



ORIGINAL ARTICLE

# Longitudinal association of nighttime sleep duration with emotional and behavioral problems in early childhood: results from the Danish Healthy Start Study

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## Abstract

**Study Objectives:** To examine the longitudinal and bidirectional association between nighttime sleep duration and emotional and behavioral problems (EBPs) over 15 months among preschool children.

**Methods:** Data of children aged 2 to 6 years from the control group of the Danish Healthy Start Study, a 15-month obesity prevention intervention, were used. Nighttime sleep duration was measured using a 7-day sleep record. EBPs were assessed by the Strengths and Difficulties Questionnaire Total Difficulties (SDQ-TD) score and Prosocial Behavior (SDQ-PSB) score. Multivariable regression models were conducted to examine the bidirectional associations between changes in nighttime sleep duration and SDQ scores.

**Results:** With adjustment for child, family factors, and parental stress level, every hour extra nighttime sleep at baseline was associated with a 1.02 decrease in SDQ-TD score and 77% lower odds of having an abnormal SDQ-TD score ( $\geq 90$ th percentile) at the follow-up ( $p = 0.01$ ). Children who increased their nighttime sleep duration over the 15-month demonstrated a similar concurrent reduction in SDQ-TD score ( $\beta = -1.28$ ,  $p = 0.02$ ) compared with those who decreased or had no change in nighttime sleep duration. After additional adjustment for sleep problem and habit variables, the significant associations remained. No associations were found between nighttime sleep duration and SDQ-PSB scores. Examination of SDQ scores as predictors of subsequent changes in nighttime sleep duration showed no significant associations.

**Conclusions:** Among preschool children, longer nighttime sleep duration was associated with a decline in EBPs, but not vice versa. Our study provides new longitudinal evidence to support sleep interventions to improve EBPs in early childhood.

Clinical trials: The Healthy Start Study: <https://clinicaltrials.gov/ct2/show/NCT01583335>

Trial registration: ID NCT01583335

## Statement of Significance

The prevalence of emotional and behavioral problems (EBPs) in early childhood is evident and has far-reaching implications across life. Insufficient sleep duration may be linked with EBPs in children, and the association may be bidirectional. This study examined the longitudinal and potential bidirectional association between nighttime sleep duration and EBPs in 2 to 6 years old children over a 15-month follow-up. We found that nighttime sleep duration was a significant predictor of emotional and behavioral total difficulties over 15 months, but not vice versa. Our findings indicate that promoting adequate nighttime sleep duration may be an effective strategy for preventing and reducing the prevalence of EBPs in young children.

**Key words:** childhood; emotional and behavioral problems; sleep duration; longitudinal

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## Introduction

The prevalence of emotional and behavioral problems (EBPs) in early childhood is evident, and recent studies have reported that more than 10% of preschool children were affected by EBPs [1–4]. Common EBPs in early childhood include behavioral dysfunction, inattention/hyperactivity, depression, and anxiety [3, 5]. EBPs in early childhood tend to persist into later life, such as mid-childhood, adolescence, and adulthood, resulting in far-reaching and long-term consequences at individual, family, and society levels [3, 5–7]. Examples of consequences of early childhood EBPs include poor social interaction, abnormal neurocognitive functioning, delayed school readiness and problems in later childhood, and persistent mental health problems and obesity in adulthood [3, 5–7].

A number of genetic and environmental factors contribute to EBPs, and short sleep duration has been proposed as one of the contributing factors in children [8, 9]. Given the long-lasting implications of EBPs across life, understanding if and how sleep duration influences EBPs in early childhood is particularly valuable for informing future interventions and strategies for tackling the prevalence of EBPs early in life. The mechanisms through which sleep duration affects EBPs in children remain equivocal. Both genetic and environmental factors may contribute to the association via influencing hormone secretion as well as neural and psychological pathways [8]. Parental influence and family environment have also been proposed to have impacts on the association [8].

Evidence linking sleep duration and EBPs in early childhood are however limited largely to cross-sectional studies where the temporal order of the relationship cannot be determined [10–14]. Only a few prospective cohort studies are available to suggest that short sleep duration in preschool years, ranging from less than 10 to 12 hours, may influence the subsequent development of EBPs in childhood [15–17]. It has been hypothesized that the relationship between sleep and EBPs in childhood and adolescence may be bidirectional [8]. However, whether bidirectional associations exist between sleep duration and EBPs in early childhood remains to be explored. Further longitudinal studies with repeated measurements of both sleep duration and EBPs, to allow for better insights into the bidirectionality and the temporal sequence of the association, and in different study populations, are warranted [8, 18]. The aim of the present study was to explore the longitudinal and bidirectional association between nighttime sleep duration and EBPs over 15 months among 2 to 6 years old obesity-predisposed children.

## Methods

### Participants

Data from the control group ( $n = 315$ ) of the Danish Healthy Start Study [“Sund Start”], a 15-month randomized obesity prevention intervention study, were used. The study was conducted between 2009 and 2011 and aimed to prevent the development of overweight among 2 to 6 years old normal-weight children with a predisposition for future overweight as indicated by at least one of the following risk factors: a high birth weight ( $>4$  kg), maternal pre-pregnancy body mass index (BMI;  $>28\text{kg/m}^2$ ), or low maternal education level ( $\leq 10$  years). The intervention group received a 15-month intervention focusing on diet, physical

activity, sleep, and stress management. The control group was followed up at the same time, with an average follow-up time of  $15 \pm 3$  months but received no intervention. Details of the study are described elsewhere [19, 20].

