Original Article

# Sleep architecture in school-aged children with primary snoring

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## ARTICLE INFO

## ABSTRACT

*Objective:* We aimed to examine if sleep architecture was altered in school-aged children with primary snoring (PS).
*Methods:* Children ages 6 to 13 years from 13 primary schools were randomly recruited. A validated obstructive sleep apnea (OSA) screening questionnaire was completed by their parents. Children at high risk for OSA and a randomly chosen low-risk group were invited to undergo overnight polysomnography (PSG) and clinical examination. Participants were classified into healthy controls, PS, mild OSA, and moderate to severe OSA (MS OSA) groups for comparison.
*Results:* A total of 619 participants underwent PSG (mean age, 10.0 ± 1.8 years; 396 (64.0%) boys; 524 (84.7%) prepubertal). For the cohort as a whole, there were no significant differences in measures of sleep architecture between PS and nonsnoring healthy controls. In the multiple regression model, percentage of nonrapid eye movement (NREM) stage 1 (N1) sleep had a significantly positive association, whereas percentage of slow-wave sleep (SWS) had a significantly negative association with sleep-disordered breathing (SDB) severity after controlling for age, gender, body mass index (BMI) *z* score, and pubertal status. In prepubertal children with PS, no significant disruption of sleep architecture was found. However, pubertal adolescent PS participants had significantly higher adjusted percentage of N1 sleep and wake after sleep onset (WASO) compared to healthy controls.
*Conclusions:* PS did not exert significant adverse influences on normal sleep architecture in prepubertal school-aged children. Nevertheless, pubertal adolescents with PS had increased N1 sleep and WASO.

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## 1. Introduction

Sleep architecture represents the cyclical pattern of sleep as it shifts between the different sleep stages, including nonrapid eye movement (NREM) and rapid eye movement (REM) sleep. Early in the night, one transitions from lighter sleep stages (NREM stage 1 sleep [N1]) to deeper stages (slow-wave sleep [SWS]), with REM sleep more often appearing during the latter part of the night [1]. Sleep architecture allows us to produce a picture of what an overnight sleep looks like, considering various depths of sleep and arousals to wakefulness. In children, it has been demonstrated that sleep efficiency, percentage of SWS, and REM sleep decreases and percentage of lighter NREM stage 2 (N2) sleep and Tanner stage increases with age [2–4].

Sleep-disordered breathing (SDB) consists of a spectrum of diseases, with severity ranging from primary snoring (PS), upper airway resistance syndrome (UARS), and obstructive sleep apnea (OSA) [5,6]. OSA in school-aged children is associated with both neurocognitive dysfunction and behavioral problems [7,8]. Apart from intermittent hypoxia, sleep fragmentation may account for the demonstrated neurocognitive impairment. Disturbance of normal sleep architecture is one form of sleep fragmentation. Recent scientific research has shown that children with OSA were more likely to have disturbed sleep architecture compared to children with PS or healthy controls, including decreased percentage of SWS [9–11] and REM sleep [11–13], as well as increased percentage of N1 sleep [9,12,14,15] and REM latency [10,15]. Furthermore, these changes were reversible following treatment of OSA [16–18].

PS is defined as snoring in the absence of apnea or hypopnea during sleep [19]. It is positioned at the milder end of the SDB spectrum, and several studies demonstrated that even PS also might exhibit neurobehavioral impairments and cardiovascular morbidities, though the mechanism is unclear [10,20–25]. Snoring is an upper airway breathing sound or noise caused by partial or complete occlusion of the upper airway [26]. Little attention has been

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given to determine if it will disturb normal sleep architecture. The majority of the few published studies have failed to reveal any significant differences in sleep architecture between primary snorers and healthy control participants [10,14,27,28]. Miano et al. [9] found that primary snorers had a higher percentage of N1 sleep and Yang et al. [15] demonstrated increased sleep latency in participants with PS. However, these studies had small sample sizes. Additionally, the SDB groups in the previous studies were recruited from clinics; hence more severe cases may be included and selection bias may exist.

In our study, we aimed to investigate the sleep architecture of school-aged children with PS recruited from the community. We hypothesized that the percentage of certain sleep stage or wakefulness after sleep onset (WASO) in primary snorers would be different from that in nonsnoring healthy controls.

## 2. Methods

### 2.1. Participants

Our study was part of our previous research which aimed to investigate the prevalence of OSA in a community-based sample of school-aged children [29]. In brief, participants between the ages of 6 and 13 years were recruited from 13 randomly selected schools. Parents were asked to complete a validated OSA screening questionnaire [30] that stratified children into high or low risk for OSA. All high risk and a selected sample from the low risk group based on a computer-generated random number were invited to undergo overnight polysomnography (PSG) and clinical examination. Children were excluded if they had an intercurrent illness within 4 weeks of PSG; cardiac, renal, or neuromuscular diseases; chromosomal abnormalities; medication which could affect sleep or respiration within 4 weeks of PSG; or previous upper airway surgery. Written informed consent and assent were obtained from the parents and participants, respectively. Approval by the Joint Chinese University of Hong Kong-New Territories East Cluster Clinical Research Ethics Committee was obtained.

