Sleep Health 000 (2022) 1–7

Contents lists available at ScienceDirect

Sleep Health

Journal of the National Sleep Foundation

journal homepage: sleephealthjournal.org

Comparison of polysomnographic characteristics between low birthweight and normal birthweight children in the Northern Territory of Australia: A case-control study

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ARTICLE INFO

Article History:
Received 24 November 2021
Revised 28 June 2022
Accepted 12 July 2022
Available online xxx

Keywords:
Developmental origins of health
Small for gestational age
Obstructive sleep apnea
Polysomnography
Sleep studies

ABSTRACT

Objectives: To describe the sleep architecture of pediatric patients according to whether they were born low birthweight (birthweight <2500 g, LBW) or normal birthweight (birthweight >2500 g).
Design: Case control study.
Setting: Pediatric sleep laboratory in the Northern Territory of Australia during a 5-year study period (2015- 2020).
Participants: Pediatric patients (aged <18 years) referred to the specialist sleep service for assessment of clinically suspected sleep disorders.
Measurements: Sleep onset latency, rapid eye movement (REM) sleep latency, wake time after sleep onset, total sleep time, sleep efficiency, non-rapid eye movement stages N1/N2/N3, and REM sleep duration, total/spontaneous/respiratory/limb related arousal indexes, total/non-rapid eye movement/REM obstructive apnea-hypopnea index and oxygen saturation.
Results: One hundred and seventy-two pediatric patients had birthweight data available of whom 19 were LBW. LBW patients showed significantly greater sleep disruption and higher prevalence of poor sleepers (<80% efficiency). In multivariate regression models, increasing birthweight was associated with significantly greater sleep efficiency and total sleep time. After accounting for gestational age LBW was associated with increased odds of obstructive sleep apnea.
Conclusions: Among pediatric patients LBW is associated with increased sleep disruption and reduced sleep efficiency. This is attenuated by gestational age, though both gestational age and LBW significantly influence odds of obstructive sleep apnea. This sleep health deficit may contribute to development of chronic disease in this vulnerable population, and should be monitored to provide avenues for early intervention.

Introduction

Individuals born low birthweight ((LBW) birthweight <2500 g), are known to be at a heightened risk for numerous chronic diseases throughout their lifespan. Through childhood this manifests as social based difficulties including altered attention and hyperactivity,[1] reduced cognitive ability and motor control,[2] progressing to an increased prevalence of mental health disorders in adolescence and adulthood[1,3], reduced body stature and physical capacity,[3-6] impaired lung function,[7] altered body habitus,[8,9] and increased prevalence of hypertension and diabetes.[10,11] Some of these aspects, notably those related to cognition are compounded by the high prevalence of preterm births (<37 weeks gestation) in this cohort,[1] while other aspects present regardless of gestational age.[8] The impact of early childhood

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https://doi.org/10.1016/j.sleh.2022.07.003

lifestyle factors for this at risk population are starting to be studied in order to explore early intervention opportunities. Multiple studies have reported on the effects of gestational length on sleep outcomes.[12-15] Prematurity has been associated with a heightened prevalence of obstructive sleep apnea (OSA) in pediatric populations, occurring at twice the rate in ex-preterm infants.[15] Yiallourou et al. reported reduced total sleep time, non-rapid eye movement (NREM) sleep time and sleep efficiency, and heightened wakening after sleep onset (WASO) in preterm appropriate for gestational age (AGA) children compared to both term AGA and preterm fetal growth restricted children (children tested between the ages of 5 & 12).[14] Yet, preterm fetal growth restricted children had a higher NREM 2 percentage compared to both term and preterm AGA.[14] As such, there appears to be differential effects of birthweight in conjunction with gestation on sleep architecture and function. Notably however, the few studies reporting on birthweight utilized inconsistent definitions including small for gestational age (SGA),[16] intrauterine growth restricted, very low birthweight ((VLBW) birthweight <1500 g)[17] or extremely low birthweight ((ELBW) birthweight <1000 g).[18]

Gestational age is noted to have significant effects upon sleep architecture across the range of preterm births.[12,14] These effects differ dependent upon the length of gestational maturity, which is due to the timing of the development of a recognizable sleep cycle in the fetus. The fetal sleep cycle, as much as can be determined, begins to consolidate between 25 and 30 weeks gestation, with a 4 stage cycle beginning to appear around 32 weeks.[12] Thus, when a baby is delivered prematurely, the natural development of this sleep cycle is interrupted.

