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High-dose vs standard-dose vitamin D and sleep in early childhood: A randomized clinical trial

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ABSTRACT

Objective: Low 25-hydroxyvitamin D concentration (25(OH)D) has been associated with sleep problems. There are no randomized trials on the association in childhood. We tested whether a high vs standard vitamin D_3 dose has beneficial effects on sleep until 2 years of age and whether early life 25(OH)D is associated with sleep.
Methods: VIDI-study [Vitamin D Intervention in Infants] is a double-blind randomized clinical trial. Children received vitamin D_3 supplementation of 400-IU (n = 421) or 1200-IU (n = 424) daily until 24 months of age. Blood 25(OH)D was analyzed and parents completed questionnaires at 11.7 (SD = 0.7) and at 26.0 (SD = 3.0) months (Brief Infant Sleep Questionnaire, Sleep Disturbance Scale for Children). Maternal 25(OH)D was analyzed at mean 11.3 (SD = 2.2) gestational weeks. We used regression models adjusted for season of birth, breastfeeding, gestational age, parental education, and mother's age, body-mass-index, smoking and sleep.
Results: High-dose versus standard-dose supplementation was not associated with child's sleep up to 24 months. Children with 25(OH)D ≥ 75 nmol/L at 12 months had less sleep disturbances at 24 months (adjusted for all covariates: B = −1.46 points 95 % CI: 2.89; −0.04, p = 0.05), especially disorders of arousal (B = −0.40, 95 % CI: 0.64;-0.15, p = 0.002). We also found associations between maternal 25(OH)D ≥ 75 nmol/L during pregnancy and child's more favorable sleep characteristics.
Conclusion: We observed that high-dose versus standard-dose vitamin D_3 provides no benefits for child's sleep, even if we found an association between child's higher 25(OH)D and less sleep disturbances. Higher maternal 25 (OH)D during pregnancy was associated with child's better sleep, which should be studied further.

1. Introduction

Sleep problems are common in early childhood. More than one third of children experience short sleep duration [1], and one fifth suffer from sleep problems during early childhood [2]. Sleep problems often persist into adulthood [3] and are associated with adverse health outcomes, including higher blood pressure [4], and poorer cognitive development [5,6] and academic outcomes [7]. Sleep regulation originates in prenatal brain development [8–10] and matures most rapidly in the first two years of life [11]. While modifiable socioenvironmental factors, such as bedtime routines, influence sleep characteristics (e.g. sleep duration) [12,13] biological determinants persist as key contributors to

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its regulation [14]. Vitamin D, a steroid hormone with multifaceted functions, has been suggested to influence sleep regulation [15] Although the precise mechanisms remain unclear, vitamin D receptors are expressed in sleep related regions of the brainstem, subcortical structures and cortical areas [16–23]. Furthermore, the active circulating form of vitamin D, serum 25-hydroxyvitamin D (25(OH)D), has been reported to cross-regulate with melatonin, a neurohormone involved in circadian rhythms and sleep characteristics [24].

Recent observational studies suggest an association between low 25 (OH)D concentration and sleep disorders, problems and characteristics in pediatric populations [25–27]. Two of the studies included children under or at two years of age and reported cross-sectional associations between lower 25(OH)D concentration and shorter sleep duration [28, 29]. However, to date, no randomized trials have been conducted to examine the effects of vitamin D supplementation on sleep in childhood. Randomized controlled trials are needed to study the effects of vitamin D supplementation, as observed correlations between 25(OH)D and health outcomes do not prove causality [30,31].

We tested whether randomly assigned high-dose (1200-IU) versus standard-dose (400-IU) daily vitamin D_3 supplementation from 2 weeks to 24 months of age influenced sleep problems (sleep disturbances and poor sleep) and sleep characteristics (sleep duration, sleep latency, bedtime and nocturnal awakenings and wakefulness) at 12 and 24 months of age in children born healthy. To extend the existing literature, we examined whether child's 25(OH)D $\geq$ 75 nmol/L at 12 and 24 months of age, as well as maternal 25(OH)D $\geq$ 75 nmol/L during pregnancy — which, to our knowledge, has not been examined in previous studies — were associated with fewer sleep problems and more favorable sleep characteristics in early childhood.

