Symptoms of Sleep Apnea and Polysomnography as Predictors of Poor Quality of Life in Overweight Children and Adolescents

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Objective The goal of this study was to examine the relationship between quality of life (QOL) and symptoms of obstructive sleep apnea (OSA) as well as objectively measured severity of OSA using polysomnography (PSG) in a cohort of overweight and at risk for overweight children and adolescents. **Methods** One hundred and fifty-one overweight subjects [90 males, average ages of 12.52, mean body mass index (BMI) *Z*-score of 2.27) and their parent/guardian completed surveys assessing QOL and symptoms of OSA syndrome. The subjects also underwent overnight PSG. **Results** Overweight patients reported poor QOL. Polysomnographic variables did not correlate with QOL. However, symptoms of OSA as reported on the Pediatric Sleep Questionnaire significantly correlated with QOL from both the parent and the subject. **Conclusions** Overweight youth with symptoms of OSA have a lower QOL both by their report and parental report. Interestingly, objective measures of OSA did not correlate with QOL.

Key words OSA; overweight; quality of life; youth.

Obstructive sleep apnea (OSA) is a syndrome of repetitive closure of the airway during sleep resulting in sleep disturbance, cyclic oxyhemoglobin desaturation, and frequently sustained hypercapnea (Ali, Pitson, & Stradling, 1993; Gislason & Benediktsdottir, 1995). OSA is a common medical problem in children with a prevalence rate of ~2% in the general pediatric population (Redline el al., 1999; Rosen et al., 2003) and is associated with significant cardiopulmonary (Young, Pepperd, & Gottlieb, 2002) and neurobehavioral (Lipton & Gozal, 2003) sequelae. Furthermore, children with sleep apnea consume a disproportionate amount of health care dollars (Reuveni, Simon, Tal, Elhayany, & Tarasiuk, 2002). While snoring is a primary symptom of OSA, clinical history and physical examination alone are poor at distinguishing primary snoring from true obstructive sleep apnea (Carroll, McColley, Marcus, Curtis, & Loughlin, 1995; Lam, et al., 2006; O'Brien & Gozal, 2002; Xu, Chuek, & Lee, 2006). Thus, polysomnography (PSG) is commonly used to establish the presence of and quantify severity of OSA (American Academy of Pediatrics, 2002). Obese children are at increased risk of OSA with reported prevalence rates of 26–46% (Ali et al., 1993; Brouilette et al., 1984; Marcus et al., 1996; Wing et al., 2003). Obesity has reached epidemic proportions in the United States with 15.3% of children and 15.5% of adolescents now classified as overweight [body mass index (BMI) ≥95%ile] (Slyper, 2004). Obesity-associated OSA is likely to affect a growing number of children as the prevalence of childhood obesity increases (Miller, Rosenbloom, & Silverstein, 2004).

The impact of chronic medical conditions on affected individuals is typically assessed using health-related quality of life (QOL) questionnaires in which subjects and their families report a subjective perception of their physical and emotional functioning as it relates to their disease and treatment. Multiple published studies demonstrate that reported QOL is quite poor for overweight youth (de Beer et al., 2007; Fallon et al, 2005; Hughes, Farewell,

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Harris, & Reilly, 2007; Schwimmer, Burwinkle, & Varni, 2003; Swallen, Reither, Haas, & Meier, 2005; Tyler, Johnston, Fullerton, & Foreyt, 2007; Zeller & Modi, 2006) and is likewise poor for those with OSA (Crabtree, Varni, & Gozal, 2004; Mitchell & Kelly, 2004; Rosen, Palermo, Larkin, & Redline, 2002; Stewart, Glaze, Friedman, Smith, & Bautista, 2005; Tran, Nguyen, Weedon, & Goldstein, 2005) mirroring the decrement in QOL reported for children with cancer (Schwimmer et al., 2003) and rheumatologic diseases (Sawyer et al., 2004). Published literature demonstrates that obese children and adolescents have difficulties with physical function and physical health (Friedlander, Larkin, Rosen, Palermo, & Redline, 2003; Swallen et al., 2005), psychosocial function (Wake, Salman, Waters, Wright, & Hesketh, 2002; Friedlander et al., 2003), emotional (Schwimmer et al., 2003) and social (Pinhas-Hamiel et al., 2006; Schwimmer et al., 2003; Williams, Wake, Hesketh, Maher, & Waters, 2005) function, mental health (Wake, Salman, Waters, Wright, & Hesketh, 2002), self-esteem (Friedlander et al., 2003), and overall health (Swallen et al., 2005). Furthermore, self-reported health-related quality of life is inversely related to BMI and is only partially explained by obesity-related comorbidities (deBeer et al., 2007).

