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# Relationships between obstructive sleep apnea, anthropometric measures, and neurocognitive functioning in adolescents with severe obesity

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## Abstract

**Objective—**We explored whether there were associations between measures of obstructive sleep apnea (OSA) and sleep quality, anthropometrics, and neurocognitive functioning in severely obese adolescents.

**Study design—**This was a cross-sectional pilot study performed at an academic medical center in 37 severely obese (BMI >97%ile) adolescents. Study evaluations included polysomnography, BMI and waist circumference, and standardized neurocognitive tests to assess memory, executive functioning, psychomotor efficiency, academic achievement, and an approximation of full-scale IQ. Outcome data were evaluated categorically, based on clinically utilized criteria for the diagnosis of OSA, and continuously to quantify associations between sleep parameters, anthropometrics, and neurocognitive test results.

**Results—**Sleep fragmentation and poorer sleep quality were associated with reduced psychomotor efficiency, poorer memory recall, and lower scores on standardized academic tests. Having evidence of OSA was associated with lower math scores, but not with other neurocognitive measures. BMI and waist circumference were negatively associated with $SaO_2$.

**Conclusion—**Our pilot study findings suggest that sleep fragmentation and poorer sleep quality have implications for the neurocognitive functioning of obese adolescents. The epidemic of



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childhood obesity has dire implications; not only for increasing cardiometabolic pathology, but potentially also for promoting less apparent neurologic alterations associated with poor sleep quality.

**Keywords**

sleep disordered breathing; obstructive sleep apnea; cognitive functioning; obesity; body mass index; waist circumference

Severe childhood obesity is strongly associated with severe adult obesity and co-morbidities, including obstructive sleep apnea (OSA) (1, 2). OSA is characterized by episodes of airflow limitation, hypoxemia, and disruption of the restorative qualities of sleep (3). Although the consequences of severe OSA are clear, whether or not mild OSA adversely affects cognition in obese adolescents is not understood. There is some evidence that mild OSA in adults is *not* associated with neurocognitive deficits (4). It is not clear whether this is true in adolescents, whose cortical synaptic development is occurring rapidly. We assessed the associations between the presence and degree of OSA and other measures of sleep quality, anthropometrics, and neurocognitive functioning, in 37 severely obese adolescents (BMI >97%ile) without clinically apparent OSA.

## Methods

The study was approved by the University of Pittsburgh Institutional Review Board and performed in the Pediatric Clinical and Translational Research Center at Children's Hospital of Pittsburgh. Patients referred for evaluation of obesity who met study criteria were invited to participate. After obtaining informed assent/consent, we studied 37 severely obese (BMI >97$^{th}$ %ile), non-diabetic, otherwise healthy adolescents (eligible age range 12–18 y; mean age=14.5±1.7 y; 54% male; 40% minority race). Exclusion criteria included airway disease, smoking, chronic disease or medications, psychiatric diagnoses, and obesity associated with a syndrome, such as Prader-Willi syndrome.

Overnight polysomnogram (PSG) was performed and data were recorded using Sensormedics Somnostar Pro version 7.2 software and analyzed for sleep and respiratory variables. Central and obstructive apneas were scored separately and the apnea-hypopnea index (AHI; number of obstructive apneas + hypopneas/hour of sleep) was hand-scored utilizing pediatric criteria (5). For our purposes, sleep efficiency refers to the percentage of the total recording time spent asleep.

The cognitive assessment measured an approximation of full-scale IQ (Wechsler Abbreviated Scale of Intelligence [WASI]), short-term memory recall (Rey Auditory Verbal Learning Test [RAVLT], list B recall), executive functioning (Stroop Color and Word Test), psychomotor efficiency (Grooved Pegboard), and reading, spelling and math achievement (Wide Range Achievement Test [WRAT]). Tests were administered under the direction of a licensed clinical psychologist on the day prior to the PSG.

Two-sided t tests were used to compare continuous variables between individuals with AHI≥1.5 and those with AHI<1.5; as previous studies show that healthy children and adolescents have AHI<1.5 (6, 7). Mann Whitney U tests were utilized for non-normally distributed outcomes. Because of the small sample size of this pilot study, Cohen *d* was calculated to measure effect sizes, defined as small (*d*=0.2), medium (*d*=0.5), and large (*d*=0.8) (8). Pearson correlation coefficients were calculated for parametric data and nonparametric correlation coefficients were calculated to quantify the associations between

sleep parameters, anthropometric measures, and neurocognitive test results. Data are expressed as the mean ±SEM.

