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Effects of exercise and nutritional intake on sleep architecture in adolescents

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Abstract

PURPOSE—Few studies have evaluated the relationship between sleep architecture and BMI, nutrition, and physical activity in children. This study determined the relationship between sleep architecture and diet and exercise.

METHODS—319 Caucasian and Hispanic children aged 10 to 17 years participated in the follow up assessment of the Tucson Children's Assessment of Sleep Apnea (TuCASA) study. The children and parents completed several questionnaires on dietary habits, amount of physical activity, and sleep habits. Subjects also underwent a home polysomnogram (PSG) to characterize their sleep.

RESULTS—Significant bivariate correlations were noted between Stage II sleep percentage and the following: BMI (r=.246, p<.01), estimated total recreational energy expenditure (r=.205, p<.01), vigorous activity (r=.130, p=.009), and total estimated activity (r=.148, p=0.009). In girls, significant correlations were noted between Stage II percentage sleep and BMI score (r=.279, p<.01). Also in girls, significant negative correlation was noted between REM sleep percentage and total fat intake (r=-.168, p=.039). In boys, significant correlations were again seen between Stage II percentage sleep and the following: BMI score (r=.218, p=.005), estimated total recreational energy expenditure (r=.265, p=.001), vigorous activity (r=.209, p=.008), and total estimated activity (r=.206, p=0.010). When controlling for BMI percentile and age, significant bivariate correlation was also noted between REM sleep percentage and total fat intake (r=.176, p=.034) in boys.

CONCLUSIONS—BMI and exercise were associated with increases in Stage II sleep. In girls, total fat intake was associated with a reduction in REM sleep, while in boys (after controlling for BMI percentile and age) total fat intake correlated with REM sleep.

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Conflict of Interests

The authors declare that they have no conflict of interest.

Keywords

Sleep architecture; BMI; exercise; children; nutrition; Stage II sleep

Introduction

Levels of physical activity, dietary intake, and adiposity are highly inter-related. In general, reductions in physical activity and high caloric intake are correlated, and both are associated with increased adiposity. More recently, there is accumulating evidence that reductions in sleep time may be a risk factor for obesity as well [1-3]. However, there is no clear consensus with regards to the complex inter-relationships among physical activity, dietary intake, obesity, and sleep. Although some studies have found strong links between high body mass index (BMI) and reduced total sleep time [1-4], attempts to associate obesity with alterations in sleep architecture have produced conflicting data [5-7]. With respect to the effects of exercise, a number of studies have found that acute exercise results in increased total sleep time as well as changes in sleep architecture such as increased amounts of slow wave sleep (SWS) and decreased REM sleep [8, 9]. In addition, population-based studies have shown that increased levels of physical activity are associated with reduced risk of sleep disturbances and better sleep quality [8, 10, 11]. In contrast to the evidence linking exercise and/or physical activity to alterations in sleep, studies on the impact of dietary intake on sleep are relatively limited. Most studies have been in adults and showed that a low carbohydrate diet increased SWS and decreased rapid eve movement (REM) sleep [12, 13]. The few studies in children have focused on obese subjects with polycystic ovarian syndrome [14, 15] or those placed on a ketogenic diet [6].

Given the relative lack of information in children, the purpose of this study was to investigate the relationships among sleep architecture and BMI, nutrition, and physical activity using data from a community based cohort of children, the Tucson Children's Assessment of Sleep Apnea study (TuCASA). We hypothesized that increased BMI, greater exercise, and increased caloric and carbohydrate intake would be predictive of sleep disruption, increased non REM non SWS and decreased SWS. This sleep disruption could potentially lead to negative cognitive and behavioral effects, such as hyperactivity, inattention, aggression, poor school performance, and depression/anxiety [16].

