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Original Article

# Cognitive and academic functions are impaired in children with all severities of sleep-disordered breathing

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ABSTRACT

*Study objective:* The impact of the broad spectrum of SDB severity on cognition in childhood has not been well studied. This study investigated cognitive function in children with varying severities of SDB and control children with no history of SDB.

*Methods:* One hundred thirty-seven children (75 M) aged 7–12 were studied. Overnight polysomnography (PSG) classified children into four groups: primary snoring (PS) ($n$ = 59), mild obstructive sleep apnea syndrome (OSAS) ($n$ = 24), moderate/severe OSAS ($n$ = 19), and controls ($n$ = 35). Cognition was measured with a short battery of psychological tests including the Wechsler Abbreviated Scale of Intelligence (WASI), the Wide Range Achievement Test-3rd Edition (WRAT-3), the Rey Complex Figure Test (RCFT) and the Controlled Oral Word Association Test (COWAT).

*Results:* There was lower general intellectual ability in all children with SDB regardless of severity. Higher rates of impairment were also noted on measures of executive and academic functioning in children with SDB.

*Conclusions:* Our findings suggest that neurocognitive deficits are common in children with SDB regardless of disease severity, highlighting that such difficulties may be present in children in the community who snore but are otherwise healthy; thus our results have important implications for the treatment of pediatric SDB.



## 1. Introduction

Sleep disordered breathing (SDB) is a common but under-diagnosed condition in children ranging in severity from Primary Snoring (PS) with no associated hypoxia or sleep disruption, to obstructive sleep apnea syndrome (OSAS). OSAS is characterised by snoring associated with sleep fragmentation, exaggerated upper airway resistance, intermittent hypoxia, hypercarbia, apnea, and repeated arousals. The incidence of PS in children has been reported to be between 7% and 34.5% [1–4] and OSAS between 0.7% and 3.0% [5–7].

In adults OSAS is associated with deficits in a number of cognitive domains, and there is now mounting evidence that children with moderate to severe SDB are at increased risk of deficits in attention and concentration [5,8,9], intelligence [10,11], executive function [12–18], memory, learning and school performance [8,19–26]. The link between SDB and cognitive deficits is further strengthened by reports of improvement in function following treatment of SDB with adenotonsillectomy [27–30]. It has been suggested that a combination of repeated episodes of hypoxia and sleep disruption, both features of OSAS but not PS, contribute to these cognitive impairments [31].

Previously it was thought that only the severe end of the SDB spectrum (OSAS) was of functional significance in children. More recent studies, however, have suggested that even mild SDB can have a significant effect on children's cognition [8,17,32]. These studies included methodological weaknesses which may have confounded the interpretation of findings. For example, not all studies used the gold standard of polysomnography (PSG) for the assessment of SDB severity, and several included children with PS as controls rather than recruiting a healthy comparison group. As PS represents the majority of cases referred for clinical assessment, it is vital to determine the level of severity of SDB that is associated with adverse neurobehavioral effects.

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The issue is of particular importance as childhood is a vulnerable period of CNS development [33–36]. In support of the unique vulnerability of the child's brain to mild and/or transient disruptions, research investigating other childhood conditions has documented subtle consequences not observed in adult populations [34,35]. This age-specific effect is critical as the effects of SDB in childhood may not be completely reversible [12,21,37,38]. Thus, the current study aimed to compare cognitive and academic function in children with varying degrees of SDB as defined by PSG with control children with no history of SDB. We predicted that children with SDB would show impaired cognitive and academic performances compared to age-matched controls and that the degree of impairment would be related to SDB severity.

## 2. Methods

Ethical approval for this study was obtained from the Southern Health and Monash University Human Ethics Committees. Written informed consent was obtained from parents and verbal assent from the children prior to commencement of the study. This study formed part of a larger study which also evaluated the effects of SDB on blood pressure and cardiovascular control in these children funded by the National Health and Medical Research Council of Australia.

### 2.1. Participants

Children aged between 7 and 12 years referred for suspected SDB were recruited prior to their clinical PSG study. All children underwent a thorough medical examination performed by a pediatrician. Height and weight were recorded and body mass index (BMI) calculated. BMI was converted to BMI *z*-score [39]. Patients taking medications known to affect sleep or with medical conditions known to affect sleep, breathing, blood pressure, or neurobehavioral function, such as craniofacial syndromes, previous otorhinolaryngologic surgery, renal disease, cerebral palsy, developmental delay, and intellectual disability, were not recruited. Control children with no symptoms of SDB were recruited from the general community via poster and email advertisements and underwent an identical protocol.