## Results

Total sleep time was 420±5.3 minutes (range 354–484). Thirty-three participants snored (90%). Twenty had no evidence of apnea/hypopnea (AHI<1.5), and 17 had some degree of OSA: 9 had AHI ≥1.5 – <5, 2 had AHI ≥5 – <10, and 6 had AHI ≥10. The AHI was positively associated with the arousal index ($r$=0.45, $p$=0.005) and negatively associated with sleep time ($r$=−0.34, $p$=0.039), sleep efficiency ($r$=−0.66, $p$<0.001), and mean and minimun oxygen saturation ($SaO_2$; $r_{mean}$=−0.73, $p$<0.001; $r_{min}$=−0.77, $p$<0.001).

Participant characteristics and results according to AHI are shown in Table I. Comparison of continuous variables between individuals with AHI≥1.5 and those with AHI<1.5 showed that participants with AHI ≥1.5 had lower math computation scores (Mann-Whitney $U$=107, $p$=0.055, $d$=−0.54). There were no significant differences in IQ, short-term memory recall, executive functioning, psychomotor efficiency, or reading and spelling achievement scores in participants with AHI≥1.5 versus those with AHI<1.5.

Results of correlation analysis to quantify the associations between sleep parameters, anthropometric measures, and neurocognitive test results are shown in Table II. More frequent arousals were associated with slower performance on the Grooved Pegboard and lower vocabulary scores, and poorer sleep efficiency was associated with worse memory recall (*RAVLT*) and lower spelling scores. Minimum and mean $SaO_2$ values were not associated with measures of neurocognitive functioning, but were both negatively associated with BMI and waist circumference. Shorter sleep times during the PSG were also associated with higher BMI values.

## Discussion

We show that in severely obese adolescents, sleep fragmentation is associated with reduced psychomotor efficiency and lower scores on standardized vocabulary testing, and poorer sleep efficiency is associated with poorer memory recall and lower spelling scores. These findings indicate that clinically unrecognized sleep disruption leading to poorer sleep quality has implications for the neurocognitive functioning of obese adolescents. Having evidence of OSA also appears to be associated with lower scores on a speeded measure of computational math. From a neuropsychological perspective, this is intriguing because optimal performance on *WRAT* math requires the ability to focus attention and hold relevant information in working memory while rapidly performing a series of increasingly challenging problems. Math scores may be sensitive to OSA because multiple component cognitive skills have been compromised to some extent. These effects are not likely to be due to differences in the classroom experience because subjects with and without OSA performed equally well on reading, which relies on performance of a highly practiced school-based skill.

Other studies in children have shown relationships between primary snoring or mild OSA and measures of attention, neurocognitive functioning, and school performance including math and spelling scores; however, the majority of studies are in younger children (9–13). The relationships between OSA and neurocognitive functioning in the obese adolescent population are relatively unexplored. Decreased cognitive functioning among a large sample of obese children has been shown and functional changes have been demonstrated in the brains of obese adolescents compared with lean adolescents (14). However, information is lacking on the additional burden of OSA and whether OSA was a contributing factor to these observations. One previous study demonstrated that OSA was related to deficits in

memory, learning, and vocabulary among 14 obese children referred for evaluation (15). To our knowledge, no additional studies focusing on OSA and cognition in obese adolescents have been reported.

Consistent with previous study findings, we found significant relationships between measures of BMI and waist circumference with both shorter sleep times and greater oxygen desaturation during overnight sleep evaluation (16, 17). There is substantial evidence in the literature that short sleep duration is associated with obesity, and this is apparent in children and adolescents (16). Moreover, we have previously reported a significant relationship between visceral adiposity and sleep-disordered breathing among obese adolescents (17). These findings highlight the importance of the relationships between sleep quality and adiposity in the pediatric population. We speculate that poor sleep quality and OSA, in combination with severe obesity and its associated cardiometabolic risk factors may promote subtle neurovascular alterations leading to mild changes in cognitive functioning, which may progressively worsen with increasing age and obesity. Longitudinal observations should test this postulate.

Obesity hypoventilation syndrome is characterized by recurrent episodes of upper airway obstruction that result in disruption of the restorative qualities of sleep, hypoventilation, hypoxemia, and hypercapnea during sleep and wakefulness. It is associated with significant cardiometabolic clinical sequelae (18). Other features of the syndrome are morning headaches, excessive daytime sleepiness, difficulty in concentrating, and impaired memory, all of which are associated with poorer school performance (18, 19). A limitation of our study is that we were unable to objectively quantify end-tidal $CO_2$ values and thus, cannot comment on the presence of obesity hypoventilation syndrome in the study population and whether it impaired neurocognitive functioning. It will be important to include end tidal $CO_2$ measures in future studies evaluating sleep quality, OSA, and neurocognitive functioning in obese pediatric subjects.