2. Methods

2.1. Study design and procedure

The Vitamin D Intervention in Infants (VIDI) study is a double-blind, randomized clinical trial [32]. Children were randomized on a 1:1 basis to receive either 400-IU (10 μg) or 1200-IU (30 μg) of vitamin D_3 supplementation daily from 2 weeks to 24 months of age. Further information of the vitamin D_3 supplementation is given in Supplementary Appendix 1, and elsewhere [32]. Parents filled in questionnaires on child's sleep, health and family demographics at birth and at the follow-up visits. Children's blood samples were collected during follow-up visits at mean age of 11.7 (SD = 0.7) and 26.0 (SD = 3.0) months. In addition, hospital records were used to collect perinatal information on the mother and child. Maternal serum samples were collected during routine maternity clinic visits at a mean gestational age of 11.3 (SD = 2.2) weeks and stored in the Finnish Maternity Cohort serum bank organized by the Finnish Institute for Health and Welfare. Parents signed informed consent forms at baseline. The study was approved by the ethics committee at the Hospital District of Helsinki and Uusimaa and performed in line with the principles of the Declaration of Helsinki. The study is registered with ClinicalTrials.gov (NCT01723852). The study follows the Consolidated Standards of Reporting Trials (CONSORT) reporting guideline.

2.2. Study participants

Initially, 987 (492 female) healthy term-born children and their parents were recruited from Kätilöopisto Maternity Hospital in Helsinki, Finland between January 1, 2013, and June 30, 2014 [32]. The current study included 845 children who completed at least one of the follow-up visits at 12 and 24 months of age. Fig. 1 shows study enrollment, allocation, and follow-up. All mothers resided in Finland and self-reported