Aside from neural development, the mechanics of how intrauterine growth may influence sleep outcomes are less clear. Self or parent/caregiver reports have identified a significantly increased frequency of sleep disordered breathing among young adults born VLBW.[17] Hysing et al. (2019) reported varied sleep outcomes according to neonatal risk categorization with increased nighttime awakenings at 18 months of age for ELBW and SGA, decreased sleep duration for ELBW at 6 months, and decreased sleep duration for SGA at 6 and 18 months.[16] Other reports have shown increased odds of low sleep efficiency and decreased total sleep time with reduced birthweight.[13,19] Yet, an earlier study which linked birth records to polysomnographic (PSG) diagnosis of sleep apnea for a large sample population found no significant effect of SGA on early childhood (<6 years of age) sleep apnea.[20]

Sleep health is strongly associated with both current and future mental and physical health aspects.[21] Impaired sleep health among children and adolescents has shown significant correlation with prevalence of future mental health disorders in early adulthood.[18] In adulthood, presence of OSA is strongly correlated with hypertension, heart disease and metabolic syndrome.[22] Among the LBW population there is already a significantly heightened risk for these outcomes.[1,10] It is plausible that impaired sleep health may contribute to this risk disparity or provide an additionally heightened risk for LBW individuals. Therefore, early investigation of and intervention for sleep health has the potential for significant lasting effects across a range of health domains.

It is apparent that there is a dearth of evidence in the literature for an effect of birthweight on sleep outcomes. Limited studies to date have reported in detail on the range of PSG outcomes among this at-risk cohort. This study sets out to describe PSG data among LBW children (aged <18 years) and compare patterns of sleep architecture to a matched cohort of normal birthweight (NBW) children.

Methods

Setting and study participants

This study was conducted at the Top End Health Service region of the Northern Territory of Australia. All pediatric patients aged <18 years who underwent a diagnostic PSG during a 5-year study period (2015-2020) were included for analysis. Indigenous and non-Indigenous patients residing in the Top End Health Service region were referred to the specialist sleep service by general practitioners, pediatricians and otorhinolaryngologists for assessment of clinically suspected sleep disorders. Patients underwent a diagnostic PSG as per the discretion of the treating pediatric sleep specialist following an initial consultation. The PSG's were performed at the Darwin based sleep service facility, Darwin Respiratory and Sleep Health, Darwin Private Hospital.

Ethical consideration

Individual consent from the study participants was not obtained, as the study was retrospective in nature with data collected during the normal course of clinical activity and no active interventions were investigated in this study. This study was approved by the Human Research Ethics Committee of the Northern Territory Health Service and Menzies School of Health Research. (Reference no: HREC 2019-3434).

Clinical data

As per standard protocol at the Darwin Respiratory and Sleep Health sleep diagnostic facility, all patients were administered with a detailed questionnaire by the sleep technologist prior to undergoing a diagnostic PSG. Parents assisted with questionnaires when required. The questionnaire provided information on demographics, self-reported Indigenous status, age, sex and any significant co-morbid conditions. Medical records were also reviewed to corroborate significant medical comorbidities. Living location was categorized by the Australian Statistical Geography Standard (ASGS), a measure of relative access to services for a population in a defined area and classified as either Outer Regional (ASGS 3), Remote (ASGS 4), or Very Remote (ASGS 5) (Fig. 1).[23] To assess subjective day time sleepiness, the Pediatric Daytime Sleepiness Scale (PDSS) was utilized. Anthropometric measurements including height, weight, and body mass index (BMI) were recorded. BMI was classified as underweight, normal weight, overweight or obese according to the Australian standards for age and sex centile chart.[24] Birthweight and gestational age were retrospectively collected through medical record linkage. Due to the high prevalence of interstate and/or remote births in our cohort, birth information (weight and gestational age) was often not entered directly into the local healthcare system and relied on forwarded clinical information from previous care providers. LBW was defined as birthweight <2500 g and NBW as birthweight ≥2500 g, prematurity was defined as gestational age <37 weeks and SGA as a birthweight at or below the 10th centile for that gestational age according to time period matched norms.[25]

Polysomnography data

All PSGs were performed by registered PSG technologists according to Australasian sleep association recommended standards.[26] The sleep studies were analyzed in accordance with the American Academy of Sleep Medicine recommendations by a senior pediatric sleep scientist.[26] The PSG data extracted for this study included: sleep onset latency, rapid eye movement (REM) sleep latency, WASO, total sleep time, sleep efficiency, NREM stages N1/N2/N3, and REM sleep duration. Poor sleepers were defined in 2 stages: Poor Sleep I (sleep efficiency <90%) & Poor Sleep II (<80%). Other data included for analysis were total/spontaneous/respiratory/limb related arousal indexes, total/NREM/REM obstructive apnea-hypopnea index (OAHI), baseline oxygen saturations in NREM and REM sleep and desaturations including saturation nadir and time and percentage of total sleep time spent below 90%, 85%, & 80% oxygen saturation (SpO_2). OSA was classified