### Assessment of nighttime sleep duration

Parents completed a 7-day sleep record from Monday to Sunday to capture nighttime sleep duration on weekdays and weekends, at both baseline and follow-up. Parents reported the time when their child fell asleep at night and when they woke up in the morning. Nighttime sleep duration was calculated as the average over six nights (i.e. Monday evening to Tuesday morning, Tuesday evening to Wednesday morning, etc.) in hours. Only children with complete nighttime sleep duration over six nights were included in the analysis.

### Assessment of EBPs

Child’s EBPs were collected using a Danish single-sided version of the Strengths and Difficulties Questionnaire (SDQ) at both baseline and follow-up. The SDQ is a validated tool that was developed to screen for EBPs among 3 to 16 years old children and adolescents [21]. It is composed of 25 items that measure four “difficulties” domains (emotional symptoms, conduct problems, hyperactivity/inattention, and peer relationship problems) and one “strength” domain (prosocial behavior [PSB]). The responses to each item were: not true, somewhat true, and certainly true. The four “difficulties” domains were summed up to generate the Total Difficulties score (TD score) based on a scoring syntax available from the SDQ-webpage with higher scores indicating more difficulties [22]. The “PSB” score was analyzed separately as it is conceptually different from the presence of psychological difficulties. Higher PSB scores indicate a more positive prosocial behaviors. The SDQ scores have shown to be a useful screening tool among both population and clinical studies in Scandinavian countries including the Danish population [23, 24]. The internal consistency of the SDQ scores in Danish children was previously examined and found to be high [23]. In the present sample, Cronbach’s alpha between the individual subscales and total difficulties scale was 0.75 and 0.72 at baseline and follow-up, respectively.

### Assessment of child and parental covariates

Child’s height and body weight were measured by stature meter (Soehnle 5002 or Charter ch200P) and a mechanical weight or beam-scale type weight (Tanita BWB-800 or SV-SECA 710), respectively. A parental questionnaire was completed at baseline to collect information on the number of siblings living with the child, whether parents were divorced (yes, no), annual household income, parental education, parental height and weight, child’s physical activity habits, and parental stress level. Parental education was categorized into low (primary and lower secondary school, upper secondary, one or more short courses, or skilled worker), medium (short-term or medium-term post-high school education 3–4 years), or high (further long-term education  $> 4$  years, research worker level). Parental BMI was calculated by body weight in kilograms divided by height in centimeter squared. Parental perception of child’s physical

activity compared with peers was used to represent the physical activity of the child (low: not as active, fairly active and high: very active).

The parental stress level was measured by 10 questions selected from the Swedish version of the parenting stress index (PSI), which is a reliable and valid instrument for measuring parental experiences of stress in the parent–child interaction [25]. The instrument explores the changes in life that parents had perceived since they had the child. It comprises 10 questions measuring sleep, stress, worries, time for themselves, household conflicts, workload, social gatherings in the home, joy of life, everyday surplus energy, and complexity of being a parent compared with expectations. The responses to these questions were: more, less, or no difference compared with before having the child. The internal consistency of the questionnaire was reported previously [26]. Based on Cronbach's alpha and principal component analysis, 9 of 10 questions were combined to get a score to represent the overall parental stress level [26].

In addition to the 7-day nighttime sleep record, parents responded to five questions relating to child sleep problems and habits at baseline: difficulty falling asleep, scared of falling asleep, nightmare, afternoon sleep, and sleep in parents' bed with two response options: yes or no. These questions were adapted from a Danish sleep questionnaire [27] and the Tayside Children's Sleep Questionnaire [28]. Both questionnaires showed good reliability for assessing sleep problems and patterns in young children [27, 28].

### Statistical analysis

Descriptive analysis of participant characteristics by child sex was conducted. Change in nighttime sleep duration and SDQ scores were calculated as the follow-up minus the baseline values. Multivariable linear regression with adjustment for covariates was used to examine the associations between nighttime sleep duration at baseline or nighttime change in sleep duration from baseline to follow-up in relation to changes in SDQ-TD and SDQ-PSB scores from baseline to follow-up. Nighttime sleep duration was also analyzed as tertiles based on data distribution to assess a potential linear dose–response relationship. SDQ-TD score was analyzed further as a categorical variable (normal/borderline: <90th percentile and abnormal: ≥90th percentile) using the cutoffs proposed by Goodman [21]. Multivariable logistic regression was performed to investigate the association between nighttime sleep duration at baseline and the odds of having an abnormal SDQ-TD score at follow-up. Models were initially adjusted for the following child and family-related factors: child age, sex, BMI z-score at baseline, the number of siblings living with the child, whether parents were divorced (yes, no), annual household income (million Danish kroner/year), parental education (low, medium, and high), and duration of the follow-up (model 1). The length of follow-up was also included as a covariate as it varied between individuals ( $15 \pm 3$  months). Parental stress level was documented as a predictor for the development of EBPs in children [29, 30]. Parental stress level was, therefore, also considered as a potential covariate and was also adjusted in model 2. Interaction between child sleep duration and parental stress level was examined, and no interaction was found. Previous studies have associated sleep problems and habits with EBPs in children [31–34]. In model 3, we adjusted for