Methods

Study Population

The Tucson Children's Assessment of Sleep Apnea study (TuCASA) was designed to investigate the incidence, prevalence and correlates of objectively measured sleep-related breathing disorders in a prospective cohort study of preadolescent Hispanic and Caucasian children ages 6 to 11 years. Detailed description of the protocol has been previously published [17]. Originally, Hispanic and Caucasian children aged 5 to 12 years were recruited to participate in the TuCASA study by soliciting the cooperation of selected elementary schools in the Tucson Unified School District (TUSD)—a large district with an elementary school population representative of children living in Southern Arizona. To assure an adequate recruitment of Hispanic children, elementary school populations were pre-screened so that at least 25%, but less than 75%, of the children were of self-reported Hispanic origin. Children were sent home with a short sleep habits questionnaire. Parents completed the questions and provided demographic information at a minimum and contact information if they would allow study personnel to call them. To increase participations, incentives were provided to classrooms and schools.

This current study is based on data collected in a follow up assessment performed approximately 5 years after the initial phase. Previous participants in the initial phase of TuCASA were enrolled in the second phase of the study after obtaining informed consent of a parent or guardian, and assent of the participating children. The subjects were 10-17 years of age at the time of the follow up study. The children and parents completed several questionnaires on dietary habits, amount of physical activity, sleep habits, and underwent a repeat home polysomnogram (PSG) to characterize their sleep.

Polysomnography

The TuCASA home polysomnography (PSG) methodology, quality assurance, and scoring procedures have been described elsewhere [17]. The PSG included a recording of the electroencephalogram, electrooculogram, chin electromyogram, chest and abdominal effort, airflow by thermistor and nasal pressure, electrocardiogram, and pulse oximetry. Sleep was scored by a single registered polysomnographic technologist using standard criteria [18].

Physical Activity

The Block Kids Physical Activity Screener was used to assess the children's physical activity (http://www.nutritionquest.com), which is designed for school-age children aged 8-17 years. The Screener inquires about frequency and duration of activities in the past week. It quantifies the amount of time spent in leisure and school activities, chores, and part-time jobs.

Dietary Intake

The Rockett Youth/Adolescent Questionnaire (YAQ), a self-administered food frequency questionnaire designed for children ages 9-18 years was used to assess dietary habits [19]. This questionnaire has been tested qualitatively and quantitatively in this age group [20]. It includes 152 questions pertaining to food intake over the last year. Implausible energy intakes <500 kcal/day or >5000 kcal/day were excluded, per the developers of the food frequency questionnaire [19].

Anthropometric Measures

Standardized procedures and identical equipment were used to obtain weight to the nearest 0.1 kg and height to the nearest 0.1 cm. Subjects were measured without shoes on a horizontal surface with heads in the Frankfort Plane. BMI was calculated from weight in kg divided by height in meters-squared.

Data Analysis

Continuous variables were checked for normal distribution. Non-normal variables were log transformed. Pearson correlation coefficients were calculated to determine whether sleep stage percentages were associated with dietary components, BMI, and calculated energy expenditure. Partial correlations were performed controlling for age and/or BMI. All statistical procedures were conducted using SPSS 17.0 for Macintosh (SPSS®, Inc; Chicago, IL, 2010). A significance alpha level of 0.05 was used for all statistical tests. Data where appropriate are shown as mean ± SD.

Results

In the initial TuCASA examination, 503 children were studied, of whom 319 participated in the follow-up examination. The remainder were not enrolled in the follow up study because of refusal or inability to be located. In the initial phase of the study, there were 52.5% Hispanics, while at follow up there were 35.6% Hispanics. As such, more Hispanics were

lost to follow up. No other statistically significant demographic or sleep architecture differences were noted between those who participated in the follow up examination and those who did not. As shown in Table 1, we had 312 subjects in our current analyses (7 subjects were excluded due to implausible energy intakes, as noted above). There were 130 subjects under the age of 13, 128 subjects between the ages of 13 to 15, and 54 subjects above the age of 15 years. Overall, standardized and percentile BMI increased and total sleep time decreased with increasing age for both boys and girls. Interestingly, reported caloric intake decreased with increasing age (despite an increase in BMI).