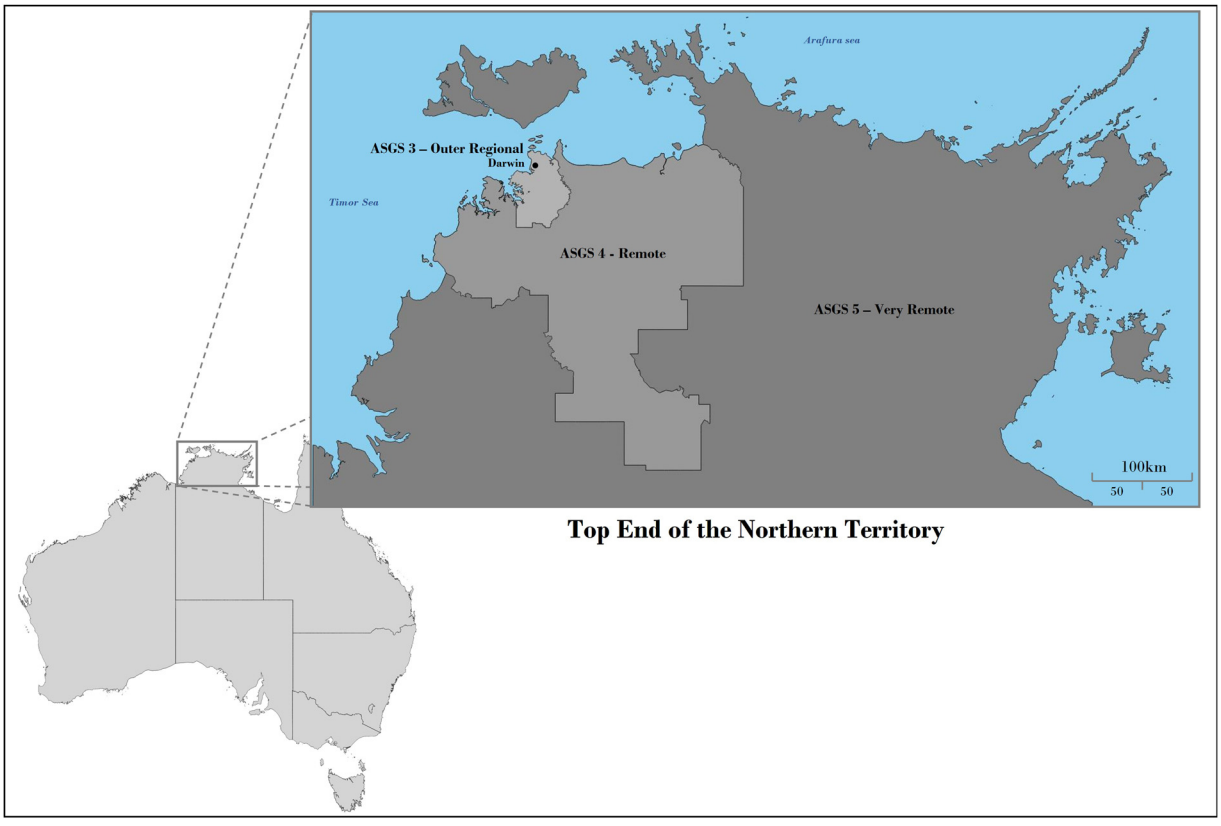

Fig. 1. Map of the top end region of the Northern Territory showing the extent of ASGS zones. **Abbreviation**: ASGS, Australian statistical geographic standard.

using the obstructive apnea hypopnea index (OAHI) as normal (OAHI <1), mild (OAHI 1 < 5), moderate (OAHI 5 < 10), or severe (OAHI ≥10). Studies in which the patient was intolerant to the monitoring device and reported significantly affected sleep, and/or removed the device at some point through the study were considered failed and excluded from the analysis.

