0.02 (−0.09–0.06)	0.00 (−0.02–0.02)	−0.01 (−0.04–0.01)	0.00 (−0.02–0.01)	−0.01 (−0.02–0.01)	0.01 (−0.01–0.04)

We have also demonstrated that age is a significant influencing factor on the clinical characteristics of pediatric patients with SDB. Such association is expected as SDBs, particularly OSA, have a bimodal prevalence peak [36, 37]. The first peak lies within the 2–8 age group parallel to the presence of adenotonsillar hypertrophy, while the 2nd arises during adolescence in parallel with weight gain. From a physiological point of view, younger children, despite their high respiratory rate, have lower functional residual capacity and utilize muscle-based abdominal breathing [38]. Within the context of OSA, such breathing mechanics should mainly affect REM sleep [39]. Nonetheless, age differences within our cohort were confined to NADIR O2 saturation and non-REM breathing, which is more related to adult sleep-associated diseases or the post-pubertal forms of OSA [40].

On another note, we demonstrated that biological sex, age, and severity of SDB were all shown to have an influence on QoL, or at least one of its facets as measured by the OSA-18 questionnaire. There is a considerable body of evidence showcasing that SDB has adverse effects on both QoL and behavior irrespective of age [41–44]. In fact, Friedman et al. documented neurocognitive and behavioral deficits among children with SDB, who got significantly better after treatment [45]. However, the latter treatment effect is contested by Bixler et al., who believe that such conclusions are merely placebo effects or regression to the mean [30].

Across our cohort, older children and females had worse QoL per OSA-18; findings which are echoed across the literature [23]. The association between increased age and poorer QoL is mainly attributed to the unique social contexts imposed upon affected children, particularly adolescents. Such a particular affected population might be heavily impacted by non-specific symptoms such as headaches. Moreover, other symptoms such as snoring might be perceived as socially unacceptable from the child's point of view, considering the sensitive social dynamics of adolescent children [46]. We could add to that argument that older children afflicted with snoring, anxiety, or depression may suffer from a greater risk of being bullied; thus, their day-to-day QoL could be compromised [47, 48].

Nonetheless, Horne et al. attribute the effect of OSA on QoLto intra-sex dynamics [23]. Females report a higher likelihood of non-specific symptoms (e.g., headache, fatigue, and anxiety) and insomnia compared to their male counterparts [49]. In addition to the impact of sensitive social considerations among the two biological sexes, a number of physiological differences may account for pathological manifestations of SDB and thus QoL. Those differences include men having higher upper airway collapsibility, increased pharyngeal resistance to inspiratory loading, and more sensitivity of chemo-reflex to hypoxemia [46, 50]. Moreover, it appears that hormonal factors also play a role as reduced airway resistance in women is positively correlated with progesterone levels [50]. This may also partially explain age differences across females. It should be noted that the association itself between biological sex and OSA is under scrutiny. While Dekany et al. failed to observe the association among their cohort of 921 children [51], Wang et al. observed male sex as an independent risk factor for OSA after adjusting for a variety of clinical variables, including age [52]. Therefore, larger epidemiological studies are needed.

Similarly, the association between different SDB severities and QoL is well documented within the literature [29, 53]. Our findings showed that the impact is greatest among the concerns of caregivers and the sleep function across affected patients.

We acknowledge that our conclusions are bound to some limitations. First, the sample size is relatively small. Second, the number of boys was not equal to the number of girls, which could lead to bias. Third, all the data was collected from a single tertiary center, which may have led to the inclusion of more severe cases. However, the study has several strengths, including it being the first study in the region to investigate the impact of biological sex on OSA severity. Future research should be done with a larger sample size and a normal control group to minimize patient bias and increase the generalizability of the results.

5 | Conclusion

Obesity is a key risk factor in the development and progression of OSA among children. However, it appears that the impact of obesity after a certain threshold of BMI is behavioral rather than clinical. Moreover, while biological sex did not show any kind of clinical influence on our target population, clinicians should meticulously assess OSA per age strata as they are associated with both physiological and neurocognitive changes that may heavily impact the course of OSA among children and their caregivers. Future studies should assess whether OSA-related interventions have different outcomes among different sexes.

Acknowledgments

We would also like to thank all the participants for their contributions to this study and technical support of the sleep unit staff.