Of the 155 children recruited (39 controls, 67 PS, 26 mild OSAS and 23 moderate/severe (MS) OSAS) as part of the larger project, 137 (35 controls, 59 PS, 24 mild OSAS and 19 MS OSAS) completed the cognitive and academic performance protocol and are included in the analyses presented. Demographic and polysomnographic characteristics are presented in Table 1. Parents completed the Behavior Rating Inventory of Executive Function (BRIEF) and the Child Behavior Checklist (CBCL) on the night of their child's PSG study with respect to their child's behavior over the previous six months; the results of this aspect of the study are reported in a separate paper [40]. Results of spectral EEG analysis from this group have been previously reported [41].

### 2.2. Assessment of sleep disordered breathing severity

Each child underwent full overnight PSG using standard clinical pediatric techniques and a commercially available system (Series S Sleep System, Compumedics, Melbourne, Australia). Electroencephalograms (EEG: C4/A1, O2/A1), electrooculograms (EOG: left and right outer canthus), a submental electromyogram (EMG), electrocardiogram (ECG), left and right leg EMG and body position were recorded. Oxygen saturation ($SaO_2$) was measured by pulse oximetry (Biox 3700e, Ohmeda, Boulder, CO, USA) and thoracic and abdominal breathing movements were recorded via uncalibrated respiratory inductance plethysmography (z-RIP belts, Pro-Tech Services Inc., Mukilteo, WA, USA). Both end tidal and transcutaneous carbon dioxide ($PetCO_2$: Capnocheck Plus, BCI Inc., Waukesha, WI, USA; $TCO_2$: TCM3, Radiometer, Copenhagen, Denmark) were recorded, and airflow was measured via nasal pressure and oronasal thermistor (Compumedics, Melbourne, Australia). In addition to standard PSG recordings, continuous BP recordings were made using finger photoplethysmography (Finometer™, Finapres

**Table 1**
Demographics and respiratory characteristics of control and SDB children.

| | Control ($n$ = 35) | PS ($n$ = 59) | Mild OSAS ($n$ = 24) | MS OSAS ($n$ = 19) |
|---|---|---|---|---|
| Gender | 17M/18F | 39M/20F | 13M/11F | 8M/11F |
| Age (y) | 9.6 ± 0.3 | 9.8 ± 0.2 | 9.1 ± 0.3 | 9.0 ± 0.3 |
| SES | 3.7 ± 0.1 | 4.2 ± 0.1[$] | 4.2 ± 0.1 | 4.4 ± 0.2[*] |
| BMI *z*-score | 0.6 ± 0.2 | 0.6 ± 0.2 | 0.6 ± 0.2 | 1.5 ± 0.2[*,#,†] |
| OAHI (hTST) | 0.1 ± 0.04 | 0.3 ± 0.04 | 2.4 ± 0.2 | 15.9 ± 3.0[*,#,†] |
| RDI (hTST) | 0.7 ± 0.1 | 0.9 ± 0.2 | 3.2 ± 0.3 | 16.8 ± 2.9[*,#,†] |
| ArI | 11.1 ± 0.6 | 11.0 ± 0.5 | 13.5 ± 0.9 | 23.8 ± 2.6[*,#,†] |
| $SpO_2$ nadir | 93.7 ± 0.7 | 93.1 ± 0.4 | 92.6 ± 0.6 | 88.2 ± 1.2[*,#] |
| % Time $SpO_2$ <90% | 0.03 ± 0.03 | 0.02 ± 0.01 | 0.06 ± 0.03 | 1.77 ± 0.83[*,#,†] |
| Total sleep time (min) | 413 ± 8 | 394 ± 6 | 395 ± 9 | 362 ± 14[*] |
| Sleep efficiency (%) | 86 ± 1 | 83 ± 1 | 81 ± 2 | 79 ± 2[*] |
| NREM 1 (%TST) | 9 ± 1 | 9 ± 1 | 12 ± 1 | 13 ± 2[*,#] |
| NREM 2 (%TST) | 49 ± 2 | 50 ± 1 | 44 ± 1[§,‡] | 46 ± 1 |
| NREM 3 (%TST) | 5 ± 0 | 5 ± 0 | 5 ± 1 | 4 ± 0 |
| NREM 4 (%TST) | 20 ± 1 | 19 ± 1 | 22 ± 1 | 20 ± 1 |
| NREM (%TST) | 82 ± 1 | 83 ± 1 | 83 ± 1 | 83 ± 2 |
| REM (%TST) | 18 ± 1 | 17 ± 1 | 17 ± 1 | 17 ± 2 |
| WASO (%TST) | 11 ± 1 | 10 ± 1 | 13 ± 1 | 15 ± 2 |