five sleep problems and habit variables (difficulty falling asleep, scared of falling asleep, nightmare, afternoon sleep, and sleep in parents' bed) as additional covariates. Pearson correlation was conducted between sleep duration and sleep problem and habit variables to assess potential multicollinearity ( $r > 0.8$ ) [35], all correlations are smaller than  $r < 0.23$ ; [Supplementary Table S1](#). Sleep problem and habit variables were, therefore, included in the model simultaneously. In models between concurrent change in sleep and SDQ scores, adjustment for change in BMI z-score and parental stress level from baseline to follow-up was made. To assess a potential bidirectional relationship, we conducted analyses assessing associations of SDQ scores at baseline and change in SDQ scores from baseline to follow-up in relation to change in sleep duration from baseline to follow-up with adjustment for the aforementioned covariates.

To further investigate the bidirectional relationship between nighttime sleep duration and SDQ scores from baseline to follow-up, a two-wave cross-lagged panel model with maximum likelihood estimation via structural equation modeling was performed. Longitudinal invariance of nighttime sleep duration and SDQ scores was assumed. The model was adjusted for covariates mentioned in models 2 and 3. The following model fit statistics were considered good model fit: likelihood ratio Chi-square ( $\chi^2$ ) test ( $p > 0.05$ ), Tucker–Lewis Index (TLI  $> 0.95$ ), comparative fit index (CFI  $> 0.95$ ), and root mean square error of approximation (RMSEA  $< 0.05$ ). All analyses were conducted using Stata 15.0.

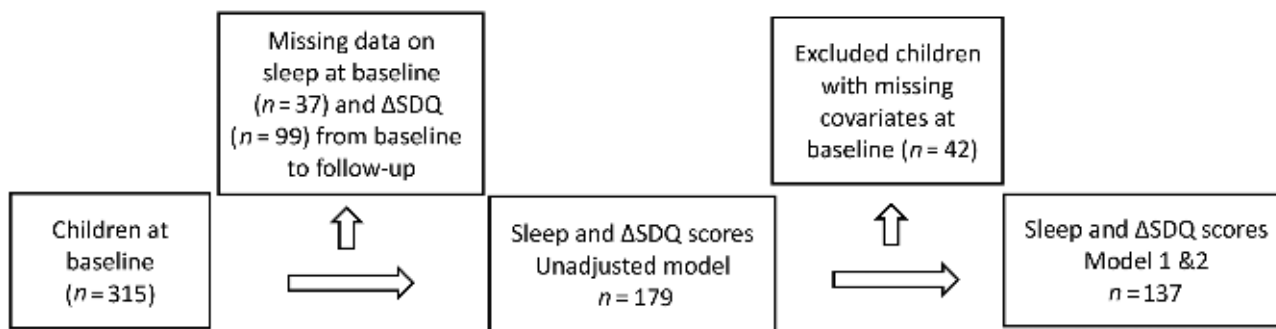
### Results

The number of children included in the prospective analysis between sleep at baseline and change in SDQ scores from baseline to follow-up and the longitudinal analysis between concurrent changes in sleep and SDQ scores from baseline to follow-up was 179 and 167, respectively. A flow chart showing the number of participants in each analysis is provided in [Figure 1](#). A comparison of study characteristics between those included ( $n = 179$ ) versus excluded ( $n = 136$ ) in the analysis revealed no significant differences, apart from BMI z-scores. Children included versus excluded in the analysis appeared to have a slightly lower BMI z-scores at baseline (mean difference  $-0.20$ ; 95% CI  $-0.40, 0.03$ ;  $p = 0.10$ ) and follow-up (mean difference  $-0.30$ ; 95% CI  $-0.60, 0.04$ ;  $p = 0.10$ ) (data not shown).

Study characteristics including child age, nighttime sleep duration, SDQ-TD score, SDQ-PSB score, child and family covariates, parental stress level, and sleep problem and habit variables by child sex are shown in [Table 1](#). Apart from boys having a 0.5 lower SDQ-PSB score than girls at baseline ( $p = 0.048$ ), no differences were seen for other variables.

Nighttime sleep duration at baseline (hours/day) was associated with a decrease in SDQ-TD score at follow-up in the unadjusted model ( $\beta = -1.11$ ,  $p = 0.002$ ) ([Table 2](#)). After adjusting for child and family factors, every one-hour extra sleep at baseline was associated with 1.06 points decrease in SDQ-TD score (model 1). The association remained significant after additional adjustment for parental stress level ( $\beta = -1.02$ ,  $p = 0.01$ ) (model 2). Controlling for sleep problem and habit variables strengthened the association between nighttime sleep duration and SDQ-TD score ( $\beta = -1.37$ ,  $p = 0.001$ ) (model 3). Assessment of nighttime sleep duration in tertiles at baseline showed a significant linear dose–response trend for subsequent change in SDQ scores in all