The increasing prevalence of childhood obesity predicts an increasing prevalence of both physical health risks associated with being overweight as well as psychological distress associated with obesity. There is a growing awareness in the medical community about the long-term health consequences of childhood obesity, yet the most prevalent complications of childhood obesity may be psychological rather than physical. Overweight youth are more likely to report hopelessness, consider themselves a poor student, attempt suicide, and less likely to spend free time with friends (Falkner et al., 2001). Overweight children are taller than normal weight peers and progress through puberty at a younger age (Wang, 2002). Thus, they frequently appear older than their chronologic age, resulting in unrealistically high expectations being placed. When these expectations are not met, children may become discouraged and develop a negative self-image. Children with OSA frequently struggle academically (Gozal, 1998) and have poor executive functioning (O'Brien et al., 2003), further compounding the image of the overweight child with OSA as socially inept, academically challenged, and physically unfit.

The goal of this study was to examine the relationship between parent and self-reported QOL and symptoms of OSA as well as objectively measured severity of OSA using nocturnal PSG (NPSG) in a cohort of overweight and at risk for overweight children and adolescents. We hypothesized that both symptomatic severity as well as polysomnographic severity of OSA would predict decrements in QOL. The secondary aim of this study was to examine the relationship between parent and self-reported QOL and severity of obesity as measured by body mass index (BMI) *Z*-score. We hypothesized that progressive degrees of obesity would predict measurable decrements in QOL.

Methods

Sample and Procedures

This sample was a convenience sample of subjects consecutively evaluated at a regional pediatric sleep lab for possible OSA. Overweight (BMI > 95%ile) and at-risk for overweight (85%ile≤BMI ≤95%ile) youth using the Centers for Disease Control (CDC) guidelines who presented with habitual snoring (e.g., habitual snoring of at least 3 nights/week) were invited to participate. Normal weight youth (BMI < 85%ile) were not included. Parental written consent and age appropriate verbal or written assent was obtained from the youth at enrollment. Verbal assent was obtained from youth between the ages of 8 and 12 years, and written assent was obtained for those youth between 13 and 17 years of age. Evaluation at the time of study enrollment included history and physical examination, stage of pubertal development, and BMI. The accompanying parent/guardian completed the Pediatric Sleep Questionnaire (PSQ) (Chervin, Hedger, Dillion, & Pituch, 2000) to assess severity of symptoms suggestive of OSA. The subject and accompanying parent/guardian completed questionnaires assessing QOL (Pediatric Quality of Life Inventory 4.0™; Varni, Burwinkle, Seid, & Skarr, 2003). Subjects subsequently underwent NPSG. Subjects and their families returned on a later visit to discuss PSG results. Thus, subjects and their families were unaware of study results at the time of questionnaire completion. All procedures were approved by the local Institutional Review Board.

Measures and Instruments

Anthropometric

Height was measured using a wall mounted stadiometer. Weight was obtained on a calibrated scale. BMI percentile and *Z*-scores were calculated using 2002 CDC growth charts containing age- and gender-specific percentiles (CDC, 2004). Youth were grouped based on the CDC classification in which children with a BMI percentile

(BMI%ile) of 85 or greater for age and gender as ''at risk for overweight'' and those with a BMI%ile of equal to or greater than the 95 percentile for age and gender are classified as overweight. Degree of pubertal development was classified by Tanner stage and was determined by one study team member to control for inter-rater reliability and variance using standard criteria (Burns, Dunn, Brady, Starr, & Blosser, 2004).