Values are expressed as mean ± SEM. PS, primary snoring, OSAS, obstructive sleep apnea.
SES, socio-economic status; BMI, Body Mass Index; OAHI, obstructive apnea hypopnea index; RDI, respiratory disturbance index; ArI, Arousal Index; $SpO_2$ nadir (the lowest $O_2$ saturation associated with a respiratory event); NREM, non-rapid eye movement; REM, rapid eye movement; WASO, wake after sleep onset.
[*] $p < 0.05$ MS OSAS vs. control.
[#] $p < 0.05$ MS OSAS vs. PS.
[†] $p < 0.05$ MS OSAS vs. mild.
[‡] $p < 0.05$ mild OSAS vs. control.
[§] $p < 0.05$ mild OSAS vs. PS.
[$] $p < 0.05$ PS vs. control.

Medical Systems, Arnhem, The Netherlands), but the data are not reported here.

Sleep and arousal scoring was performed in 30 s epochs following standard paediatric criteria [42]. A total of five experienced sleep technologists were involved in the sleep staging of all the studies, including controls. Sleep scoring was performed while blinded to the subjects' statuses. Average inter-scorer reliability determined as a quality control measure was 87% and average intra-scorer reliability was 89%. Both cortical (ASDA arousal criteria of a change in EEG frequency lasting $\geqslant$3 s and requiring an EMG increase in REM [43]) and sub-cortical ($\geqslant$2 of: increase in heart rate, increase in EMG, distortion of respiratory effort belts) arousals included were per our current clinical practice. Respiratory events were scored if $\geqslant$2 respiratory cycles in duration and included central apnea (absent flow and effort), obstructive apnea (absent flow with continued respiratory effort associated with $\geqslant$3% $SpO_2$ desaturation and/or arousal), obstructive hypopnea (discernible decrease in flow with continued respiratory effort associated with $\geqslant$3% $SpO_2$ desaturation and/or arousal) and mixed apnea (discernible decrease in flow with period of no respiratory effort and a period of continued respiratory effort associated with $\geqslant$3% $SpO_2$ desaturation and/or arousal) [44]. The respiratory disturbance index (RDI) was defined as the total number of obstructive, central and mixed apneas and hypopneas per hour of total sleep time (TST). The obstructive apnea hypopnea index (OAHI) was defined as the number of obstructive apneas and hypopneas per hour of total sleep time. The $SpO_2$ nadir was defined as the lowest $O_2$ saturation associated with a respiratory event. Diagnostic criteria for the classification of SDB severity followed current clinical practice: primary snoring (PS, OAHI $\leqslant$1 event/h and history of snoring); mild OSAS (OAHI between >1 and 5 events/h); or moderate/severe (MS) OSAS (OAHI >5 events/h). Control children all had an OAHI $\leqslant$1 event/h and no reported snoring either at home or during the PSG study.

Sleep duration for each sleep stage and wake period was calculated as a percentage of the sleep period (SP), defined as the amount of time in minutes from sleep onset until the end of the study, including all periods of wake in between. TST was defined as SP excluding all periods of wake, and sleep efficiency was defined as the ratio of TST to time in bed, or the time the patient was in bed and allowed to sleep.

#### 2.2.1. Cognitive and academic function protocol

Parents were contacted following their child's PSG study and cognitive assessments were carried out within three weeks of the PSG study before commencement of any treatment. The assessments were conducted by trained clinical neuropsychologists blinded to SDB severity on an individual basis in the children's homes to aid in recruitment and to avoid the added stress of being in a hospital environment. Efforts were made to ensure the testing environment was quiet and away from distractions, which was achieved by utilizing a quiet room with the door closed away from background household noise. Parents were also asked to assist in maintaining a quiet assessment environment. All children were assessed on non-school days between 10 am and 4 pm to avoid any affects of fatigue. Tasks were administered in fixed order as listed below.