## Prospective analysis



## Longitudinal analysis

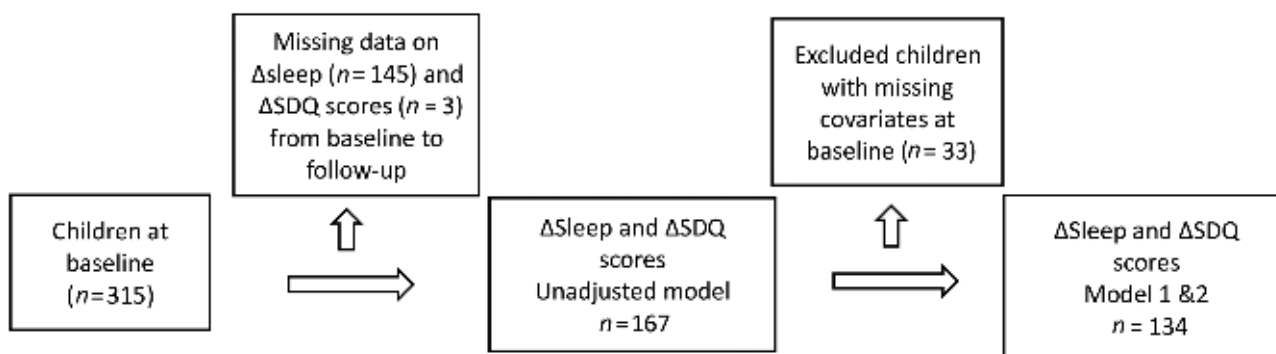


Figure 1. Flow chart showing the number of participants included in each analysis. Covariates: baseline Body mass index z-score, number of siblings living with the child, whether parents were divorced, maternal education, paternal education, household income, length of follow-up, parenting stress level and sleep problem, and habit variables.

models, apart from model 2 with adjustment for parental stress level (Figure 2).

Compared with children who decreased or had no change in nighttime sleep duration from baseline to follow-up, those children who increased their sleep duration had a concurrent decrease in SDQ-TD score in all models (Table 2). With adjustment for child and family factors in model 1, children who increased their nighttime sleep duration had a lower SDQ-TD score ( $\beta = -1.44$ ,  $p = 0.01$ ) at the follow-up than those who decreased or had no change in sleep duration. After additional adjustment for parental stress level, the significant difference in SDQ-TD score remained but was reduced slightly to  $-1.28$  ( $p = 0.02$ , model 2). In contrast, adjusting for sleep problem and habit variables enhanced the estimates of the association ( $\beta = -1.36$ ,  $p = 0.02$ , model 3).

The percentage of children with abnormal SDQ-TD scores ( $\geq 90$ th percentile) at baseline and follow-up was 16% and 15%, respectively. Nighttime sleep duration (hours/day) at baseline was also associated with lower odds of abnormal SDQ-TD score at follow-up in all models (Table 3). Every hour extra sleep at baseline was associated with 76% lower odds of having an abnormal SDQ-TD score (95% CI 0.07, 0.89) after adjusting for child and family factors, parental stress level, sleep problem, and habit variables (Table 3, model 3).

No associations were found between nighttime sleep duration at baseline, whether included in linear models as hours

per day (Table 2) or tertiles (data not shown) and subsequent change in SDQ-PSB score. Neither before nor after adjustment were the concurrent associations between changes in nighttime sleep duration and SDQ-PSB scores from baseline to follow-up significant (all  $p > 0.05$ , Table 2). Associations between any of the SDQ scores and nighttime sleep duration were weak and not significant (data not shown).

Results of the cross-lagged panel model between nighttime sleep duration and SDQ-TD and SDQ-PSB scores with adjustment for covariates were depicted in Figures 3 and 4, respectively. The model between nighttime sleep duration and the SDQ-TD score with adjustment for child and maternal factors and parental stress level demonstrated a good model fit ( $\chi^2$  test  $p = 0.29$ , TLI = 0.96, CFI = 1.00, and RMSEA = 0.04). The model produced similar results to multivariable regression analyses. Nighttime sleep duration at baseline was a predictor for SDQ-TD score at follow-up ( $\beta = -0.96$ ,  $p = 0.01$ ), but not vice versa ( $\beta = 0.005$ ,  $p = 0.71$ ) (Figure 3a). Further adjustment for sleep problem and habit variables also enhanced the strength of the association between nighttime sleep duration and SDQ-TD score (Figure 3b). Likewise, consistent with multivariable regression analyses, no significant association was found for nighttime sleep duration and SDQ-PSB score (model fit:  $\chi^2$  test  $p = 0.48$ , TLI = 1.07, CFI = 1.00, and RMSEA < 0.001) (Figure 4).