Statistical methods

Continuous parameters were initially analyzed for normality via the Shapiro Wilks distribution test and all bar 2 parameters (total sleep time, REM sleep time) were found to have non-parametric distribution ($p < .01$) thus reported as medians (interquartile ranges) while categorical variables were reported as numbers (percentages). Demographic and clinical parameters were compared between LBW and NBW using Wilcoxon rank-sum test for continuous parameters, and 2-tailed proportions z-test for categorical parameters. Differences in distribution of total sleep time, WASO, sleep efficiency and total OAHI were graphically displayed by kernel density graphs utilizing Epanechnikov kernels and a bandwidth of 5. Kernel density graphs display data distribution (measured as density on the y-axis) of a continuous variables' potential scores (labelled on the x-axis). Multivariate 50th quantile linear regression adjusting for age, sex, indigenous status and BMI was utilized to describe the effect of a 100 g increase on birthweight on selected PSG parameters (WASO, sleep efficiency, total sleep time, SpO_2 nadir, total OAHI), reporting beta coefficients, and 95% confidence intervals (CI's). Multivariate logistic regression adjusting for age, sex, indigenous status, and BMI was utilized to describe the effect of a 100 g increase in birthweight on presence of OSA. A separate model for both quantile linear regressions and logistic regressions was run utilizing gestation as an additional confounder and reported the effect of both a 100 g increase in birthweight and a 1 week increase in gestational age. Alpha was set to $p = .05$ throughout and all analysis was conducted in STATA IC 15.1 (StataCorp, TX).

Results

Study participants

A total of 710 patients were identified to have undergone a diagnostic PSG during the study period (2015- 2020). Of these, 178 (25%) had birthweight data available, including 51 (30%) Indigenous children, of which 6 (3%) had failed studies, giving a total 172 patients available for analysis. The majority of patients were male (63%), non-Indigenous Australians (70%), lived in regional areas (ASGS level 3) (84%) with a median age of 5.4 years (interquartile range 3.2, 8.5). Nineteen LBW patients were identified, of whom the majority were born prematurely (82%) and 3 were SGA (27%). Aside from differences in birth variables, the LBW cohort was older and had a significantly higher frequency of obesity (33% vs. 14%, $p = .034$) (Table 1).

Polysomnography data

Polysomnography findings were compared between LBW and NBW patients (Table 2). Sleep latency and sleep architecture variables (N1-N3 percent and REM latency and percent) did not significantly differ between cohorts. LBW patients had significantly more WASO ($p = .032$) and reduced sleep efficiency ($p = .002$), with the majority of LBW patients having a sleep efficiency below 80%. Fig. 2 highlights the overlap in distribution of results for sleep and REM latency between LBW and NBW patients, while also showing the shifted distribution of WASO and sleep efficiency. In the overall cohort total OAHI did not significantly differ between LBW and NBW patients. Though REM OAHI and OSA prevalence was heightened among LBW patients this did not reach statistical significance. A greater

Table 1
Clinical characteristics of patients split by birthweight status

Clinical parameters		NBW (n = 153)	LBW (n = 19)	*p*-value
Age (y)		5.1 (3.1, 8)	7.2 (4.2, 10)	.106
Sex (male)		96 (63%)	12 (63%)	.972
Birthweight (grams)		3400 (3080, 3720)	2300 (1713, 2410)	-
Gestation (wk)^		39 (37.4, 40.3)	34.3 (29.9, 36.4)	<.001*
Preterm (wk < 37)^		5 (16%)	9 (82%)	<.001*
SGA^		3 (9%)	3 (27%)	.140
Indigenous Australians		43 (29%)	8 (42%)	.237
Outer regional (ASGS level 3)		129 (84%)	15 (79%)	.550
Remote (ASGS 4 or 5)		24 (16%)	4 (21%)	.550
BMI	Underweight	23 (15%)	0 (0%)	.075
	Normal weight	91 (59%)	9 (50%)	.402
	Overweight	16 (11%)	3 (17%)	.441
	Obese	21 (14%)	6 (33%)	.034*
PDSS		14(9, 17)	13 (7, 18.5)	.985
Any comorbidity		17 (11%)	4 (21%)	.212

Abbreviations: LBW, low birthweight; NBW, normal birthweight; SGA, small for gestational age; ASGS, Australian statistical geography standard - remoteness area; BMI, body mass index; PDSS, pediatric daytime sleepiness scale; IQR, interquartile range.
Data displayed as median (IQR) for continuous parameters and n (%) for categorical parameters.
p-value derived from Wilcoxon rank-sum test for continuous parameters and 2 tailed proportions z-test for categorical parameters.
^ Data available for 32 NBW and 11 LBW patients.
* *p*-value significant at <.05.