### 2.3. Cognitive measures

#### 2.3.1. Intellectual ability

The Wechsler Abbreviated Scale of Intelligence (WASI) [45] includes tests of verbal function (Vocabulary and Similarities) and nonverbal function (Block Design and Matrix Reasoning) and provides estimates of Verbal IQ (VIQ), Performance IQ (PIQ), and Full Scale IQ (FSIQ) ($M$ = 100, SD = 15). Raw scores for each subtest were converted into age-scaled $T$-scores ($M$: 50, SD: 15) in accordance with the manual. The two verbal and two nonverbal subtest $T$-scores were combined to generate VIQ and PIQ scores, respectively, and all four subtest $T$-scores were combined to general FSIQ scores.

#### 2.3.2. Academic function

The Wide Range Achievement Test-3 (WRAT-3) [46] was utilized to assess word reading, spelling, and arithmetic skills ($M$ = 100, SD = 15).

#### 2.3.3. Executive skills

(i) *The Rey Complex Figure Test (RCFT)* [47,48], a commonly used clinical tool, was used to measure organizational ability and strategic decision making. Children were instructed to copy a complex geometrical figure as accurately as possible, and RCF-Organizational Strategy Scores were generated. Raw scores were converted to age-standardized $z$-scores using published normative data [49]. (ii) *Verbal fluency*: The Controlled Oral Word Association Test (COWAT) [50] required children to generate as many words as possible beginning with the letters F, A, and S within 1-min timeframes for each letter. They were instructed not to say names of people or places, repeat words, or say numbers. The total number of words generated for each letter was recorded and these values were combined to generate a total score. The total raw score was converted to an age-scaled $z$-score using published normative data [49]. Factor analytic findings have linked the COWAT to attentional control/working memory in both adults and children [51]. It is also thought to involve the executive skills of strategy generation, cognitive flexibility, and self-monitoring [52].

Socio-economic status (SES) was quantified with the Daniel's Scale of Occupational Prestige [53], which yields values ranging from 1 (high) to 6.9 (low) based on parental occupation.

### 2.4. Statistical analysis

Statistical analysis was performed using Statview (Version 5.0, SAS Institute Inc., USA). Data were first tested for normality and equal variance. When test assumptions were satisfied, one-way analysis of variance (ANOVA) with Student–Newman–Keuls post hoc analysis was performed. For demographic and sleep characteristic data that were not normally distributed, Kruskal–Wallis one-way ANOVAs were performed with Dunn's method post hoc analyses. Mean scores for the WASI, WRAT-3, RCFT and COWAT were compared across SDB severity groups using analysis of covariance (ANCOVA) with SES and BMI as covariates. We co-varied for BMI to control for the finding that BMI was higher in the MS group compared with the other three groups. When the interaction term was not significant, only main effects were considered. When test assumptions were satisfied, Pearson correlation coefficients were also calculated between neurobehavioral outcome measures (FSIQ, VIQ, PIQ, Reading, Spelling, Arithmetic, COWAT, and RCFT) and respiratory characteristics (OAHI, RDI, ArI, $SpO_2$ nadir and % of night with $SpO_2$ <90%). For neurobehavioral outcomes and respiratory characteristics that were not normally disturbed, Spearman rank correlations were performed. In addition to comparing group mean scores, which provides an understanding of high-level trends, the percentages of individual children per severity group with scores in the clinically impaired range were compared. Scores above and less than one standard deviation (SD) below the normative mean were classified as "normal" and scores one SD or more below the mean were classified as "impaired." Percentages of impairment for each group were compared with Chi Square or Fisher Exact tests. All tests met the desired power of 0.8. All data are presented as mean ± standard error of the mean (SEM) unless otherwise indicated and significance taken at the $p < 0.05$ level.

## 3. Results

### 3.1. Demographic, sleep, and respiratory characteristics (Table 1)

No significant group differences were found for age. The control group had a significantly higher mean SES ($p < 0.05$) than the PS and MS OSAS groups. The MS group had a significantly higher mean BMI *z*-score ($p < 0.05$) than the other three groups.

As expected, respiratory characteristics during sleep (OAHI, RDI, ArI and $SpO_2$ nadir) differed between the MS OSAS group and the other three groups. In addition, TST was less in the MS OSAS group compared to the control group ($p < 0.05$), but there were no group differences for time in bed or sleep efficiency. No group differences were found in the percentage of time spent in total non-rapid eye movement (NREM) sleep, NREM 3, NREM 4, REM sleep, or wake after sleep onset (WASO). The MS OSAS group spent a significantly greater percentage of time in NREM 1 sleep compared with the control and PS groups ($p < 0.01$ and $p < 0.001$, respectively), and the mild group spent a smaller percentage of time in NREM 2 sleep than the control and PS groups ($p < 0.05$ and $p < 0.01$, respectively).