### 2.2. PSG

All recruited children underwent standard overnight PSG at a dedicated sleep laboratory with CNS 1000P polygraph (CNS Inc., Chanhassen, Minnesota, USA). In brief, electroencephalogram (EEG) from four leads (C3/A2, C4/A1, O1/A2, and O2/A1), bilateral electrooculogram, submental electromyogram, bilateral leg electromyogram, and electrocardiogram were recorded. The positions of the participant, respiratory airflow (nasal cannula connected to pressure transducer), thoracic and abdominal respiratory efforts (piezo-based effort belts), arterial oxyhemoglobin saturation (oxygen saturation [SpO2] by Ohmeda 3700 pulse oximeter, Boulder, CO, USA), and snoring sound (microphone) were measured. All data were scored by four PSG technologists and the reports were reviewed and finalized by a chief technologist.

Sleep architecture was scored in 30-s epochs according to the criteria outlined by Rechtschaffen and Kales [31]. The following parameters of sleep architecture were measured: total sleep time (TST), defined as time from sleep onset to the end of the final sleep minus wakefulness after sleep onset; sleep latency, defined as the time occurred from lights out to the first epoch of any sleep; the percentage of each sleep stage out of TST (stage 1, 2, SWS [NREM sleep stages 3 and stage 4], and REM sleep); and WASO, defined as the time spent awake during sleep period time (time from sleep onset to the end of final sleep).

Respiratory events and arousals were scored according to standardized criteria [32]. An obstructive apnea was defined as the absence of airflow with persistent respiratory effort lasting longer than two baseline breaths, irrespective of arterial oxygen saturation changes. An obstructive hypopnea was defined as a reduction of 50% or more in the amplitude of the airflow signal with persistent respiratory effort. It was only quantified if it was longer than two baseline breaths and was associated with oxygen desaturation of at least 4% or arousals. Arousal was defined as an abrupt shift in EEG frequency during sleep, which may include theta, alpha, or frequencies greater than 16 Hz but not spindles of 3 to 15 s in duration. In REM sleep, arousals are only scored when accompanied by concurrent increases in submental electromyogram amplitude. Obstructive apnea–hypopnea index (OAHI) was defined as the total number of obstructive apneic and hypopneic episodes per hour of sleep. Oxygen desaturation index was defined as the total number of dips in arterial oxygen saturation >3% per hour of sleep. The $SpO_2$ nadir also was noted. Arousal index (ArI) was defined as the total number of arousals per hour of sleep. Participants were classified into four groups according to the PSG and questionnaire results: group 1 was the healthy control group (OAHI < 1 and history of snoring <3 nights per week); group 2 was the PS group (OAHI < 1 and history of snoring $\geqslant$3 nights per week); group 3 was the mild OSA group (OAHI 1–5); and group 4 was the moderate to severe OSA (MS OSA) group (OAHI $\geqslant$ 5).

### 2.3. Anthropometry assessment

The weight, height, and Tanner stage of all participants were assessed on the day of PSG. Body mass index (BMI) was calculated as weight/height$^2$ (kg/m$^2$). Weight, height, and BMI were converted to z scores appropriate for age and gender, according to local reference [33]. Pubertal stage was evaluated using a self-assessment questionnaire to categorize Tanner stages [34]. Prepubertal was defined as Tanner stage 1 and pubertal defined as Tanner stage 2 or higher.

### 2.4. Statistical analyses

All statistical analyses were performed using SPSS 16.0 (SPSS Inc., Chicago, Illinois) and a P value <.05 was considered statistically significant. The participants were divided into four groups (healthy controls, PS, mild OSA, and MS OSA) for comparison. The mean (standard deviation [SD]), median (interquartile range), and percentage were presented for parametric, nonparametric, and categorical data, respectively. Parametric with equal variances data were compared using 1-way analysis of variance and the Bonferroni correction was used for post hoc pairwise comparisons (P < .05 was statistically significant). Nonparametric and parametric data without equal variances were compared, and the Kruskal–Wallis test and Mann–Whitney tests with adjusted P values (significant at P < .0083) were used for pairwise comparisons. The $\chi^2$ test was used to assess the differences in proportion between the four groups.

Multiple linear regression analysis was used to assess the relationship between SDB category (control, PS, mild OSA, and MS OSA) and sleep architecture outcomes, controlling for age, gender, BMI z score, and puberty status. SDB category, gender, and puberty status were converted to dummy variables (0 for controls, 1 for PS, 2 for mild OSA, and 3 for MS OSA; 0 for girls and 1 for boys; 0 for prepubertal and 1 for pubertal). Residual analysis was performed for each regression model to test the validity of model assumptions.

Analysis of covariance models were used to further examine differences in sleep architecture variables among control, PS, and OSA groups after separately controlling for age, gender, and BMI z score in prepubertal and pubertal children. Nonparametric variables were normalized using logarithmic transformation.