Table 2
Polysomnography results split by birthweight status

PSG parameters	NBW (n = 153)	LBW (n = 19)	*p*-value
Sleep latency (min)	0 (0, 25.5)	9.6 (0, 30)	.418
REM latency (min)	168 (126, 219.5)	177 (131, 222.5)	.664
Wake after sleep onset (min)	63.2(36, 107)	125 (54.4, 164)	.032*
Sleep efficiency (%)	84.2 (79.3, 90.1)	77.6 (72.6, 83.2)	.002*
Poor sleep I (<90%)	114 (75%)	18 (95%)	.049*
Poor sleep II (<80%)	44 (29%)	11 (58%)	.010*
Total sleep time (min)	495.1 (436.5, 549.5)	470.8 (391.5, 501.7)	.020*
N1 sleep (%)	0.9 (0.5, 1.4)	0.9 (0.4, 1.9)	.864
N2 sleep (%)	45 (39.3, 54.2)	50.6 (37.9, 55.1)	.344
N3 sleep (%)	29.5 (24.7, 34.7)	29.9 (24.9, 34.3)	.860
REM sleep (%)	22.1(19, 27)	19.9 (15.5, 23.4)	.107
Total arousal index (arousals/h of sleep)	6.9 (5.3, 8.4)	6.5 (4.5, 8.6)	.899
Limb related arousal index	0 (0, 0)	0 (0, 0)	.450
Respiratory arousal index	0.4 (0.1, 1.6)	0.7 (0.1, 2.1)	.450
Spontaneous arousal index	5.8 (4.3, 7)	4.9 (3.9, 6.4)	.193
Total OAHI (/h of sleep)	0.8 (0.3, 3.3)	1.4 (0.4, 5.5)	.184
NREM OAHI (/h of NREM sleep)	0.5 (0.1, 1.7)	0.5 (0.3, 3.5)	.399
REM OAHI (/h of REM sleep)	1.5 (0.4, 6.7)	3.7 (0.5, 12.6)	.263
Any OSA	72 (47%)	12 (63%)	.189
Mild OSA	47 (65%)	7 (58%)	.642
Moderate OSA	14 (19%)	2 (17%)	.821
Severe OSA	11 (15%)	3 (25%)	.403
SpO_2 awake (%)	98 (97, 98)	98 (96, 98)	.717
SpO_2 NREM (%)	97 (97, 98)	98 (97, 98)	.481
SpO_2 REM (%)	96 (90, 97)	95 (85, 97)	.301
SpO_2 nadir (%)	86 (82, 91)	84 (70, 91)	.431
Time spent < SpO_2 90% (min) (n = 124 & 17)	18.4 (2, 46.2)	17.4 (7.8, 27.4)	.902
Time spent < SpO_2 90% (%)	3 (0.4, 7.3)	2.8 (1.3, 5.4)	.934
Time spent < SpO_2 85% (min) (n = 113 & 16)	17.9 (2.3, 46)	15.1 (4.3, 39.1)	.830
Time spent < SpO_2 85% (%)	2.9 (0.4, 7.2)	2.6 (0.8, 6.4)	.850
Time spent < SpO_2 80% (min) (n = 102 & 15)	22 (5.4, 51.1)	16.1 (1.4, 50.1)	.585
Time spent < SpO_2 80% (%)	3.6 (0.9, 8)	2.7 (0.2, 7.3)	.622

Abbreviations: LBW, low birthweight; NBW, normal birthweight; PSG, polysomnography; REM, rapid eye movement; N1/N2/N3, Stage 1/2/3 non-rapid eye movement; OAHI, obstructive apnea-hypopnea index; NREM, non-rapid eye movement; OSA, obstructive sleep apnea; SpO_2, oxygen saturation; IQR, interquartile range.
Data displayed as median (IQR) for continuous parameters and n (%) for categorical parameters.
p-value derived from Wilcoxon rank-sum test for continuous parameters and 2 tailed proportions z-test for categorical parameters.
* Denotes statistically significant difference between LBW and NBW patients ($p < .05$).

proportion of LBW patients experienced an oxygen saturation nadir below 80% although the median levels reached did not significantly differ between the 2 groups, nor did the median amount of time spent below 80%.