### 3.2. General intellectual ability

No main or interaction effects of BMI were observed for any of the WASI scores, but interaction effects of SES were observed for all subtest and summary scores. Table 2 outlines each group's mean subtest *T*-scores and VIQ, PIQ, and FSIQ scores. After covarying for SES, the FSIQ scores of all three SDB groups were lower than the control group ($p < 0.001$ for all). The VIQ scores of all three SDB groups were also lower than the control group ($p < 0.001$ for all). Individual subtest analyses revealed significantly reduced scores relative to the control group for both the Vocabulary (PS $p < 0.001$; mild OSAS $p < 0.001$; MS OSAS $p < 0.05$) and Similarities subtests ($p < 0.001$ for all). Nonverbal subtests scores and PIQ did not vary across severity groups.

Percentages of individual children per group with scores in the impaired range were calculated for VIQ, PIQ, and FSIQ (see Fig. 1). No child in the control group had IQ scores in the impaired range. While some group differences were identified, the pattern of these differences was inconsistent and none of the SDB groups had percentages of impairment greater than expected in the normal population (16%).

**Table 2**
Summary of WASI mean standard scores and subtest *T*-scores according to group identity.

| Standard score | Control ($n = 35$) | PS ($n = 59$) | Mild OSAS ($n = 24$) | MS OSAS ($n = 19$) |
|---|---|---|---|---|
| FSIQ | 112.5 ± 2.0 | 101.0 ± 1.9*** | 100.8 ± 2.3*** | 99.9 ± 2.9*** |
| VIQ | 110.0 ± 2.0 | 97.4 ± 1.7*** | 97.0 ± 1.9*** | 99.9 ± 2.9*** |
| Vocabulary | 52.4 ± 1.5 | 45.0 ± 1.2*** | 44.5 ± 1.5*** | 47.5 ± 2.2* |
| Similarities | 59.3 ± 1.1 | 51.3 ± 1.3*** | 51.7 ± 1.3*** | 52.2 ± 1.8*** |
| PIQ | 112.2 ± 2.2 | 104.6 ± 2.3 | 104.8 ± 2.6 | 99.8 ± 3.3 |
| Block Design | 57.8 ± 1.6 | 54.5 ± 1.6 | 54.5 ± 1.3 | 49.3 ± 2.6 |
| Matrix Reasoning | 56.8 ± 1.2 | 50.5 ± 1.4 | 51.3 ± 1.6 | 50.3 ± 2.4 |

FSIQ, Full Scale Intelligence Quotient; VIQ, Verbal Intelligence Quotient; PIQ, Performance Intelligence Quotient.
Values are expressed as mean ± SEM.
SDB vs. control.
* $p < 0.05$.
*** $p < 0.001$.

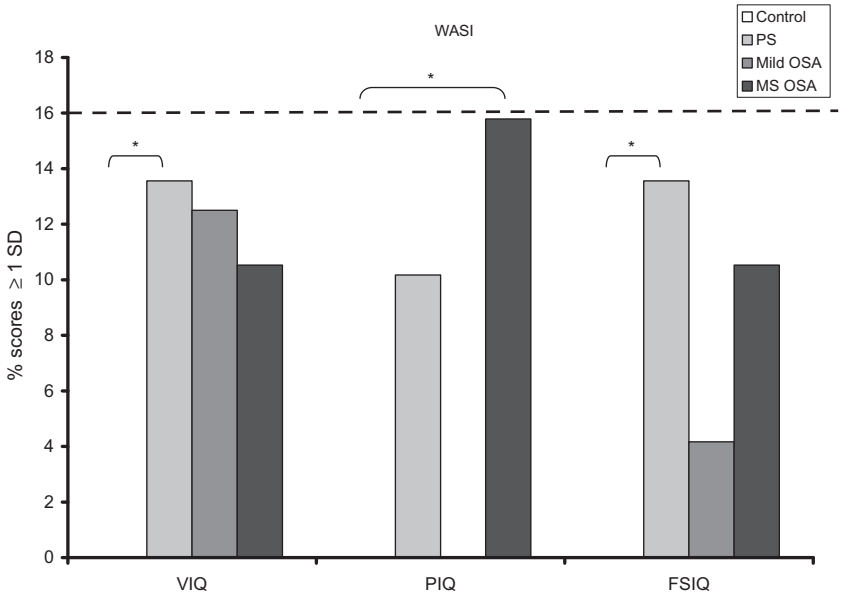


**Fig. 1.** The percentage of children displaying impairments (1 SD or more below the mean) on the WASI. The broken line represents the expected level (16%) of impairment in a normal population. Note that no children in the control group exhibited scores below 1 SD from the mean. Similarly no children in the mild OSA group had scores below 1 SD of the mean for PIQ. *$p < 0.05$.