Ethics Statement

The study was conducted according to the guidelines of the Declaration of Helsinki and approved by the Institutional Review Board of Jordan University Hospital (reference number 2021/371).

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

The data from the present research are accessible from the corresponding author upon reasonable request.

References

1. J. C. Lumeng and R. D. Chervin, "Epidemiology of Pediatric Obstructive Sleep Apnea," *Proceedings of the American Thoracic Society* 5, no. 2 (2008): 242–252, https://doi.org/10.1513/pats.200708-135MG.

2. C. L. Marcus, L. J. Brooks, K. A. Draper, et al., "Diagnosis and Management of Childhood Obstructive Sleep Apnea Syndrome," *Pediatrics* 130, no. 3 (2012): e714–e755, https://doi.org/10.1542/peds.2012-1672.

3. H. H. Chuang, C. G. Huang, L. P. Chuang, et al., "Relationships Among and Predictive Values of Obesity, Inflammation Markers, and Disease Severity in Pediatric Patients With Obstructive Sleep Apnea Before and After Adenotonsillectomy," *Journal of Clinical Medicine* 9, no. 2 (2020): 579, https://doi.org/10.3390/jcm9020579.

4. L. C. Nisbet, S. R. Yiallourou, L. M. Walter, and R. S. C. Horne, "Blood Pressure Regulation, Autonomic Control and Sleep Disordered Breathing in Children," *Sleep Medicine Reviews* 18, no. 2 (2014): 179–189, https://doi.org/10.1016/j.smrv.2013.04.006.

5. S. N. Biggs, G. M. Nixon, and R. S. C. Horne, "The Conundrum of Primary Snoring in Children: What Are We Missing in Regards to Cognitive and Behavioural Morbidity?," *Sleep Medicine Reviews* 18, no. 6 (2014): 463–475, https://doi.org/10.1016/j.smrv.2014.06.009.

6. J. Mochol, J. Gawrys, D. Gajecki, E. Szahidewicz-Krupska, H. Martynowicz, and A. Doroszko, "Cardiovascular Disorders Triggered by Obstructive Sleep Apnea—A Focus on Endothelium and Blood Components," *International Journal of Molecular Sciences* 22, no. 10 (2021): 5139, https://doi.org/10.3390/ijms22105139.

7. L. Wang, L. Kong, F. Wu, Y. Bai, and R. Burton, "Preventing Chronic Diseases in China," *Lancet* 366, no. 9499 (2005): 1821–1824, https://doi.org/10.1016/S0140-6736(05)67344-8.

8. S. Bin-Hasan, S. Katz, Z. Nugent, et al., "Prevalence of Obstructive Sleep Apnea Among Obese Toddlers and Preschool Children," *Sleep & Breathing* 22, no. 2 (2018): 511–515, https://doi.org/10.1007/s11325-017-1576-4.

9. I. Narang and J. L. Mathew, "Childhood Obesity and Obstructive Sleep Apnea," *Journal of Nutrition and Metabolism* 2012 (2012): 134202, https://doi.org/10.1155/2012/134202.

10. S. L. Verhulst, L. Van Gaal, W. De Backer, and K. Desager, "The Prevalence, Anatomical Correlates and Treatment of Sleep-Disordered Breathing in Obese Children and Adolescents," *Sleep Medicine Reviews* 12, no. 5 (2008): 339–346, https://doi.org/10.1016/j.smrv.2007.11.002.

11. G. B. Mallory, D. H. Fiser, and R. Jackson, "Sleep-Associated Breathing Disorders in Morbidly Obese Children and Adolescents," *Journal of Pediatrics* 115, no. 6 (1989): 892–897, https://doi.org/10.1016/s0022-3476(89)80738-3.

12. R. Carter and D. E. Watenpaugh, "Obesity and Obstructive Sleep Apnea: Or Is It OSA and Obesity?," *Pathophysiology* 15, no. 2 (2008): 71–77, https://doi.org/10.1016/j.pathophys.2008.04.009.

13. T. Young, M. Palta, J. Dempsey, J. Skatrud, S. Weber, and S. Badr, "The Occurrence of Sleep-Disordered Breathing Among Middle-Aged Adults," *New England Journal of Medicine* 328, no. 17 (1993): 1230–1235, https://doi.org/10.1056/NEJM199304293281704.