Adjusted effects of birthweight

Multivariate 50th quantile linear regression and logistic regression models adjusting for age, sex, indigenous status, and BMI category were developed to explore the effect of birthweight on selected PSG variables (Table 3). A model incorporating gestation was also run separately, due to the significantly reduced number of patients with this information available. For each 100 g increase in birthweight there was an associated mean 3.1 minute (95% CI 1.4, 4.9) decrease in WASO ($p = .001$). As a result, increasing birthweight was significantly associated with increased sleep efficiency ($p = .015$), as total sleep time did not significantly differ. Each 100 g increase in birthweight was associated with reduced total OAHI, and reduced odds for presence of OSA, though each failed to reach statistical significance ($p = .347$ & $p = .140$, respectively). In the limited model adjusting for gestation, the previously significant effect of birthweight on WASO and sleep efficiency was attenuated, and no significant effect of gestation was identified. However, increasing birthweight was associated with significantly reduced odds of OSA, while increasing length of gestation was associated with significantly increased odds of OSA.

Discussion

This is one of the few studies to explore in depth the association between birthweight and the full range of PSG outcomes, particularly in an Australian population. LBW children displayed significantly reduced sleep efficiency, greater wakenings after sleep onset, and a trend for decreased oxygen saturation nadir. Following multivariate adjustment, a 100 g increase in birthweight was associated with increased sleep efficiency (0.3% change) and a reduction in time spent awake after sleep onset of 3.1 minutes. When gestational length was added to the multivariate model increasing birthweight was found to be a protective factor against OSA, while increasing gestation appeared to increase the risk of OSA.

The potentially increased prevalence of OSA among the LBW cohort noted is not only associated with negative physical health outcomes later in life, but also social and behavioral issues in the short term. Few studies thus far have reported on the effect of birthweight on OAHI, though there is some evidence for increased prevalence of sleep disordered breathing among individuals born VLBW,[17] but not SGA.[20] Pre-school and school aged children with sleep disordered breathing have been reported to have reduced behavioral control; while school aged children have also shown impaired academic ability.[27-29] In a similar manner, LBW children have been reported to show increased behavioral problems in early years of schooling and poorer academic outcomes at this age.[30,31]

Multiple studies have shown a significant association between preterm birth and sleep architecture, efficiency and OSA.[14,15] In contrast, the current study showed increased odds of OSA with increasing