### 3.3. Academic ability

BMI had no main effect or interaction with any of the WRAT-3 measures although SES did have an interaction with all measures. Mean scores for Reading, Spelling and Arithmetic are provided in Table 3. Although there was a trend for higher performances in the control group compared to all the SDB groups, when covaried for SES this failed to reach statistical significance. There was an overall significant difference in Reading but post hoc analyses failed to identify which groups were implicated.

Fig. 2 illustrates the percentage of individual children in each group who scored one SD or more below the mean. All SDB groups showed a trend for elevated percentages of impairment on measures of academic function compared with both controls and normative data. Differences from the control group achieved statistical significance in the PS group for both Reading and Arithmetic ($p < 0.05$ for both) and in the MS OSAS group for Arithmetic ($p < 0.01$).

### 3.4. Executive skills

BMI had no main effect or interaction with any of the measures of executive skill, but SES had an interaction with RCFT scores. No significant group differences were found for mean COWAT or RCFT *z*-scores, with all groups scoring within one-third of a standard deviation of the mean on both tests (see Table 4). Percentages of impairment in each group for both tests are illustrated in Fig. 3. There was a non-significant trend for more severe SDB groups to have higher rates of impairment on the COWAT. On the RCFT, only the PS group had a higher percentage of children with scores in the impaired range (33%) than expected in the normal population.

### 3.5. Associations between respiratory and neurobehavioral variables

Pearson correlation coefficients were calculated between neurobehavioral outcome measures (FSIQ, VIQ, PIQ, Reading, Spelling, Arithmetic, COWAT, and RCFT) and respiratory characteristics (OAHI, RDI, ArI, $SpO_2$ nadir and % of night with $SpO_2$ <90%). No significant relationships were identified between any of these variables.

## 4. Discussion

The current study identified several neurocognitive deficits in children with SDB compared with normal controls. These effects, however, were not related to severity of SDB. Further, respiratory characteristics, including measures of hypoxia and disruption to sleep were not significantly correlated with any cognitive or academic measures. The prediction that mild levels of SDB would be associated with deficits in neurocognitive function was supported with all severity groups showing reduced mean VIQ and FSIQ scores relative to the control group and more children in the PS group receiving impaired scores on measures of academic function than in the control group.

### 4.1. Intellectual ability

Reductions in FSIQ and VIQ were apparent in children with SDB relative to healthy controls. Using the same abbreviated measure of intellectual function, Blunden et al. [32] also found reduced FSIQ and VIQ in a small group ($n = 11$) of children who snored relative to a control group, but found no reduction in PIQ. In a larger study, O'Brien and colleagues also found specific reductions in general

**Table 3**
Summary of WRAT-3 standard scores according to group identity.

| Standard score | Control ($n = 35$) | PS ($n = 59$) | Mild OSAS ($n = 23$) | MS OSAS ($n = 19$) |
|---|---|---|---|---|
| Reading | 107.5 ± 2.3 | 97.3 ± 2.0 | 99.8 ± 2.5 | 99.1 ± 3.2 |
| Spelling | 107.1 ± 2.6 | 100.1 ± 1.9 | 99.2 ± 2.6 | 99.2 ± 3.7 |
| Arithmetic | 99.5 ± 1.8 | 92.8 ± 2.2 | 92.6 ± 2.5 | 91.0 ± 4.2 |

Values are expressed as mean ± SEM. Note that there were no significant differences observed between groups for any sub-score of the WRAT-3.