Table 2 shows correlations for the entire cohort between sleep architecture and the following variables: minutes of exercise, nutritional intake (fat, protein, and carbohydrates), and BMI. There were significant bivariate correlations between Stage II sleep percentage and the following: BMI ($r=.246$, $p < .01$), estimated total recreational energy expenditure ($r=.205$, $p<.01$), vigorous activity ($r=.130$, $p=.009$), and total estimated activity ($r=.148$, $p=0.009$).

To determine whether there were stronger associations between sleep architecture and BMI/caloric intake in very active subjects, we limited our analyses to children in the bottom and top 10^{th} percentiles of total estimated recreational energy expenditure, excluding the rest of the population. An independent t-test was performed comparing the bottom and top 10^{th} percentile with respect to sleep stage and caloric intake. Versus the least active individuals, the most active had increased stage II percentage (29 subjects with a mean 58.92% ± 6.27, $p=.006$ versus 32 subjects with a mean 54.20% ± 6.64), increased caloric intake (27 subjects with a mean 1898 Kcals ± 642, $p=.031$ versus 32 subjects with a mean 1528 Kcals ± 639), increased carbohydrate intake (27 subjects with a mean 275 gm ± 104, $p=.006$ versus 32 subjects with a mean 206 gm ± 88), and a larger BMI z score (32 subjects with a mean 1.23 ± 1.04, $p=.003$ versus 32 subjects with a mean 0.41 ± 1.10).

To determine whether there were any gender specific associations between dietary and physical activity factors and sleep architecture, bivariate correlations were performed separately for boys and girls. Data for girls are shown in Table 3. Again significant correlations were seen between Stage II percentage sleep and BMI score ($r=.279$, $p<.01$), but not energy expenditure. Significant negative bivariate correlation was also noted between REM sleep percentage and total fat intake ($r=-.168$, $p=.039$).

Data for boys are shown in Table 4. Significant correlations were seen between Stage II percentage sleep and the following: BMI score ($r=.218$, $p=.005$), estimated total recreational energy expenditure ($r=.265$, $p=.001$), vigorous activity ($r=.209$, $p=.008$), and total estimated activity ($r=.206$, $p=0.010$). When controlling for BMI percentile and age, significant bivariate correlation was also noted between REM sleep percentage and total fat intake ($r=.176$, $p=.034$).

Discussion

In our cohort of 312 Caucasian and Hispanic adolescents, we found that a higher BMI and energy expenditure were associated with increased Stage II sleep. The more active the subject, the greater the percentage of Stage II sleep, carbohydrate, and caloric intake. Associations were also noted between REM sleep and fat intake. Girls had a negative correlation: with increasing fat intake; there was a reduction in REM sleep. In contrast, boys had a positive correlation: with increasing fat intake; there was an increase in REM sleep.

We observed that BMI is positively correlated with Stage II sleep in children. This finding is consistent with our hypothesis that increasing BMI is associated with more non REM non SWS. Although not statistically significant we also observed a corresponding negative relationship between BMI and SWS. Somewhat similar results have been noted in male

adults in whom SWS decreases with increasing BMI [5]. Studies of sleep architecture in children are not robust and are complicated by confounding factors [6, 7]. Willi et al. showed that obese adolescents, undergoing a ketogenic diet, had increased SWS and decreased REM sleep. These measures normalized after weight loss [6]. Similarly, obese children were noted to have decreased REM sleep [7], but this latter study included children with psychiatric diagnoses, including depression, which can cause changes in sleep architecture [21]. As such the results of that study may not be applicable to a general population of children. Overall, our data in conjunction, with findings from adult studies, seem to indicate increasing BMI is related to an increase in non REM non SWS.