Quality of Life

The Pediatric Quality of Life Inventory 4.0 (PedsQL^{TM} 4.0) was used to assess overall self and parental reported QOL. The child report was completed by subjects age 8–12, the Teen report form age 13–18, and parent/guardian completed the corresponding parental report form. Parents and subjects were instructed not to collaborate on the completion of questionnaires but remained in an examination room together. The 23-item PedsQL^{TM} 4.0 takes on average <5 min to complete and assesses physical functioning (eight items), emotional functioning (five items), social functioning (five items), and school functioning (five items). Responses consisted of a 5-point Likert scale in which $0 =$ never a problem and $4 =$ almost always a problem. Items were reverse scored to a scale of 0–100 ($0 = 100$, $1 = 75$, $2 = 50$, $3 = 25$, $4 = 0$) such that higher scores indicated better health-related QOL. A total health-related QOL score was obtained by summing individual question responses. Scale scores are computed as mean of the nonmissing items A published validation study used a cut-off score for subjects and parents report of total health-related QOL score to be >69.71 and 65.43, respectively as normal (Varni et al., 2003). Thus, for the purposes of our study, low QOL was defined as a self-reported total score of ≤ 69.71 or a parentally reported total score of ≤ 65.43. This questionnaire is valid and reliable with a reported α of .91 for the child and teenager scale, and .92 for the parent scale (Varni et al., 2003). This questionnaire was selected because it has been extensively used in the pediatric population and has been used in children with chronic illness (Schwimmer et al., 2003; Varni, Burwinkle, & Katz, 2004).This questionnaire has also been used in children/adolescents with OSA (Crabtree et al., 2004).

OSA

Subjective Symptoms of OSA. The PSQ developed by Chervin et al. (2000) was used to assess subjective symptoms of OSA as reported by the child's parent/guardian. The Sleep Related Breathing Disorder Subscale (SRBD) of the PSQ contains 22 items and assesses three domains: nighttime symptoms, daytime symptoms, and neurobehavioral symptoms associated with sleep apnea. The SRBD of the PSQ demonstrates adequate reliability (Cronbach's $\alpha = .89$; test–retest $= .75$.) (Chervin et al., 2000). A cut-off score of 0.33 (e.g., 33% of the 22 questions/items answered positively) suggests the presence of polysomnographically demonstrable sleep apnea (defined by the authors as an apnea hypopnea index of five events or greater per hour of sleep) with a reported sensitivity and specificity of 0.85 and 0.87, respectively (Chervin et al., 2000).

Objective Measure of OSA. NPSG are noninvasive tests that by definition can objectively quantify the severity of sleep disordered breathing. NPSG was performed in accord with the American Thoracic Society guidelines for sleep studies in children (American Thoracic Society, 1996) and included the recording of electrooculogram, electroencephalogram, submental electromyogram, leg and arm electromyogram, electrocardiogram, body position, videotape (Embla diagnostics equipment and Somnologica software, Medcare, Buffalo, NY,USA), respiratory effort (Xactrace, Medcare, Buffalo, NY, USA), snoring sound (Sleepmate snoring sensor, Sleepmate Technologies Midlothian, VA, USA), nasal air pressure (Pro-Flow Plus, Pro-Tech, Mukilteo, WA, USA), end tidal carbon dioxide (Capnocheck Plus capnograph, Smiths Medical, Weston, MD, USA), and pulse oximetry (Onyx, Nonin Medical Inc. Plymouth MN, USA). Polysomnographic recordings were scored by a single technician certified by the Board of Registered Polysomnographic Technicians. Sleep staging was scored according to published standards (Rechtschaffen & Kales, 1968). Arousals were defined using criteria published by the American Sleep Disorders Association (American Sleep Disorders Association, 1992). Each study was reviewed and interpreted by a single pediatric trained study physician certified by the American Board of Sleep Medicine.