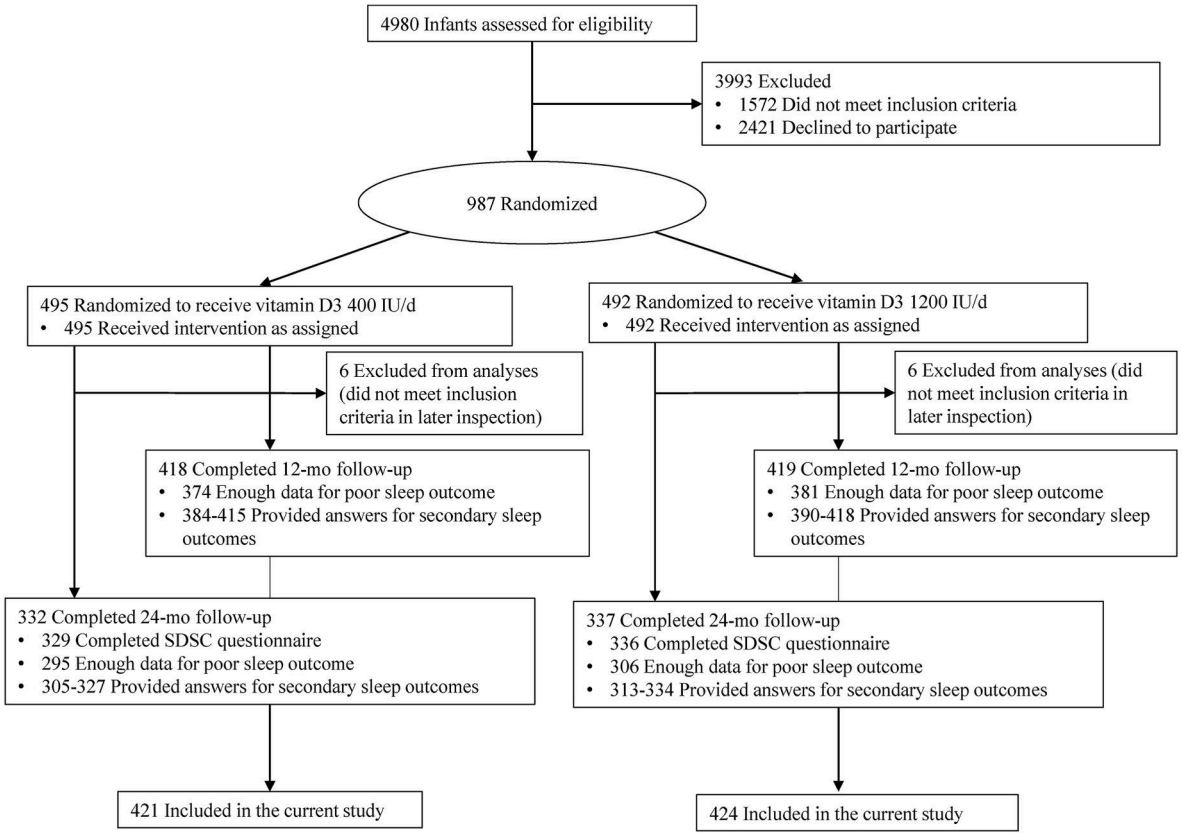

Fig. 1. Flowchart of study enrollment, allocation, and follow-up.

their skin color as white. Comparison of study participants against the attrition group are shown in Supplementary Table 1. In the analytic sample, parents had higher educational level, mothers smoked less and had higher 25(OH)D concentrations (all p-values<0.05) than in the attrition group.

2.3. Biochemical analysis

Blood 25(OH)D concentrations were analyzed at the Pediatric Research Centre, University of Helsinki. A fully automated IDS-iSYS immunoassay system with chemiluminescence detection (Immunodiagnostics System) was used. Details of biochemical analyses are described in Supplementary Appendix 2, and elsewhere [32].

2.4. Outcome and covariate measures

Brief Infant Sleep Questionnaire (BISQ) is a 13-item screening tool designed to assess sleep-related concerns in infants [33]. Parents filled out BISQ at 12- and 24-month follow-up. The items assess sleep duration, sleep latency (e.g., "how long does it take to put the child to sleep"), nocturnal wakefulness, frequency of awakenings, bedtime and parental concern. In addition, BISQ includes items about sleeping habits, such as items about sleeping arrangements and the preferred sleeping position.

We constructed a poor sleep -variable based on normative sleep data of a large Finnish sample separately at 12 and 24 months of age [2] as the primary outcome. Poor sleep was defined as belonging in the 10th highest percentile in awakening times during night or belonging in the 10th highest percentile in nocturnal wakefulness or total sleep time being at least two standard deviations less than the mean. At 12 months of age, poor sleep translates to the child waking up ≥ 4 times per night, nocturnal wakefulness being ≥45 min or the total sleep time being ≤10.6 h on average. At 24 months of age poor sleep translates to the child waking up ≥ 2 times per night, nocturnal wakefulness being ≥15min or the total sleep time being ≤10.1 h on average.

As secondary outcomes we used parent responses for individual items of the questionnaire. We used total sleep, night sleep, day sleep, sleep latency and bedtime. In addition, to improve the detection of sleep problems, we used the highest 10th percentile in nocturnal wakefulness and frequency of awakenings and named these variables problematic nocturnal wakefulness and problematic nocturnal awakenings. The parental concern -item was treated as a categorical variable (perceived problem vs. no perceived problem) and named perceived problem.

Sleep Disturbance Scale for Children (SDSC) is a well-established measure of sleep disorders, and it has been validated in 24 month-year-old children [34–36]. Parents filled out SDSC at 24 months follow-up. The items (e.g., "The child experiences daytime somnolence") are rated on a 5-point Likert scale (1 = never, 5 = always) with higher scores reflecting more disturbances. An overall (SDSC total score) and six indices can be derived from SDSC. The indices are Disorders of Initiating and Maintaining Sleep, Sleep Breathing Disorders, Disorders of Arousal, Sleep-Wake Transition Disorders, Disorders of Excessive Somnolence, and Sleep Hyperhidrosis.

As a primary sleep outcome, we used SDSC total score and as secondary sleep outcomes, the six SDSC indices. SDSC total score ranged from 26 to 65 and the overall mean score was 39.23. The internal consistency for SDSC was acceptable (Crohnbach's alpha for total score = 0.74). We imputed missing values using multiple imputations (n = 5) by chained equations with SDSC questionnaire data only and used pooled estimates in further analysis. Up to 20% missing values were allowed (n = 47).

As covariates, we used child's season of birth, breastfeeding duration, gestational age at birth, parental education and mother's age, smoking and body-mass-index (BMI), gestational week and season of the maternal blood sampling as well as mother's sleep problems and depressive symptoms. Supplementary Appendix 3 presents the details of the covariate measures and selection.