14. E. Quintana-Gallego, C. Carmona-Bernal, F. Capote, et al., "Gender Differences in Obstructive Sleep Apnea Syndrome: A Clinical Study of 1166 Patients," *Respiratory Medicine* 98, no. 10 (2004): 984–989, https://doi.org/10.1016/j.rmed.2004.03.002.

15. O. Resta, G. E. Carpagnano, D. Lacedonia, et al., "Gender Difference in Sleep Profile of Severely Obese Patients With Obstructive Sleep Apnea (OSA)," *Respiratory Medicine* 99, no. 1 (2005): 91–96, https://doi.org/10.1016/j.rmed.2004.05.014.

16. M. F. Wu, Y. H. Chen, H. C. Chen, and W. C. Huang, "Interactions Among Obstructive Sleep Apnea Syndrome Severity, Sex, and Obesity on Circulatory Inflammatory Biomarkers in Patients With Suspected Obstructive Sleep Apnea Syndrome: A Retrospective, Cross-Sectional Study," *International Journal of Environmental Research and Public Health* 17, no. 13 (2020): 4701, https://doi.org/10.3390/ijerph17134701.

17. P. E. Brockmann, D. Koren, L. Kheirandish-Gozal, and D. Gozal, "Gender Dimorphism in Pediatric OSA: Is It for Real?," *Respiratory Physiology & Neurobiology* 245 (2017): 83–88, https://doi.org/10.1016/j.resp.2016.11.010.

18. S. Redline, A. Storfer-Isser, C. L. Rosen, et al., "Association Between Metabolic Syndrome and Sleep-Disordered Breathing in Adolescents," *American Journal of Respiratory and Critical Care Medicine* 176, no. 4 (2007): 401–408, https://doi.org/10.1164/rccm.200703-375OC.

19. A. Inoshita, T. Kasai, R. Matsuoka, et al., "Age-Stratified Sex Differences in Polysomnographic Findings and Pharyngeal Morphology Among Children With Obstructive Sleep Apnea," *Journal of Thoracic Disease* 10, no. 12 (2018): 6702–6710, https://doi.org/10.21037/jtd.2018.11.09.

20. A. Mattei, G. Tabbia, and S. Baldi, "Diagnosis of Sleep Apnea," *Minerva Medica* 95, no. 3 (2004): 213–231.

21. S. L. Garetz, R. B. Mitchell, P. D. Parker, et al., "Quality of Life and Obstructive Sleep Apnea Symptoms After Pediatric Adenotonsillectomy," *Pediatrics* 135, no. 2 (2015): e477–e486, https://doi.org/10.1542/peds.2014-0620.

22. K. T. Kang, W. C. Weng, T. H. Yeh, P. L. Lee, and W. C. Hsu, "Validation of the Chinese Version OSA-18 Quality of Life Questionnaire in Taiwanese Children With Obstructive Sleep Apnea," *Journal of the Formosan Medical Association* 113, no. 7 (2014): 454–462, https://doi.org/10.1016/j.jfma.2012.10.002.

23. R. S. C. Horne, C. Ong, A. Weichard, G. M. Nixon, and M. J. Davey, "Are There Gender Differences in the Severity and Consequences of Sleep Disordered in Children?," *Sleep Medicine* 67 (2020): 147–155, https://doi.org/10.1016/j.sleep.2019.11.1249.

24. H. Alotair and A. Bahammam, "Gender Differences in Saudi Patients With Obstructive Sleep Apnea," *Sleep & Breathing* 12, no. 4 (2008): 323–329, https://doi.org/10.1007/s11325-008-0184-8.

25. Children's Hospital of Philadelphia, "BMI Z-Score Calculator," https://zscore.research.chop.edu/calcbmi.php.

26. D. Monasor-Ortolá, J. A. Quesada-Rico, A. P. Nso-Roca, et al., "Degree of Accuracy of the BMI Z-Score to Determine Excess Fat Mass Using DXA in Children and Adolescents," *International Journal of Environmental Research and Public Health* 18, no. 22 (2021): 12114, https://doi.org/10.3390/ijerph182212114.