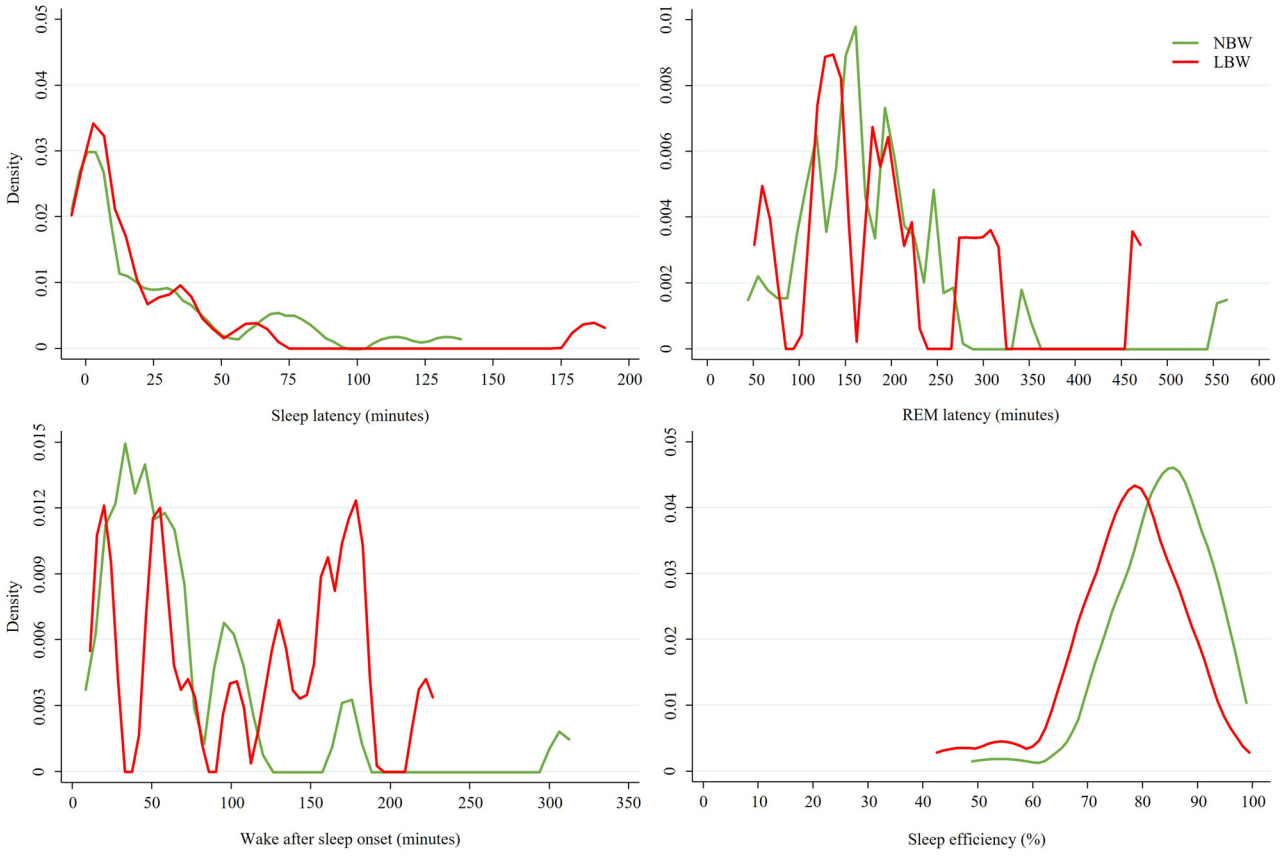

Fig. 2. Kernel density graphs displaying distribution patterns of sleep latency, REM latency, WASO and sleep efficiency for LBW and NBW patients. **Abbreviations**: REM, rapid eye movement; WASO, wake after sleep onset; LBW, low birthweight; NBW, normal birthweight.

gestation and no significant difference in sleep efficiency. It is plausible that our multivariate model incorporating BMI category, age and sex accounts for some of the difference in results. Additionally, the median gestation for LBW children in the current study was 34 weeks compared to 30 weeks reported by Yiallourou et al.[14] While not directly assessed in the current study, the increased risk of OSA via increased gestational length, in conjunction with the reduced risk of OSA via increasing birthweight, suggests that term SGA infants are likely to be at the highest risk of OSA. The observed effect of birthweight on OSA, even after accounting for gestational age, may be driven by differences in body composition and/or respiratory function among individuals born LBW.[7–9] While we accounted for BMI category, we were unable to account for muscle and/or fat mass and distribution, which previous studies have indicated to affect the severity of OSA.[32]

Although median oxygen saturations did not significantly differ between LBW and NBW patients, a higher proportion of LBW patients experienced an oxygen desaturation of <80% (79% vs. 67%), although the median length of time spent at this level was similar between groups. It is plausible that LBW would predispose an individual to a greater severity of desaturations. Although not statistically significant in our study, LBW patients had a higher median OAHI, and a higher proportion of patients with severe OSA. Previous research has shown

Table 3
Quantile linear regression adjusting for age, sex, BMI category, and indigenous status reporting beta coefficients (95% CIs) for effect of 100 g increase in birthweight on selected PSG parameters, and logistic regression for the same adjusted effect on presence of OSA (odds ratio, 95% CI)

PSG parameters	Birthweight (100 g)Beta (95% CI)	Factor *p*-value		Beta (95% CI)	Factor *p*-value
Wake after sleep onset (min)	-3.1 (-4.9, -1.4)	.001*	BW (100 g)	-0.5 (-4.8, 3.8)	.821
			Gestation (1 week)	-3.2 (-11.8, 5.5)	.450
Sleep efficiency (%)	0.3 (0.1, 0.6)	.015*	BW (100 g)	0.2 (-0.6, 1.0)	.603
			Gestation (1 wk)	0.7 (-0.9, 2.2)	.409
Total sleep time (min)	0.8 (-1.5, 3.1)	.494	BW (100 g)	-2.4 (-8.7, 4)	.451
			Gestation (1 wk)	2.8 (-10.4, 16)	.665
SpO_2 nadir	0.1 (-0.1, 0.4)	.356	BW (100 g)	-0.2 (-0.8, 0.5)	.658
			Gestation (1 wk)	0 (-1.4, 1.5)	.976
Total OAHI (/h of sleep)	0 (-0.1, 0)	.347	BW (100 g)	0 (-0.3, 0.4)	.945
			Gestation (1 wk)	0 (-0.7, 0.7)	.936
Any OSA	1 (0.9, 1)	.140	BW (100 g)	0.8 (0.6, 1)	.040*
			Gestation (1 wk)	1.8 (1, 3.2)	.040*

Abbreviations: BMI, body mass index; CI, confidence interval; PSG, polysomnography; OAHI, obstructive apnea-hypopnea index; OSA, obstructive sleep apnea; SpO_2, oxygen saturation; BW, birthweight.
The second columns show the same model additionally adjusted for gestation for the 42 patients who had gestation information available.
* Denotes statistically significant effect of BW or gestation on PSG outcome ($p < .05$).

reduced respiratory function among LBW individuals and a higher prevalence of respiratory disorders such as asthma and chronic obstructive pulmonary disease.[7] Taken together, these factors suggest an increased risk from apneas in the LBW population.