The acute effects of exercise on sleep architecture have been extensively studied. Overall, in the adult population, acute exercise increases total sleep time, increases SWS, and decreases REM sleep [8]. A recent study in children showed that acute high-intensity exercise resulted in increased SWS, decreased Stage II sleep, and shortened sleep onset latency [22]. However, there are few studies on the effects of chronic exercise on sleep architecture. Chronic exercise caused an increase in REM sleep in one study [23], while no changes were noted in another [24]. Our hypothesis is that chronic exercise may lead to sleep disruption and lighter sleep, in the non-athlete population. In support of our hypothesis, we found a positive correlation between Stage II sleep and total estimated activity. Furthermore, the most active group, compared to the least active, showed an even greater percentage of Stage II sleep (58.92% ± 6.27 vs. 54.20% ± 6.64, p=.006). Our findings are consistent with a previous study in adults [25]. In this latter study, after 12 weeks of exercise, subjects showed an increase in stage II sleep and a decrease in SWS. Therefore, our data in children, and similar findings in adults indicate that greater amounts of non-acute exercise are associated with less restorative sleep. In contrast, several epidemiologic studies in adult populations have observed an association between greater amounts of exercise, and better sleep quality. The explanation for this discrepancy is not readily apparent. However, subjective assessment and objective assessments of sleep quality are not always consistent.

Overall, the finding above of BMI and physical activity associated with Stage II sleep is clinically relevant. Alterations in sleep architecture may lead to changes in energy metabolism. A recent study showed that selective SWS deprivation induces insulin resistance [26]. Similarly, diabetic subjects have decreases in SWS [27]. As such it is plausible that increasing BMI and physical activity may lead to changes in sleep architecture which may have a negative consequence on energy metabolism.

In our population with a regular diet, total fat intake was correlated with REM sleep (positively in boys, negatively in girls). Previous studies assessing the effect of diet on sleep architecture have focused on extreme forms of diets. In adults, very low carbohydrate diets increase SWS [12, 13, 28] and decreases REM sleep [12, 13]. However, the effects of variations in the components of a “regular” diet on sleep architecture are unclear. REM sleep may be involved in lipid homeostasis. Previous studies have found that REM sleep was associated with BMI [29, 30]. Furthermore, in animal studies, leptin administration causes a decrease in REM sleep, which may explain the findings seen in our study in girls [31]. Thus the higher leptin levels found in females as compared to males [32] could be leading to a decrease in REM sleep. As such REM sleep may play a role in lipid metabolism. Further studies need to clarify this association. However, an explanation for the reverse finding in boys is not clear. There are multiple factors that could be contributing to this effect. First, boys had greater recreational activity, which could be negating the impact of total fat intake. Second, caloric and fat intake was greater in boys than in girls, possibly leading to the difference noted. Third, there are gender specific differences in lipid metabolism [33], which may be confounding the association. Gender specific differences in sleep stages have been observed previously [34], although the underlying mechanism remains unclear. Obtaining

further data on fat intake and sleep architecture is relevant as it may provide insight on the important sex-differences in lipid metabolism.

Our study has some limitations. The principal one is its cross sectional design, which hinders our ability to draw strong conclusions on the causality of the relationship between sleep architecture and physical exercise and dietary intake. Another limitation is that the study relied on self and parent reported data for diet and self reported data on exercise. However, we used validated food frequency and physical activity questionnaires and excluded data which were implausible. Finally, only 312 of our original 503 children participated in this assessment of the cohort. Thus, it is possible that there was a bias towards studying healthier children who were more likely to remain participants. However, there is no evidence that such a bias would explain our findings. Furthermore, only the percentage of children with Hispanic ethnicity was different between those who participated and those who did not, a finding that is unlikely to be related to our observations.

Despite these limitations, there are several key strengths of our study. Firstly it included a large sample size with ethnic diversity. Secondly, home polysomnograms, with a low failure rate and potentially less first night laboratory effect, were performed in contrast to self-report of sleep duration and quality [17]. Thus, we believe that the sleep architecture data are generally reflective of the children's normal sleep environment.