Obstructive apnea was defined as a reduction in airflow signal to $\leq 20\%$ of baseline flow with persistent respiratory effort lasting longer than two baseline breaths, irrespective of changes in oxygen saturation. Central apnea was defined as an absence of respiratory effort associated with absence of airflow lasting longer than 20 s or an absence of respiratory effort associated with absence of airflow of any duration associated with oxygen desaturation of at least 4% culminating in a cortical arousal. Postarousal central apneic events were only scored if followed by an additional arousal or desaturation $>4\%$. Hypopneas were defined as a reduction in airflow signal of at least 50% but not >80% of baseline flow, lasting longer than 10 s and temporally associated with either an arousal on EEG or oxygen desaturation of at least 3% (Uliel, Tauman. Guenther, & Sivan, 2004). Mixed apneas in which both central and obstructive components were present were tallied as mixed events but included as obstructive events in the apnea index (AI). Obstructive apnea and obstructive apnea–hypopnea indices (AHI) were defined as the total number of respective events per hour of recorded sleep. The AHI was used to assess the severity of OSA. Primary snoring was defined as an AHI of <2; mild OSA as an AHI of 2 but <5; and moderate to severe OSA as an AHI of greater than five events per hour. Normative data describing statistically abnormal apnea indices obtained from a population of normal children define an apnea index >1 event/hr (Moser, Phillips, Berry, & Harbison, 1995; Uliel et al., 2004). However, this normative data describes *statistical* abnormality. It remains unknown what level of apnea or hypopnea is *clinically* significant (American Thoracic Society, 1999) and many published studies use an AHI >5 to define OSA (e.g., TuCASA). Thus, we intentionally selected a conservative scale by which to define sleep apnea.

Statistical Analysis

Demographic, questionnaire data, and polysomnographic data are expressed as mean ± standard deviation. Values for AHI were log-transformed to improve normality and the log-transformed values were used in the analyses, unless otherwise noted. Paired *t*-test was used to assess for statistical differences between the parent and youth reported QOL. Stepwise forward linear regression was performed between polysomnographic variables and QOL reports, while controlling for BMI *Z*-score in order to ascertain independent effects of OSA and severity of obesity on QOL. Pearson or Spearman's ρ-correlation was performed between QOL and age, Tanner stage, BMI, polysomnographic and demographic variables. Chi-square and ANOVA analysis was performed to examine differences between groups. Because published literature uses different "cut-off" points to define disease versus nondisease, post hoc data analysis was performed using both a "cut-off" AHI of ≥1 event/hr as well as an AHI of ≥5 events/hr to define the presence of OSA.

Results

The sample consisted of 151 subjects consecutively referred to a regional pediatric sleep center for possible OSA from 2005 to 2007. Subjects in the sample were limited to those who were overweight or at risk for overweight. Normal weight subjects (BMI < 85%ile) were not included. Thirteen (9%) subjects were at risk for overweight and 138 (91%) were overweight per CDC guidelines. NPSG data were available on 96 subjects. There were no statistically significant differences in demographic and anthropometric data obtained from those who underwent and those who failed to complete a NPSG, except that subjects who failed to complete a NPSG had a higher BMI *Z*-score (2.21 vs. 2.38; $p = .017$). Demographic data corresponded to the demographics of the catchment area served by the pediatric sleep center where the study was conducted. BMI *Z*-score did not correlate with either the AHI (using Spearman ρ) or the log transformed AHI (using Pearson) ($r = .118$ and .105; $p > .05$, respectively). BMI *Z*-score did correlate with SaO_2 nadir ($r = -.211$, $p = .04$) but did not correlate with other measured polysomnographic measures including total sleep time (TST). General overall information on PSG variables is presented in Table I and differences in PSG data categorized by severity of OSA are presented in Table II.

Polysomnographic Variables and Parent Reported Symptoms of OSA

Of the subjects with a NPSG, 7.3% had an AHI of <1, 4.1% had an AHI 1.0–1.9, 27.1% had an AHI of 2.0–4.9,

Table I. Demographic and Anthropometric Characteristics of Youth, Total Sample, those with Primary Snoring and those with OSA

	Total group	Primary snoring (AHI<2 events per hour)	Mild OSA (AHI = 2 but <5 events per hour)	Moderate to severe OSA (AHI = or> 5 events per hour)
Number in group	151	13	24	59
Age (years) (mean ± *SD*)	12.52 ± 2.85	11.36 ± 3.41	12.25 ± 3.04	12.86 ± 2.64
Male (*N*,%)	*N* = 90; 60%	*N* = 5; 39%	*N* = 13; 54%	*N* = 39; 66%
African American (N,%)	*N* = 56; 37%	*N* = 3; 23%	*N* = 10; 42%	*N* = 23; 39%
Hispanic (*N*,%)	*N* = 13; 9%	*N* = 2; 15%	*N* = 0	*N* = 6; 10%
BMI%ile (mean ± *SD*)	98.10 ± 2.26	98.55 ± 1.25	97.36 ± 2.84	97.98 ± 2.02
BMI Z-score (mean ± *SD*)	2.27 ± 0.43	2.30 ± 0.36	2.11 ± 0.42	2.23 ± 0.43

No significant differences between primary snoring, mild OSA and moderate to severe OSA. *SD*, standard deviation; *N*, number; BMI, body mass index.