Even though this pilot study is small and cross-sectional in design, these findings highlight the need to probe the relationships between sleep quality and OSA, and neurocognitive functioning in obese adolescents. It will also be crucial to consider the impact of socioeconomic status and parental education. The epidemic of childhood obesity has dire implications; not only for increasing cardiovascular and metabolic pathology, but also for promoting less apparent neurologic alterations associated with poor sleep quality. This has implications not only for clinical care, but also for educational goals, as high degrees of social and academic functioning are necessary for success in an increasingly demanding, technologically-driven society

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**Table 1**

Participant characteristics and neurocognitive functioning test results of study participants according to AHI

| | **AHI <1.5 (N = 20)** | **AHI ≥1.5 (N = 17)** | **Cohen's *d*** | ***P*** |
|---|---|---|---|---|
| Age (years) | 14.3 ± 0.4 | 14.9 ± 0.4 | | 0.318 |
| BMI (kg/m$^2$) | 37.4 ± 1.5 | 41.8 ± 2.2 | 0.54 | 0.113 |
| BMI SDS* | 3.64 ± 0.28 | 4.48 ± 0.42 | 0.56 | 0.096 |
| Waist circumference (cm) | 112.0 ± 3.0 | 120.9 ± 4.4 | 0.55 | 0.099 |
| *WRAT* math computation T score | 96.2 ± 2.7 | 87.9 ± 4.4 | −0.54 | 0.055 |
| *WRAT* spelling T score | 97.6 ± 4.9 | 89.8 ± 5.0 | −0.37 | 0.271 |
| *WRAT* reading T score | 105.2 ± 3.9 | 102.5 ± 6.1 | −0.12 | 0.706 |
| *WASI* matrix reasoning T-score (abstract problem solving) | 49.0 ± 2.2 | 49.2 ± 2.4 | 0.03 | 0.930 |
| *WASI* vocabulary T-score | 50.7 ± 2.4 | 47.7 ± 2.7 | −0.27 | 0.412 |
| *WASI* IQ score, abbreviated | 97.8 ± 3.4 | 95.4 ± 4.0 | −0.16 | 0.638 |
| *RAVLT* List B (short-term recall) | 6.3 ± 0.4 | 5.6 ± 0.5 (n=16) | −0.36 | 0.283 |
| Grooved Pegboard, seconds for dominant hand (psychomotor speed) | 80.2 ± 3.9 | 83.0 ± 4.2 | 0.16 | 0.352 |
| Grooved Pegboard, seconds for non-dominant hand | 87.3 ± 4.1 | 89.6 ± 4.7 | 0.12 | 0.772 |
| *Stroop* Color and Word T score (executive functioning) | 44.2 ± 2.0 | 42.5 ± 2.3 | −0.18 | 0.602 |

*standard deviation score

**Table 2**

Correlations of sleep, anthropometric, and neurocognitive functioning measures

| | *RAVLT* B Score | Grooved Pegboard[§] | WRAT math | WRAT spelling | WASI vocab | BMI | BMI SDS[£] | Waist Circ. |
|---|---|---|---|---|---|---|---|---|
| Sleep Time | 0.220 | −0.237 | 0.131 | 0.018 | −0.013 | −0.331** | −0.363** | −0.283 |
| Arousal index | −0.255 | 0.322* | −0.137 | −0.031 | −0.349** | 0.015 | 0.030 | −0.049 |
| Sleep efficiency | 0.324* | −0.252 | 0.220 | 0.326** | −0.099 | −0.277 | −0.257 | −0.286 |
| Minimum $SaO_2$ | 0.138 | −0.230 | 0.192 | 0.206 | 0.091 | −0.316 | −0.281 | −0.396** |
| Mean $SaO_2$ | 0.170 | −0.136 | 0.088 | −0.003 | 0.032 | −0.439¥ | −0.404** | −0.476¥ |
| AHI | −0.314 | 0.271 | −0.177 | −0.220 | −0.045 | 0.225 | 0.246 | 0.226 |
| BMI | −0.109 | 0.015 | −0.195 | −0.093 | −0.136 | | 0.958€ | 0.804€ |
| BMI SDS | −0.122 | 0.036 | −0.174 | −0.130 | −0.133 | | | 0.811€ |
| Waist circ | −0.128 | 0.075 | −0.282 | −0.185 | −0.052 | | | |

*$p$=0.05;

**$p$<0.05;

¥$p$<0.01;

€$p$<0.001

§longer times indicate less efficiency

£standard deviation score