In conclusion, we found that BMI and exercise were associated with increases in Stage II sleep. In girls, total fat intake was associated with a reduction in REM sleep, while in boys (after controlling for BMI percentile and age) total fat intake correlated with REM sleep. These data may be potentially useful for interpretation of future studies (including interventional trials) on the impact of diet and exercise on sleep.

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Table 1

Sleep and Dietary Data for all subjects stratified by age and gender.

	Age Division					
	< 13 yo		13-15 yo		>15 yo	
	Boy	Girl	Boy	Girl	Boy	Girl
N	65	65	70	58	23	31
Percentile of BMI score	62.94 ± 5.11	58.44 ± 32	61.58 ± 30.7	63.93 ± 29.6	68.77 ± 32.3	67.34 ± 27.7
Total sleep Time	478 ± 57.8	493 ± 56	460 ± 60.7	476 ± 74.8	435 ± 63.7	454 ± 71
Stage I Percentage	4.0 ± 2.53	3.9 ± 2.4	4.1 ± 2	3.5 ± 1.6	4.3 ± 1.9	3.6 ± 2.4
Stage II Percentage	52 ± 5.7	52 ± 6	56 ± 6.5	57 ± 6.1	62 ± 7.9	59 ± 5.4
Stage III/IV Percentage	4.0 ± 1.7	4.0 ± 1.6	3.9 ± 2.2	3.5 ± 1.5	3.9 ± 1.8	3.9 ± 1.9
Stage REM Percentage	23 ± 4.3	22 ± 3.7	22 ± 4.7	23 ± 4.5	24 ± 6.7	24 ± 5.9
Calories kcal	1792 ± 859	1716 ± 528	1711.9 ± 583.5	1570.7 ± 570	1635.1 ± 639.9	1538.1 ± 716
Protein gm	68.91 ± 35.3	69.81 ± 23 .4	64.95 ± 24	58.52 ± 23.4	61.80 ± 25.45	59.92 ± 24.45
Total Fat gm	62.74 ± 30.8	60.23 ± 21.7	60.83 ± 22	56 ± 23	56.74 ± 25	52.27 ± 25.36
Carbohydrates gm	244.53 ± 121	229.18 ± 76.1	232.70 ± 86	213.80 ± 80	226.83 ± 90	212.65 ± 106.68

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Table 2

Correlations between sleep architecture and BMI, diet, and exercise for the entire cohort.

		Total sleep Time	Stage I Percentage	Stage II Percentage	Stage III/IV Percentage	Stage REM Percentage
BMI score at phase 2	r	±.046	.056	.246	±.017	.048
	p	.412	.318	.000	.768	.396
	n	319	319	319	319	319
Calories	r	±.025	.010	±.008	.036	.007
	p	.660	.866	.887	.525	.907
	n	307	307	307	307	307
Protein	r	.015	±.011	±.043	.020	.001
	p	.787	.842	.457	.731	.982
	n	307	307	307	307	307
Animal fat	r	±.025	.018	±.003	.039	±.021
	p	.665	.754	.957	.496	.709
	n	307	307	307	307	307
Vegetable fat	r	.002	±.019	±.040	.066	.020
	p	.974	.739	.482	.247	.728
	n	307	307	307	307	307
Total Fat	r	±.013	.000	±.024	.059	±.001
	p	.818	.996	.674	.302	.984
	n	307	307	307	307	307
Carbohydrates	r	±.043	.022	.013	.024	.013
	p	.458	.707	.818	.673	.817
	n	307	307	307	307	307
Recreational Activity	r	±.040	.035	.205	.067	.048
	p	.488	.540	.000	.242	.402
	n	311	311	311	311	311
Vigorous Activity	r	.007	.047	.130	.063	.029
	p	.898	.404	.021	.261	.607

		Total sleep Time	Stage I Percentage	Stage II Percentage	Stage III/IV Percentage	Stage REM Percentage
	n	318	318	318	318	318
Total Activity	r	±.038	.038	.148	.020	.066
	p	.509	.505	.009	.725	.249
	n	309	309	309	309	309

Table 3

Correlations between sleep architecture and BMI, diet, and exercise for girls only.