**Table 1.** Characteristics of participant by child sex from the control group of the Healthy Start Study who participated in both baseline and follow-up assessments (n = 179)

	N	Boys			Girls		P
		Mean	SD	N	Mean	SD	
<b>Baseline</b>							
Age (years)	105	4	1.1	74	4	1.1	0.79
Nighttime sleep duration(hours/day)	105	10.7	0.6	74	10.7	0.6	0.87
SDQ Total difficulties score	105	6.2	4	74	5.9	3.7	0.63
SDQ Prosocial behavior score*	105	7.6	1.8	74	8.1	1.6	0.048
Child BMI z score	105	0.3	0.8	74	0.3	0.9	0.8
PSI score	95	12.6	2.1	71	12.5	2.3	0.74
Annual household income (Danish kroner)	142	0.8	0.2	101	0.8	0.3	0.70
	N	%		N	%		
Number of siblings	99			69			
0	7	7.1		8	11.6		0.74
1	67	67.7		43	62.3		
2	23	23.2		16	23.2		
3	2	2		2	2.9		
Maternal education	103			72			
Low	18	17.5		18	25		0.22
Medium	56	54.4		41	56.9		
High	29	28.2		13	18.1		
Paternal education	103			71			
Low	35	34.0		31	43.7		0.10
Medium	38	36.9		29	40.8		
High	30	29.1		11	15.5		
Whether parents were divorced (no)	104	99.0		70	94.6		0.08
<b>Sleep problems and habits</b>							
Difficulty falling asleep (yes)	19	18.3		13	18.1		0.97
Scared of falling asleep (yes)	15	14.3		7	9.6		0.35
Nightmare (yes)	22	21.0		10	13.5		0.20
Afternoon nap (yes)	33	31.4		22	29.7		0.81
Sleep in parents' bed (yes)	70	66.7		47	63.5		0.66
<b>Follow-up</b>							
	N	Mean	SD	N	Mean	SD	
Age (years)	105	5.3	1.1	74	5.3	1.0	0.90
Nighttime sleep duration (hours/day)	97	10.7	0.5	70	10.8	0.5	0.23
SDQ-TD score	105	5.7	3.8	74	5.2	3.5	0.36
SDQ- PSB score	105	8.0	1.6	74	8.4	1.5	0.06
Child BMI z score	103	0.4	0.8	73	0.3	0.8	0.95
PSI score	100	12.7	2.0	72	12.6	2.3	0.18

\*p &lt; 0.05; SD, standard deviation; reference category for whether parents were divorced is "yes," and for sleep problems and habits is "no."

## Discussion

Among 2- to 6-years-old obesity-predisposed children, both nighttime sleep duration at baseline and an increase in nighttime sleep duration from baseline to follow-up were associated with lower emotional and behavioral total difficulties at a 15-month follow-up. Independent of various child and family factors and parental stress level, every 1-hour increase in nighttime sleep duration at baseline was associated with a 1.02-point reduction in emotional and behavioral total difficulties score at follow-up. Moreover, every hour increase in nighttime sleep duration at baseline was associated with 77% lower odds of having an abnormal SDQ-TD score at follow-up. Furthermore, a dose-response relationship was found when nighttime sleep duration was modeled as tertiles. Additionally, relative to children who had a decrease or no change in nighttime sleep duration from baseline to follow-up, those who showed an increase in their nighttime sleep duration had a 1.28-point lower SDQ-TD score at the follow-up. Consistently in all models between nighttime sleep duration and SDQ-TD score, the estimates of association

became slightly stronger after adjusting for sleep problem and habit variables. We found no evidence of an association between nighttime sleep duration and prosocial behaviors (SDQ-PSB scores). Examination of SDQ scores as predictors of nighttime sleep duration also showed no significant associations. These findings were replicated in the cross-lagged panel model.

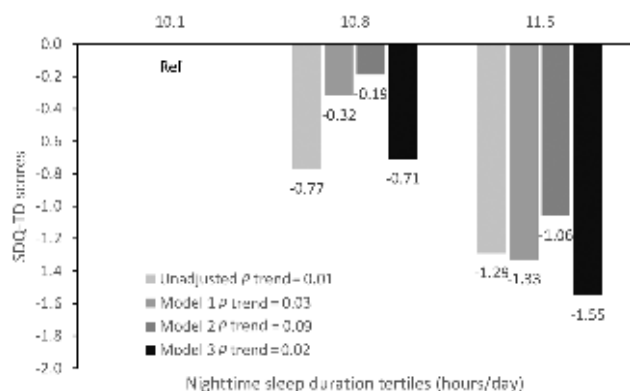
The few previous studies that have examined prospective associations between total/24-hour sleep duration and development of EBPs in early childhood all reported significant associations in line with our findings [15–17]. For instance, by using the same SDQ tool to measure EBPs, among 1,046 US children, Taveras et al. [17] found that insufficient sleep (<10 hours) in the preschool years (ages 3–4 years) was associated with 1.91 points higher SDQ scores at 5 to 7 years compared with those with sleep duration greater than or equal to 10 hours. Likewise, Jansen et al. [15], in a large cohort of Dutch children (n = 4,782), reported that a short sleep duration (<12.5 hours) at the age of 2 years was associated 32% greater risk of having EBPs at the age of 3 years. However, in contrast to the results from these two studies that both reported

**Table 2.** Linear regression analyses between nighttime sleep duration at baseline, change in nighttime sleep duration, and change in SDQ-TD and SDQ-PSD scores from baseline to follow-up