21.9% had an AHI of 5 −9.9, and 39.6% had an AHI ≥ 10 events/hr. The PSQ correlated positively with AHI (Spearman $\rho = .238$, $p = .022$) and negatively with the nadir O_2 saturation ($r = -.369$, $p < .01$). The Cronbach's α from our study population was .765. There was no difference in PSQ score between those who underwent and those who failed to complete PSG. Using a cut-off score of 0.33 (e.g., 33% of the 22 items answered positively), the PSQ correctly identified OSA with a sensitively of 84% but only a specificity of 23% using an AHI ≥5 (as was used for the validation study performed by Chervin et al., 2000).

Quality of Life

No differences were found between the reported child and teen PedsQL™ 4.0 and the content is identical between the two versions of the questionnaires. Therefore, the data for the Child and Teen questionnaire were combined for analysis purposes and is labeled ''Youth'' QOL. Parent/guardian QOL total score was significantly correlated with the subjects reported QOL total score ($r = .43$, $p < .001$). Examining QOL subscale scores between parent and subject indicated that all four subscales were also significantly correlated ($r = .38$–.46, $p < .05$). However, parents consistently reported significantly lower mean scores than their youth on the total QOL and all subscales ($p < .05$) as shown in Table III. The Cronbach's α for our study population was .90 for both the parent QOL total score and the youth QOL total score. Using the published cut-off score of 65.43 for parent report and 69.71 for child/adolescent indicating low QOL, 66% of parents and 60% of subjects reported a substandard QOL. Neither Tanner stage nor chronological age significantly contributed to the prediction of total or subscale QOL as reported by either parents or study subjects.

QOL and Obesity

BMI Z-score predicted parental reported physical functioning ($\beta = -.22$, Adj $R^2 = .08$, $p < .05$) and weakly correlated with parental reported total QOL ($r = -0.17$, $p < .05$). BMI Z-score did not correlate with subject-reported total or subscale QOL. Neither age nor pubertal status correlated with severity of obesity.

QOL and OSA

Neither AHI, AI, peak $ETCO_2$, time spent with $ETCO_2 > 45$ mmHg; time spent with $ETCO_2 > 50$ mmHg nor sleep efficiency significantly contributed to the prediction of total or subscale QOL of either parent or youth surveys. There were no significant differences in reported QOL between the primary snoring group compared to subjects with an AHI of 2 or greater. Fifty-five percent of parents and 64% of children with primary snoring reported substandard QOL, whereas 62% of parents and 60% of children with OSA reported substandard QOL. As shown in Table IV, whether OSA was defined as an AI of 1 event/hr or greater; an AHI of 2 events/hr

Table III. Parental Report versus Youth Report

	Parental report (Mean ± *SD*) *N* = 147	Youth report (Mean ± *SD*) *N* = 146	*t*(df)
Total score	58.71 ± 16.93	65.46 ± 17.15	−4.48(143)*
Physical function	60.94 ± 22.68	71.99 ± 19.45	−5.66(143)*
Emotional function	56.59 ± 20.94	60.53 ± 21.11	−2.09(142)*
Social function	64.35 ± 23.76	72.06 ± 23.48	−3.58(143)*
School function	53.03 ± 18.75	57.34 ± 20.97	−2.62(141)*

*$p<0.05$.