		Total sleep	Stage I Percentage	Stage II Percentage	Stage III/IV Percentage	Stage REM Percentage
BMI score	r	−.117	.069	.279	−.076	.016
	p	.145	.395	.000	.347	.844
	n	156	156	156	156	156
Calories	r	−.073	.038	.017	.054	−.111
	p	.376	.643	.834	.513	.177
	n	150	150	150	150	150
Protein	r	−.002	.000	−.074	.059	−.107
	p	.978	.996	.365	.470	.194
	n	150	150	150	150	150
Animal fat	r	−.090	.072	.028	.090	−.187
	p	.273	.382	.736	.276	.022
	n	150	150	150	150	150
Vegetable fat	r	−.072	−.015	.044	.064	−.109
	p	.378	.858	.595	.440	.185
	n	150	150	150	150	150
Total Fat	r	−.092	.032	.041	.087	−.168
	p	.260	.693	.620	.289	.039
	n	150	150	150	150	150
Carbohydrates	r	−.070	.044	.028	.024	−.058
	p	.397	.592	.735	.772	.483
	n	150	150	150	150	150
Recreational Activity	r	.033	.001	.091	.041	.119
	p	.687	.989	.264	.616	.143
	n	153	153	153	153	153
Vigorous Activity	r	.096	−.037	.012	.065	.083
	p	.237	.650	.887	.425	.302

		Total sleep	Stage I Percentage	Stage II Percentage	Stage III/IV Percentage	Stage REM Percentage
	n	155	155	155	155	155
Total Activity	r	.030	−.027	.066	−.016	.036
	p	.713	.744	.419	.848	.658
	n	152	152	152	152	152

Table 4

Correlations between sleep architecture and BMI, diet, and exercise for boys only.

		Total sleep	Stage I Percentage	Stage II Percentage	Stage III/IV Percentage	Stage REM Percentage
BMI score	r	.025	.048	.218	.029	.075
	p	.755	.539	.005	.717	.340
	n	163	163	163	163	163
Calories	r	.030	−.023	−.031	.021	.100
	p	.708	.773	.697	.794	.213
	n	157	157	157	157	157
Protein	r	.039	−.027	−.021	−.008	.084
	p	.629	.740	.790	.925	.295
	n	157	157	157	157	157
Animal fat	r	.054	−.039	−.033	.000	.116
	p	.502	.626	.682	.995	.148
	n	157	157	157	157	157
Vegetable fat	r	.082	−.029	−.113	.067	.131
	p	.309	.721	.160	.407	.103
	n	157	157	157	157	157
Total Fat	r	.076	−.038	−.080	.036	.138[a]
	p	.347	.636	.318	.655	.085
	n	157	157	157	157	157
Carbohydrates	r	−.005	−.008	−.002	.020	.072
	p	.950	.922	.980	.806	.373
	n	157	157	157	157	157
Recreational Activity	r	−.055	.019	.265	.076	.019
	p	.492	.810	.001	.346	.809
	n	158	158	158	158	158
Vigorous Activity	r	−.035	.074	.209	.058	−.007
	p	.657	.350	.008	.459	.926

		Total sleep	Stage I Percentage	Stage II Percentage	Stage III/IV Percentage	Stage REM Percentage
	n	163	163	163	163	163
Total Activity	r	−.076	.059	.206	.043	.096
	p	.342	.461	.010	.597	.232
	n	157	157	157	157	157

[a]When controlling for age and BMI percentile, significant bivariate correlations were also noted between REM sleep percentage and total fat intake (r=.176, p=.034, n=144)