	ΔSDQ-TD			ΔSDQ-PSB		
	β	SD	P	β	SD	P
<b>Sleep duration (hours)*</b>						
<i>Unadjusted</i>	-1.11	0.35	0.002	-0.28	0.18	0.13
Model 1	-1.06	0.38	0.01	-0.40	0.22	0.07
Model 2	-1.02	0.38	0.01	-0.35	0.23	0.13
Model 3	-1.37	0.41	0.001	-0.33	0.25	0.20
<b>Change in sleep duration†</b>						
<i>Unadjusted</i>						
Decrease/no change	REF			REF		
Increase	-1.52	0.41	<0.001	0.27	0.26	0.30
<b>Model 1</b>						
Decrease/no change	REF			REF		
Increase	-1.44	0.56	0.01	0.36	0.31	0.25
<b>Model 2</b>						
Decrease/no change	REF			REF		
Increase	-1.28	0.54	0.02	0.31	0.34	0.35
<b>Model 3</b>						
Decrease/no change	REF			REF		
Increase	-1.36	0.58	0.02	0.25	0.36	0.49

\*Unadjusted model includes baseline sleep duration and baseline TD or PSB scores ( $n = 179$ ); model 1: adjusted for child and family-related factors, including child age, sex, BMI z-score at baseline, the number of siblings living with the child, whether parents were divorced, annual income, maternal and paternal education (low as reference category), and length of follow-up ( $n = 137$ ); model 2: additionally adjusted for PSI upon model 1 ( $n = 137$ ); model 3: additionally adjusted for sleep problem variables (sleep in parents' bed, difficulty falling asleep, afternoon sleep, scared of falling asleep, and nightmare at baseline upon model 2 ( $n = 137$ )).

†Unadjusted model includes change in sleep duration, baseline sleep duration, and baseline TD or PSB scores ( $n = 167$ ); model 1: adjusted for child and family-related factors, including child age, sex, change in BMI z-score, the number of siblings living with the child, whether parents were divorced, annual household income, maternal and paternal education (low as reference category), and length of follow-up ( $n = 134$ ); model 2: additionally adjusted for change in parental stress level upon model 1 ( $n = 134$ ); model 3: additionally adjusted for sleep problem and habit variables: difficulty falling asleep, scared of falling asleep, nightmare, afternoon sleep, and sleep in parents' bed at baseline upon model 2 ( $n = 134$ )).



**Figure 2.** Linear regression analyses between sleep duration tertiles (hours) at baseline and change in SDQ-TD scores from baseline and follow-up ( $n = 179$ ). Unadjusted model includes baseline sleep duration tertiles and baseline SDQ-TD score; model 1: adjusted for child and family-related factors, including child age, sex, BMI z-score at baseline, the number of siblings living with the child, whether parents were divorced, annual income, maternal and paternal education, and length of follow-up; model 2: additionally adjusted for parental stress level upon model 1. Mean sleep duration (95% CI) for each tertiles: tertile 1, 10.1 (8.8, 10.5); tertile 2, 10.8 (10.5, 11.0); and tertile 3, 11.5 (11.1, 12.6); model 3: additionally adjusted for sleep problem and habit variables: difficulty falling asleep, scared of falling asleep, nightmare, afternoon sleep, and sleep in parents' bed at baseline upon model 2.

a threshold effect between sleep duration and EBPs, we found a linear dose–response relationship. Similar findings have been reported in a previous prospective cohort study of Norwegian children ( $n = 556$ ), where a dose–response relationship was found between sleep duration at 18 months and the incidence of EBPs

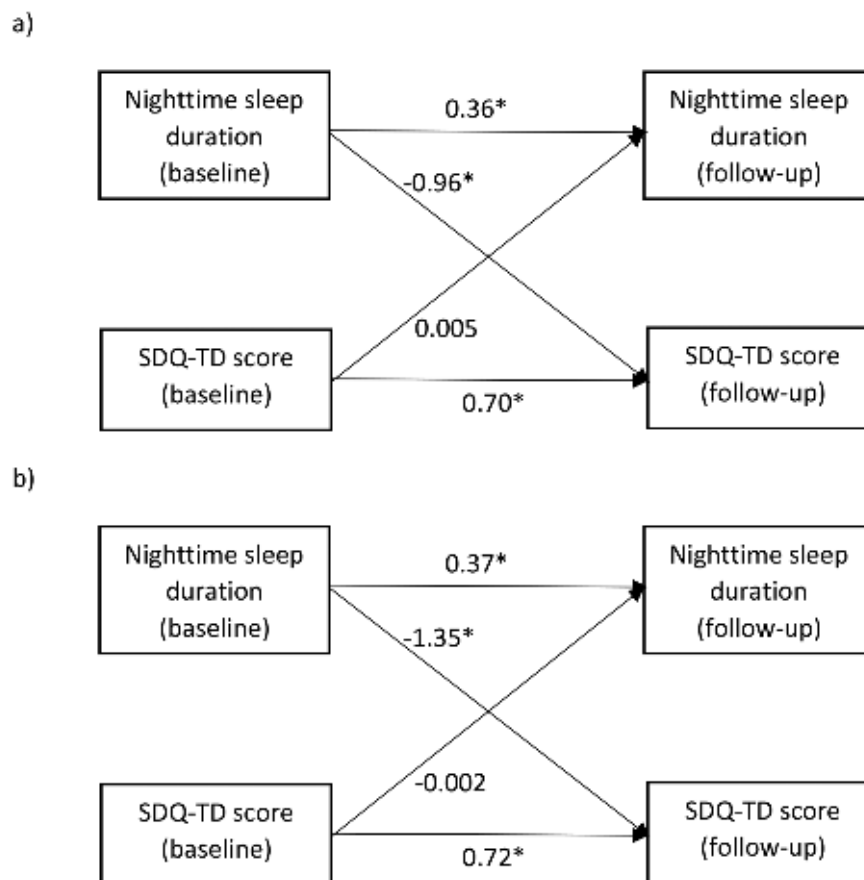
**Table 3.** Logistic regression analysis between nighttime sleep duration at baseline and odds of having an abnormal SDQ-TD score at the follow-up