27. C. Iber, S. Ancoli-Israel, A. L. Chesson, and S. Quan, *The AASM Manual for the Scoring of Sleep and Associated Events: Rules, Terminology and Technical Specifications* (American Academy of Sleep Medicine, 2007).

28. S. Savini, A. Ciorba, C. Bianchini, et al., "Assessment of Obstructive Sleep Apnoea (OSA) in Children: An Update," *Acta Otorhinolaryngologica Italica* 39, no. 5 (2019): 289–297, https://doi.org/10.14639/0392-100X-N0262.

29. R. A. Franco, R. M. Rosenfeld, and M. Rao, "First Place—Resident Clinical Science Award 1999. Quality of Life for Children With Obstructive Sleep Apnea," *Otolaryngology and Head and Neck Surgery* 123, no. 1 Pt 1 (2000): 9–16, https://doi.org/10.1067/mhn.2000.105254.

30. E. O. Bixler, A. N. Vgontzas, H. M. Lin, et al., "Sleep Disordered Breathing in Children in a General Population Sample: Prevalence and Risk Factors," *Sleep* 32, no. 6 (2009): 731–736, https://doi.org/10.1093/sleep/32.6.731.

31. R. Heinzer, S. Vat, P. Marques-Vidal, et al., "Prevalence of Sleep-Disordered Breathing in the General Population: The HypnoLaus Study," *Lancet Respiratory Medicine* 3, no. 4 (2015): 310–318, https://doi.org/10.1016/S2213-2600(15)00043-0.

32. K. Gipson, M. Lu, and T. B. Kinane, "Sleep-Disordered Breathing in Children," *Pediatrics in Review* 40, no. 1 (2019): 3–13, https://doi.org/10.1542/pir.2018-0142.

33. M. Piotto, A. Gambadauro, A. Rocchi, et al., "Pediatric Sleep Respiratory Disorders: A Narrative Review of Epidemiology and Risk Factors," *Children (Basel)* 10, no. 6 (2023): 955, https://doi.org/10.3390/children10060955.

34. R. H. Lustig, "Hypothalamic Obesity After Craniopharyngioma: Mechanisms, Diagnosis, and Treatment," *Front Endocrinol (Lausanne)* 2 (2011): 60, https://doi.org/10.3389/fendo.2011.00060.

35. J. L. Goodwin, M. M. Vasquez, G. E. Silva, and S. F. Quan, "Incidence and Remission of Sleep-Disordered Breathing and Related Symptoms in 6- to 17-Year Old Children—the Tucson Children's Assessment of Sleep

Apnea Study," *Journal of Pediatrics* 157, no. 1 (2010): 57–61, https://doi.org/10.1016/j.jpeds.2010.01.033.

36. S. J. Chang and K. Y. Chae, "Obstructive Sleep Apnea Syndrome in Children: Epidemiology, Pathophysiology, Diagnosis and Sequelae," *Korean Journal of Pediatrics* 53, no. 10 (2010): 863–871, https://doi.org/10.3345/kjp.2010.53.10.863.

37. A. Lo Bue, A. Salvaggio, and G. Insalaco, "Obstructive Sleep Apnea in Developmental Age. A Narrative Review," *European Journal of Pediatrics* 179, no. 3 (2020): 357–365, https://doi.org/10.1007/s00431-019-03557-8.

38. R. Dotan, C. Mitchell, R. Cohen, P. Klentrou, D. Gabriel, and B. Falk, "Child-Adult Differences in Muscle Activation—a Review," *Pediatric Exercise Science* 24, no. 1 (2012): 2–21, https://doi.org/10.1123/pes.24.1.2.

39. D. Y. Goh, P. Galster, and C. L. Marcus, "Sleep Architecture and Respiratory Disturbances in Children With Obstructive Sleep Apnea," *American Journal of Respiratory and Critical Care Medicine* 162, no. 2 Pt 1 (2000): 682–686, https://doi.org/10.1164/ajrccm.162.2.9908058.

40. M. Charbonneau, J. M. Marin, A. Olha, R. J. Kimoff, R. D. Levy, and M. G. Cosio, "Changes in Obstructive Sleep Apnea Characteristics Through the Night," *Chest* 106, no. 6 (1994): 1695–1701, https://doi.org/10.1378/chest.106.6.1695.