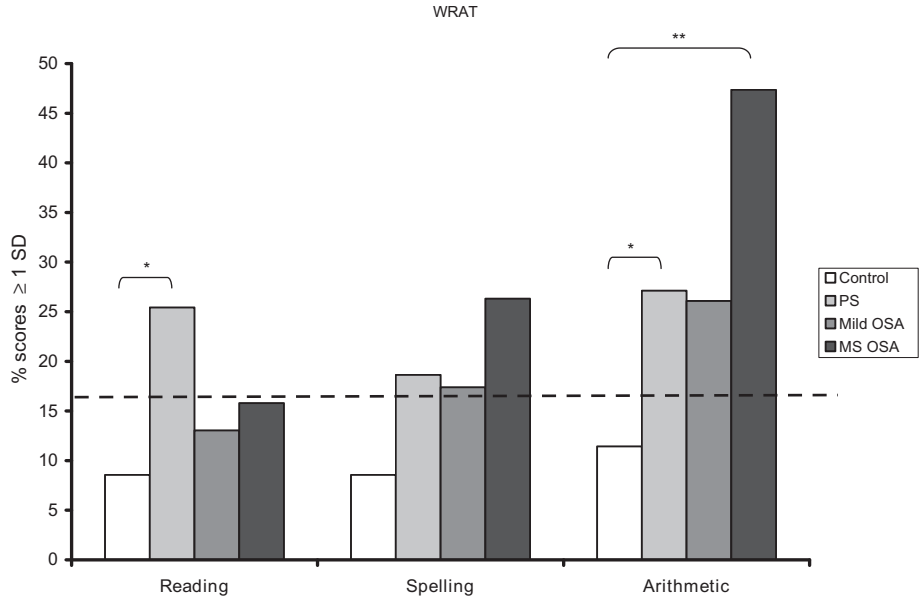


**Fig. 2.** The percentage of children who displayed impairments (1 SD or more below the mean) on the WRAT-3. The broken line represents the expected level (16%) of impairment in a normal population. $^{*}p < 0.05$; $^{**}p < 0.001$.

**Table 4**
Summary of COWAT and RCFT *z*-scores according to group identity.

| *z*-Score | Control ($n = 35$) | PS ($n = 54$) | Mild OSAS ($n = 22$) | MS OSAS ($n = 18$) |
|---|---|---|---|---|
| COWAT | −0.01 ± 0.17 | −0.32 ± 0.13 | −0.33 ± 0.22 | 0.04 ± 0.37 |
| RCFT | −0.02 ± 0.14 | −0.31 ± 0.16 | 0.35 ± 0.23 | −0.06 ± 0.27 |

Values are expressed as mean ± SEM. Note that there were no significant differences observed between groups for either the COWAT or RCFT.

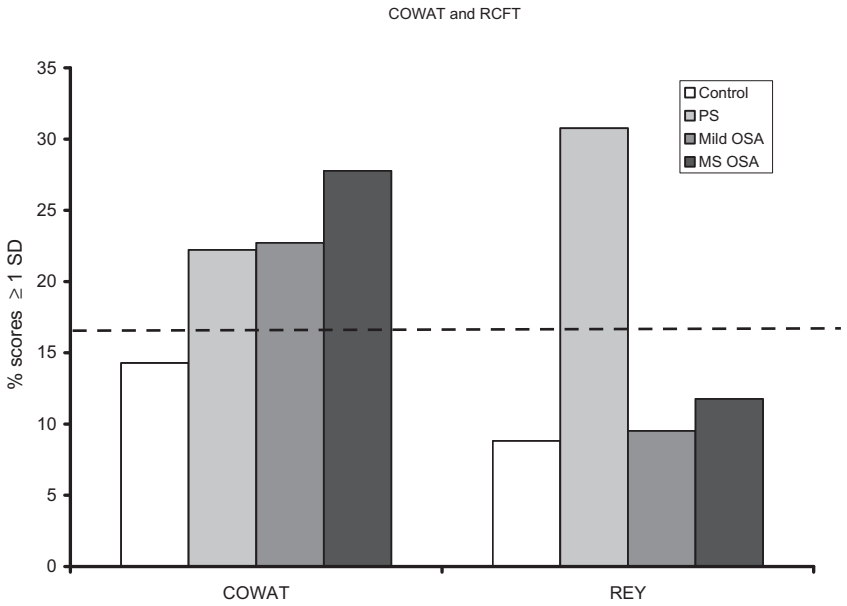


**Fig. 3.** The percentage of children who displayed impairments (1 SD or more below the mean) on the COWAT and RCFT. The broken line represents the expected level (16%) of impairment in a normal population.

verbal abilities in children with a high degree of sleep fragmentation compared to children with low sleep fragmentation [54]. While scores in the SDB groups of the current study were reduced relative to controls, group means were well within the average range. The high average performance of the control group may be partially explained by the reported tendency for school-aged Australian children to perform slightly above the mean on tests of intellectual ability with American standardization samples [55]. It is also consistent with Beebe's recent observation [56] that most IQ differences in the literature have been driven by control groups with above-average intellect.