Table II. Polysomngraphic Characteristics of Youth with Primary Snoring and OSA

	Primary Snoring (AHI< 2) (*N* = 13)	Mild OSA (AHI= 2 but less than 5) (*N* = 24)	Moderate to Severe OSA (AHI = or > than 5) (*N* = 59)
AHI (events/hour)	1.10 ± 0.41	3.4 ± 0.64	25.32 ± 29.80#,*
AI (events/hour)	0.08 ± 0.28	0.78 ± 1.17	43.31 ± 133.10
CAI (events/hour)	0.69 ± 1.49	1.11 ± 1.39	1.06 ± 2.54
Mean SaO_2(%)	97.05 ± 0.82	97.06 ± 0.81	94.86 ± 3.21#,*
SaO_2 (nadirt) (%)	88.62 ± 11.0	88.67 ± 4.52	82.78 ± 10.20*
Peak $ETCO_2$	49.35 ± 3.78	49.01 ± 2.40	50.06 ± 4.93
% TST $ETCO_2 > 45$ mmHg	30.13 ± 30.77	23.87 ± 24.66	21.00 ± 24.08
% TST $ETCO_2 > 50$ mmHg	0.11 ± 0.267	0..32 ± 1.31	1.27 ± 4.40
TST (min)	397.56 ± 47.82	384.87 ± 81.36	391.073 ± 67.30
PSQ score	0.53 ± 0.22	0.47 ± 0.17	0.57 ± 0.19

All results are reported as mean ± SD.

#$p < 0.05$ primary snoring versus moderate to severe OSA; *$p < 0.05$ mild OSA versus moderate to severe OSA.

AHI, apnea–hypopnea index; AI, apnea index; CAI, central apnea index; SaO_2, oxygen saturation; $ETCO_2$, end tidal carbon dioxide level; TST, total sleep time; PSQ, Pediatric Sleep Questionnaire.

Table IV. Percentages of Reported Poor QOL by AHI level

	Parent (reported QOL <65.43)	Youth (reported QOL <69.71)
AHI <2	62%	69%
AHI 2–4.9	50%	58%
AHI 5-or greater	64%	54%

Table V. Contribution of PSQ to QOL

	Parental		Youth	
	β	Adj. R^2	β	Adj. R^2
Total score	−0.43	.18*	−0.38	.14*
Physical functioning	−0.22	.04*	−0.23	.04*
Emotional functioning	−0.39	.14*	−0.34	.11*
Social functioning	−0.33	.11*	−0.22	.04*
School functioning	−0.44	.19*	−0.47	.21*

*$p < 0.05$.

PSQ, Pediatric Sleep Questionnaire; β, Beta; Adj.R^2, Adjusted *R*-square.

or greater or an AHI of 5 events/hr, there was no difference in QOL between those with and those without OSA. Youth emotional function correlated with OSA (Spearman $\rho = .224$; $p < .05$) but other youth QOL subscales did not. No parentally reported QOL subscales correlated with severity of OSA as measured by AHI.

Interestingly, PSQ significantly predicted QOL on all total and subscales for both parental and youth report as shown in Table V. Using the published cut-off scores for QOL, a PSQ score ≥0.33 confers a relative risk for poor quality of life (parental report) of 89%, while for youth it is 68% in our sample.

Discussion

QOL in youth is decreased in a number of chronic medical conditions ranging from velopharyngeal insufficiency to chronic pain (Barr, Thibeault, Muntz, & de Serres, 2007; Connelly & Rapoff, 2006; Merlijn et al., 2006; Youssef, Murphy, Langseder, & Rosh, 2006). Youth with recurrent headaches report lower physical functioning and psychosocial functioning when compared to healthy controls. Furthermore, reported QOL improves with response to treatment suggesting a cause-and-effect relationship (Connelly & Rapoff, 2006). Youth with chronic pain report a lower QOL that correlates with increased pain intensity and frequency. Psychosocial factors including vulnerability, reinforcement, modeling and coping are also strongly associated with QOL suggesting that adaptive family routines predict improved QOL (Merlijn et al., 2006).

Our results show that QOL is quite low in overweight and at risk for overweight youth who snore. Parents consistently report lower QOL for their children than the subjects themselves. Reported QOL in our population of overweight habitually snoring subjects is similar to that reported in the literature (Zeller & Modi, 2006) for overweight pediatric patients not screened for snoring. Recent studies (O'Brien et al., 2004; Urschitz et al., 2004) have reported negative health consequences associated with snoring. While adverse health consequences of OSA have been well described, it is now becoming clear that snoring alone may confer negative risk for health-related outcomes as well. We found no correlation between polysomnographic measures of OSA and QOL, but did find a substantial relationship between symptoms of OSA (as measured by the PSQ) and QOL. One possible explanation for these findings is that variables not traditionally measured on NPSG result in decreased QOL. Alternatively, children with milder forms of OSA and those with primary snoring may experience a more significant negative impact on health and well-being than previously appreciated. As the field of pediatric OSA progresses, more sensitive and sophisticated measures/variables, obtained during night time sleep studies may come to the forefront and be better predictors of QOL.