	(SDQ-TD score > 80th percentile)		
	OR	95% CI	P
<b>Continuous (hours)</b>			
<i>Unadjusted</i>	0.45	(0.21, 0.97)	0.04
Model 1	0.23	(0.07, 0.68)	0.01
Model 2	0.23	(0.07, 0.74)	0.01
Model 3	0.24	(0.07, 0.89)	0.03

Unadjusted model includes baseline sleep duration and SDQ-TD abnormal score at baseline ( $n = 179$ ); model 1: additionally adjusted child age, sex, body mass index z-score at baseline, the number of siblings living with the child, whether parents were divorced, annual household income, maternal and paternal (low as reference category), and length of follow-up ( $n = 137$ ); model 2: additionally parental stress level upon model 1 ( $n = 137$ ); model 3: additionally adjusted for sleep problem and habit variables: difficulty falling asleep, scared of falling asleep, nightmare, afternoon sleep, and sleep in parents' bed at baseline upon model 2 ( $n = 137$ )).

at the age of 5 years [16]. It should be noted that both Jansen et al. [15] and Sivertsen et al. [16] used the Child Behavior Checklist to measure EBPs. Despite the different tools used, their findings were consistent with ours, which further reinforces an inverse association between longer sleep duration and lower risk of EBPs. The beneficial effects of promoting adequate sleep on EBPs were also demonstrated in two sleep intervention studies among young Australian children [36, 37].

Controversy exists as to whether there is a bidirectional relationship between sleep duration and EBPs. That is, whether the



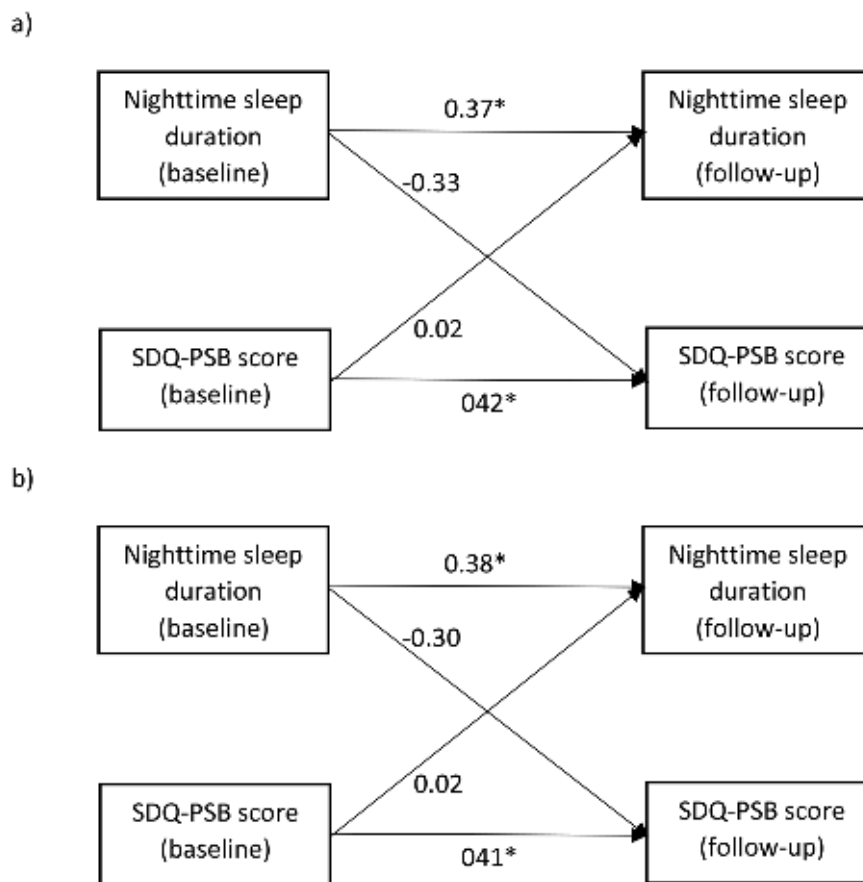
**Figure 3.** Cross-lagged panel models between nighttime sleep duration and SDQ-TD score ( $n = 137$ ). (a) The model was adjusted for covariates: child age, sex, body mass index z-score, the number of siblings living with the child, whether parents were divorced, annual household income, maternal and paternal education, length of follow-up, and parental stress level at baseline. (b) The model was additionally adjusted for sleep problem and habit variables at baseline: difficulty falling asleep, scared of falling asleep, nightmare, afternoon sleep, sleep in parents' bed. \* $p < 0.05$ .

short sleep duration contributes to the development of EBPs in children or EBPs are a persistent phenomenon leading to insufficient sleep. Given our longitudinal design, we were able to address this conundrum to provide further insights into the temporal sequence between sleep duration and EBPs in early life. Our findings demonstrate that sleep duration was a predictor of EBPs, but the reverse association was not significant. The overall low level of EBPs as indicated by average low SDQ scores in our sample may be responsible for the lack of reverse associations. However, in alignment with our findings, Jansen et al. also found no evidence for a bidirectional association between short sleep duration and EBPs and sleep duration at 2 years predicted the development of EBPs at 3 years, but not vice versa [15]. One previous study reported on a bidirectional association between sleep problems at 4 to 5 years and EBPs at 12 to 13 years in a longitudinal cohort of Australian children [18]. However, the findings from that study are not comparable to results from our study due to the different age groups, sleep measures examined, and the longer duration of follow-up.