41. A. R. Jackman, S. N. Biggs, L. M. Walter, et al., "Sleep Disordered Breathing in Early Childhood: Quality of Life for Children and Families," *Sleep* 36, no. 11 (2013): 1639–1646, https://doi.org/10.5665/sleep.3116.

42. S. N. Biggs, L. M. Walter, A. R. Jackman, et al., "Longitudinal Impact of Resolution of Snoring in Young Children on Psychosocial Functioning," *Journal of Pediatrics* 167, no. 6 (2015): 1272–1279, https://doi.org/10.1016/j.jpeds.2015.09.016.

43. R. S. Bourke, V. Anderson, J. S. C. Yang, et al., "Neurobehavioral Function Is Impaired in Children With all Severities of Sleep Disordered Breathing," *Sleep Medicine* 12, no. 3 (2011): 222–229, https://doi.org/10.1016/j.sleep.2010.08.011.

44. A. R. Jackman, S. N. Biggs, L. M. Walter, et al., "Sleep-Disordered Breathing in Preschool Children Is Associated With Behavioral, but Not Cognitive, Impairments," *Sleep Medicine* 13, no. 6 (2012): 621–631, https://doi.org/10.1016/j.sleep.2012.01.013.

45. B. C. Friedman, A. Hendeles-Amitai, E. Kozminsky, et al., "Adenotonsillectomy Improves Neurocognitive Function in Children With Obstructive Sleep Apnea Syndrome," *Sleep* 26, no. 8 (2003): 999–1005, https://doi.org/10.1093/sleep/26.8.999.

46. M. Bublitz, N. Adra, L. Hijazi, F. Shaib, H. Attarian, and G. Bourjeily, "A Narrative Review of Sex and Gender Differences in Sleep Disordered Breathing: Gaps and Opportunities," *Life (Basel)* 12, no. 12 (2022): 2003, https://doi.org/10.3390/life12122003.

47. M. Modig, L. Andersson, and I. Wårdh, "Patients' Perception of Improvement After Orthognathic Surgery: Pilot Study," *British Journal of Oral & Maxillofacial Surgery* 44, no. 1 (2006): 24–27, https://doi.org/10.1016/j.bjoms.2005.07.016.

48. Z. Ye, D. Wu, X. He, et al., "Meta-Analysis of the Relationship Between Bullying and Depressive Symptoms in Children and Adolescents," *BMC Psychiatry* 23, no. 1 (2023): 215, https://doi.org/10.1186/s12888-023-04681-4.

49. T. Lozo, D. Komnenov, M. S. Badr, and J. H. Mateika, "Sex Differences in Sleep Disordered Breathing in Adults," *Respiratory Physiology & Neurobiology* 245 (2017): 65–75, https://doi.org/10.1016/j.resp.2016.11.001.

50. M. Suzuki, Y. Kawai, and Y. Funayama, "Exploring the Dynamics of Snoring in Relation to Sleep Stages: Implications for Gender Differences, Sleep Position, and Upper Airway Collapsibility," *PLoS One* 19, no. 1 (2024): e0295232, https://doi.org/10.1371/journal.pone.0295232.

51. L. Dékány, V. Molnár, A. Molnár, et al., "Analysis of Possible Risk Factors for the Severity of Paediatric Obstructive Sleep Apnoea Syndrome," *European Archives of Oto-Rhino-Laryngology* 280, no. 12 (2023): 5607–5614, https://doi.org/10.1007/s00405-023-08237-w.

52. J. Wang, Y. Zhao, W. Yang, et al., "Correlations Between Obstructive Sleep Apnea and Adenotonsillar Hypertrophy in Children of Different Weight Status," *Scientific Reports* 9, no. 1 (2019): 11455, https://doi.org/10.1038/s41598-019-47,596-5.

53. P. Kaewkul, W. Banhiran, K. Ungkanont, A. Tanphaichitr, C. Chongkolwatana, and V. Vathanophas, "Diagnostic Properties Modified OSA-18 Questionnaire in Children With Severe Obstructive Sleep Apnea," *Journal of the Medical Association of Thailand* 101, no. 4 (2018): 427–432, http://www.jmatonline.com/index.php/jmat/article/view/8083.

Supporting Information

Additional supporting information can be found online in the Supporting Information section.