### 4.2. Academic function

Significant group differences were not identified on measures of Reading, Spelling or Arithmetic. SDB group means approximated the population mean (standard score of 100) for all academic measures, though elevated rates of individual children with impaired scores were identified in two SDB groups for Arithmetic and in the MS OSAS group for Spelling. The failure to find group differences using mean standard scores of academic ability has been documented previously in research comparing children with SDB and controls [19,57,58]. This pattern suggests that the functional risks conveyed by SDB may not be universal but may instead interact with other (currently unidentified) characteristics unique to individual children.

### 4.3. Executive abilities

No group differences were evident for RCFT or COWAT performances, though there was a trend for lower mean scores to be associated with more severe SDB. Analysis of impairments rates identified no significant group differences. There was a trend for more severe SDB to be associated with higher rates of impaired verbal fluency on the COWAT, which may support the VIQ findings. In support of our finding other studies have also found no differences between severity groups [59,60]. In contrast, other studies have reported a range of deficits on measures of executive functioning [17,18,55]. These conflicting findings may reflect the use of different executive function measures across studies. Future research addressing this domain will need to incorporate more comprehensive protocols for tapping "dynamic" neurobehavioral skills, including attention and processing speed.

### 4.4. Clinical and theoretical implications

A dose-dependent relationship between the level of hypoxia (as reflected in OAHI) and the subsequent severity of cognitive impairment has been reported in earlier studies [26,27]; however, more recent studies [56] and our own have not found this strong association. Furthermore, we previously demonstrated that sleep architecture is not significantly disrupted in this group of children regardless of SDB severity using spectral analysis of the EEG [41]. Therefore, the current study provides further evidence that mechanisms other than hypoxic brain damage and sleep disruption are important in reducing cognitive and academic function in these children.

Our results have important clinical implications for the identification and treatment of children with SDB. As the prevalence of sleep disordered breathing is high, with reports of up to 34.5% of children snoring often or always [2], it is important to identify and treat children experiencing any severity of PSG-defined SDB,

which may affect cognition and academic function. The current study highlights individual vulnerability to SDB – regardless of severity – and the need to examine each child as a unique case rather than basing treatment decisions solely on the severity of SDB. Treatment approaches may be best informed by measures of SDB taken together with measures of cognition and behavior.

### 4.5. Limitations and future directions

Generalization of these results should be viewed in the context of several study limitations. First, the current findings may have been affected by a bias in the participant sample. Parents of children who only report snoring (PS group) may bring their children for sleep assessment due to existing cognitive and academic difficulties. Our sample was derived from a tertiary pediatric centre, potentially over representing children whose parents were concerned about their child's functioning. It has been suggested that parents attend sleep clinics with children who have behavior problems more frequently than do parents with children with identical sleep problems in the community who do not have behavior problems [56]. Therefore, it may be that the current sample of children with SDB represents a subset of children who snore and who also have behavioral problems, which may be associated with cognitive or academic difficulties.

Scores on measures of intellectual ability and academic ability were generally higher in the control group than would be expected in a normal population. This may be an artifact of using American tests to assess Australian children. Alternatively, it is possible that the control children were particularly high functioning and not representative of the general population. Regardless, our findings' validity is supported – our results remained significant after covarying for SES, which has a known association with intellectual function. We acknowledge that the use of our measure of SES, based on parental occupation, may have limitations, and that other factors such as genetic predisposition, parental education, income and area of residence may also impact cognitive and academic performances in children, but Australian society tends to be more homogeneous in terms of these factors than in some other countries.

Research is needed to further explore underlying mechanisms linking mild forms of SDB to cognitive and academic deficits. Methods combining routine neuropsychological assessments of cognition and academic function together with overnight PSG should be developed to assist in identifying the subgroup of PS children who present with cognitive and academic problems and need prompt treatment. Improved measures of assessing sleep disruption such as spectral EEG analysis and sub-cortical arousal may also be useful.

## 5. Conclusions

Overall, results of the present study showed several cognitive and academic deficits in children with SDB, and these deficits were not significantly related to measures of hypoxia or sleep disruption. Children with PS were also found to have an increased incidence of impairment on measures of academic function relative to controls. This study provides further evidence that children with even mild forms of SDB are at risk of cognitive and academic deficits, highlighting individual vulnerability to SDB – regardless of severity – and the need to examine each child as a unique case rather than basing treatment decisions solely on the severity of SDB. Potential mechanisms for these problems in PS and across the SDB spectrum require further investigation.

## Conflict of interest

The ICMJE Uniform Disclosure Form for Potential Conflicts of Interest associated with this article can be viewed by clicking on the following link: doi:10.1016/j.sleep.2010.11.010.

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