Our results are in agreement with the published results from Crabtree, Varni, and Gozal. In their study, 85 clinically referred, snoring children (8–12 years) were compared to 35 asymptomatic children in relation to QOL and depressive symptoms. Using multiple cut points in AHI to define OSA, the authors did not find a difference in QOL based on definition of OSA. Overall, snoring children had a lower reported QOL grouping comparison to nonsnoring controls. (Crabtree et al., 2004).

We did not find a correlation between TST and youth or parentally reported QOL. Published literature (Wolfson & Carskadon, 1998) demonstrates that adolescents who sleep less by self-report are more likely to struggle academically, report daytime sleepiness, depressive mood, and sleep/wake behavior problems. In a large ($n = 2{,}259$) cohort study of Illinois middle school students, Fredriksen, Rhodes, Reddy and Way (2004) showed that students who reported less sleep on school nights reported heightened levels of depressive symptoms and decreased self-esteem. It is likely that our study failed to identify a relationship between sleep time and QOL because sleep time was measured during overnight PSG rather than by self-report or by more longitudinal measures in the home such as actigraphy.

Our data demonstrated that children with obesity have lower parental reported physical functioning but that children do not report this difference. This is similar to previously reported findings in the literature (Pinhas-Hamiel et al., 2006). It is possible that parental concerns for their child's health resulting from the adult's better understanding of the health-related consequences of obesity. Another possibility could be that the youth have ''adjusted'' to the lower functioning and perceive their QOL as normal.

Finally, we examined only, QOL in the subjects. It is known that there are other psychological concerns in children who are overweight. These issues include anxiety, depression, and low-self esteem (Young-Hyman et al., 2006). These other factors could be contributing to the low QOL exhibited by our subjects in addition to the OSA and weight issues.

This study has a number of strengths. This is an important study given the rising prevalence of both obesity and OSA, the interaction between them as well as the known morbidities for each. Full NPSG was utilized including end-tidal capnography, thereby enabling accurate detection of sleep-associated gas exchange abnormalities. The relatively large study population accurately reflects a ''typical'' population of overweight children and adolescents referred for possible OSA. Stratification based on pubertal development allowed us to assess for developmental aspects of the impact of obesity and OSA on QOL.

There are also several limitations to this study. Many subjects failed to complete recommended PSG. It is possible that a larger sample size could have indicated additional correlations between polysomnographic variables and QOL. Virtually, all of the children in our sample were at or about the 95%ile for BMI. Thus, the BMI *Z*-score is severely restricted. Thus, it is likely that significant correlations between BMI *Z*-score and QOL could have been identified if the distribution of BMI *Z*-scores was more evenly dispersed amongst the at-risk for overweight and overweight categories or if a group of normal weight children had been included. Likewise, it is also possible that significant correlations between NPSG variables and stronger correlations between the PSQ score and QOL could have been identified if a group of normal children without evidence of obstructive breathing during sleep had been used as the control group rather than children with habitual snoring. Larger studies including normal weight youth and those without symptoms suggestive of OSA are needed.

Interpretations of pediatric NPSGs may vary considerably from one laboratory to the next. To date, only three studies have presented normal PSG values for children and adolescents (Marcus et al., 1992; Montgomery-Downs, O'Brien, Gulliver, & Gozal, 2006; Uliel et al., 2004) and specific definitions of scoreable events differ even between these two studies. Whereas definitions of obstructive apneas are relatively straight forward, subtleties in the definition of hypopneas vary widely. Some laboratories use a definition that reflects the duration of pediatric apneas (i.e., 2-breath rule), while other laboratories (including our own) have undertaken a definition that mirrors the adult definition of hypopneas (i.e., 10 s duration) making comparison of results between laboratories more difficult.

In summary, this study demonstrates poor QOL as reported by both youth and their parents in a cohort of overweight and at risk for overweight pediatric patients evaluated for possible OSA. An increase in parental-reported symptoms of OSA predict poor QOL, whereas polysomnographic measures do not, suggesting that the symptoms of OSA themselves may impact negatively on daily functioning. If confirmed in future studies, QOL may become an important variable to assess the need for intervention in the treatment of snoring youth.

Conflicts of interest: None declared.

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