The mechanisms linking insufficient sleep and EBPs remain unclear. It is speculative that several hormonal pathways, as well as neural and psychological processes, have been implicated in short sleep and EBP. Altered hormone secretion such as melatonin and cortisol may be involved in the association between sleep deprivation and EBPs [8]. Emerging evidence also suggests that sleep deprivation may heighten the neural

response to negative emotional stimuli, thereby limiting the ability to moderate emotions [38]. Furthermore, inadequate sleep may disrupt the recognition of emotional expressions (i.e. happy and anger) [39] and neurobehavioral functioning [17] that subsequently contribute to compromised emotional and behavioral regulation. Parental and family influences may also account for the association between poor sleep and EBPs. Poor parent-child interaction, maternal depression, family disorganization, and stressful life events have all been reported to influence both sleep and EBPs in children [8]. Our study findings also suggest that the association between nighttime sleep duration and EBPs is independent of sleep problems and habits.

Our study has several strengths. The longitudinal design allows us to assess the temporal order for the relationship between nighttime sleep duration and EBPs in early childhood. We conducted both multivariable regression analysis and cross-lagged panel model to assess the potential presence of a bidirectional relationship, and the similar results from both approaches support the robustness of our findings. We controlled for a wide range of confounding factors that are potentially associated with EBPs in children, including child body weight status [26], parental education and household income as proxies for socioeconomic position [40], family factors such as number of siblings and whether parents were divorced [5], and parental stress [29]. A recently published review has highlighted the



**Figure 4.** Cross-lagged panel models between nighttime sleep duration and SDQ-PSB score ( $n = 137$ ). (a) The model was adjusted for covariates: child age, sex, body mass index z-score, the number of siblings living with the child, whether parents were divorced, annual household income, maternal and paternal education, length of follow-up, and parental stress level at baseline. (b) The model was additionally adjusted for sleep problem and habit variables at baseline: difficulty falling asleep, scared of falling asleep, nightmare, afternoon sleep, sleep in parents' bed. \* $p < 0.05$ .

importance of considering parental and family factors when assessing sleep and EBPs relationship in children [8]. The role of parental stress in the development of EBPs in children has been widely recognized [30, 41], and some studies suggest that insufficient child sleep can also result in parental stress [29, 42]. No previous studies, to the best of our knowledge, have accounted for the impact of parental stress and sleep problems and habits when examining the prospective and longitudinal association between sleep duration and EBPs.

Limitations of our study include a relatively small sample size and the sample had low levels of emotional and behavioral concerns as indicated by the low mean SDQ-TD score. However, despite these, we found a strong relationship between nighttime sleep duration and EBPs in early childhood. Also, our findings are limited to children with a high predisposition to future overweight and obesity. Nevertheless, given the high prevalence of overweight and obesity in children worldwide, confirmation of the link between sleep duration and EBPs in this vulnerable group is valuable. In addition, both nighttime sleep duration and EBPs measures were based on parent report. Potential misreporting cannot be dismissed, although the conduction of a 7-day sleep record including both weekdays and weekends may reduce reporting bias. The exclusion of more than half of the sample in the analysis due to missing data and loss to follow-up is another limitation. Given the high attrition, missing data imputation is not feasible. However, when we compared the characteristics of included versus

excluded children, we found no difference apart from borderline significant differences related to BMI z-scores. Children with higher BMI z-scores were more likely to drop out from the study. As a result, we included BMI z-scores as a confounder to minimize the attrition bias. Previous analyses of the same data revealed that children with high SDQ scores were more likely to have greater BMI z-scores [26]. Thus, including these children with higher BMI z-scores in the analyses may slightly reduce the magnitude of the association between sleep duration on SDQ scores. Another major limitation of our study is that we assessed nighttime sleep duration only. Examination of total sleep duration, including both daytime and nighttime sleep, would be desirable.

Our study provides new longitudinal evidence toward the potential beneficial effect of longer nighttime sleep duration in reducing EBPs in early childhood. Moreover, our finding that nighttime sleep duration precedes the EBPs, but not vice versa, is promising. Targeting sufficient nighttime sleep duration in early life interventions may be an effective strategy for reducing the prevalence of children's EBPs and promoting mental and psychosocial health and well-being of young children. Longitudinal studies with longer duration of follow-up and assessing a more extensive range of sleep measures are needed to further our understanding of the relationship between sleep and EBPs in children. Knowledge of the mechanisms underpinning the association between sleep and EBPs is also desirable and is warranted for investigation in future studies.



## Supplementary material

Supplementary material is available at SLEEP online. .

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