A previous report found a heightened prevalence of 'poor sleepers' (<90% sleep efficiency) among intrauterine growth restricted children in a randomly recruited kindergarten cohort.[33] The current study identified a significantly reduced sleep efficiency among the LBW cohort (median 84% for NBW vs. median 78% for LBW), and a greater prevalence of 'poor sleepers' with twice as many LBW having a sleep efficiency below 80%, in addition to a significantly increased level of sleep disruption as measured by WASO among the LBW cohort. Fragmented sleep patterns have been associated with immune compromise and increased release of pro-inflammatory cytokines.[34] In the long term, this could lead to increased risk of inflammatory associated conditions such as diabetes and hypertension.[35,36] Additionally, disrupted sleep patterns are known to contribute to short term negative behavioral and psychological outcomes, including inattention and hyperactivity, aggression, depressed mood and anxiety.[37] These short-term associations contribute to behaviors such as altered physical activity and eating habits which predict or expound on the long-term associations with obesity, diabetes and hypertension.[38,39] Indeed, it is noted both in existing literature,[3] and in the current study, that the prevalence of obesity is significantly heightened among LBW individuals.

Previous research has identified a significantly heightened risk of hypertension among adult LBW individuals.[10,11] Multiple underlying mechanisms have been identified to explain this including genetic factors, altered adipokines,[8,9,40,41] overfeeding and catch-up growth in infancy,[42] and potentially reduced physical activity.[43] The current study provides additional biologically plausible mechanisms that may contribute to hypertension risk among the LBW population, such as an increased prevalence of OSA (particularly at a greater than "mild" severity), increased arousal index and fragmented sleep patterns among LBW children. Therefore, early monitoring of sleep health may be highly beneficial in the prevention of resistant hypertension in this at-risk cohort.

Limitations

The current study is a retrospective review of PSG data among referred pediatric patients. As such, the degree of sleep abnormalities noted is not representative of the general population. The authors acknowledge that the relatively low number of LBW participants (n = 19) limits the direct comparison of the effect of LBW on sleep outcomes due to potential heterogeneity among patients. A major limitation of the current study is the relative lack of information on gestation – only 43 patients out of 172 had this information available. Previous studies have shown the importance of gestational length on sleep outcomes in pediatric populations, and the current study with limited data appears to show similar interacting effects of birthweight and gestation. Among our LBW patients there was no significant difference in sleep variables between those with or without gestation data available, suggesting an even distribution of gestation length, and thus approximately 80% being born preterm and 30% SGA (Appendix 1). Nonetheless, this is the first study to document PSG parameters amongst an Australian population with LBW in comparison to NBW. Further studies are warranted to compare our study findings to other population.

Conclusion

LBW showed an association with WASO and sleep efficiency which was attenuated after accounting for gestational age. However, both birthweight and gestational age were significantly associated with presence of OSA. OSA, WASO and sleep efficiency have the potential to impact on life course development through diverse short- and long-term associations with physical, mental and social health and functioning. Thus, these disruptions in sleep health among LBW children may be a contributing factor to later life morbidity and long-term health outcomes among this cohort. These findings provide us with opportunities to frame management strategies to promote healthier sleep in LBW and preterm children.

Author contributions

All authors made substantial contributions to conception and design, acquisition of data, or analysis and interpretation of data; took part in drafting the article or revising it critically for important intellectual content; agreed to submit to the current journal; gave final approval of the version to be published; and agree to be accountable for all aspects of the work.

Declaration of conflict of interest

All authors declare no conflicts of interest for this study.

Funding

Nil to declare.

Acknowledgment

We thank all the sleep technologists from Darwin Respiratory and Sleep Health, Darwin private hospital for their dedicated contribution in conducting pediatric sleep studies. We also thank Pediatric Respiratory and Sleep Medicine Physicians Dr Greg Blecher, Dr John Widger and former senior Pediatric Physician Dr Annie Whybourne from the Royal Darwin Hospital, Darwin, for being instrumental in establishing the Pediatric Sleep specialist service in the Top End Northern Territory of Australia. Finally we thank all the other pediatric and Ear Nose and Throat specialists at the Royal Darwin Hospital, Darwin, Northern Territory, Australia.

Supplementary materials

Supplementary material associated with this article can be found in the online version at doi:10.1016/j.sleh.2022.07.003.

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