2.5. Statistical analysis

Statistical analysis and power calculations are detailed in Supplementary Appendix 4. Two-tailed independent samples *t*-test or Pearson χ^2-test were used in describing characteristics. We assessed differences between the supplementation groups and blood 25(OH)D < 75 nmol/L versus ≥75 nmol/L [37,38] using logistic and linear regression models (participants in 25(OH)D < 75 nmol/L group 36.7 %, 21.3 % and 16.7 % in the measurement points, respectively). We used threshold models with 75 nmol/L cutoff to provide clinically applicable information within vitamin D sufficient population. We also tested whether the child's sex moderated any of the potential associations with primary sleep outcomes by entering interactions term "sex × intervention group/25(OH)D" into the equations. All associations were examined using three models: Model 1 (unadjusted), Model 2 (adjusted for season of birth, parental education, breastfeeding, child's gestational age, and mother's age, smoking and BMI. Models with maternal 25(OH)D concentration during pregnancy adjusted also for gestational week and season of the blood sampling), and Model 3 (adjusted for Model 2 covariates and mother's sleep problems and depressive symptoms). We applied False Discovery Rate (FDR) correction (Benjamini-Hochberg –method) to control for type I error in secondary analyses of individual items of BISQ.

3. Results

3.1. Characteristics

Children in the 1200-IU D_3 supplementation group (n = 424) did not differ in baseline characteristics (p-values>0.05) from those in the 400-IU group (n = 421) (Table 1). The follow-up characteristics were also similar between intervention groups, except that children's 25(OH)D

Table 1
Baseline characteristics.

	400-IU group (n = 421) n (%)/mean(SD)	n	1200-IU group (n = 424) n (%)/mean(SD)	n	p[a]
Child					
Girls	213 (50.59)	421	214 (50.47)	424	0.97
Gestational age, days	280.70 (7.75)	421	281.70 (7.54)	424	0.06
Season of birth		421		424	0.95
Winter	85 (20.19)		79 (18.63)		
Spring	172 (40.86)		179 (42.22)		
Summer	91 (21.62)		93 (21.93)		
Autumn	73 (17.34)		73 (17.22)		
Mother					
Age	30.91 (4.16)	413	31.49 (4.52)	424	0.06
BMI	23.22 (3.70)	419	23.26 (3.62)	421	0.87
Mother's sleep problems (BNSQ total)	48.74 (8.81)	364	49.21 (8.70)	370	0.47
Mother's depressive symptoms (CES-D total)	12.13 (6.57)	364	11.99 (6.05)	375	0.76
25(OH)D during pregnancy	82.54 (22.33)	363	82.16 (17.93)	345	0.80
<50 nmol/L	15 (4.13)		10 (2.90)		
50–74 nmol/L	141 (38.84)		119 (34.49)		
≥75 nmol/L	207 (57.02)		216 (62.61)		

SD=Standard Deviation, 25(OH)D = 25-hydroxyvitamin D, BMI=Body-mass-index, BNSQ=Basic Nordic Sleep Questionnaire, CES-D = Center for Epidemiologic Studies Depression Scale.

[a] Group differences calculated with *t*-test or χ2 -test.

concentration was higher in the 1200-IU than in the 400-IU group, at 12 months and at 24 months (p-values<0.001) (Table 2). Children's 25(OH)D concentration <50 nmol/L was rare in both groups (400-IU: 1.3-2.1%, 1200-IU: 0.0%). The overall mean of SDSC total score was 39.2 (range: 26–65). At 12 months 18.0% and at 24 months 24.8% of children in our sample had poor sleep compared to age-specific normative data. See Supplementary Table 2 for characteristics of secondary sleep outcomes.

3.2. Vitamin D supplementation and child's sleep

Vitamin D_3 intervention groups did not differ in primary sleep outcomes, poor sleep or SDSC total score, at 12 or 24 months (all p-values >0.23, Table 3). Child's sex did not moderate the associations (sex × intervention group interaction p-values >0.38, Supplementary Table 3). Similarly, the intervention groups did not differ in secondary outcomes of BISQ (all pFDR-values >0.53, see Supplementary Table 4) or SDSC indices (all p-values >0.09, see Supplementary Table 5).

3.3. 25(OH)D concentration in early childhood and child's sleep

No associations were found between child's 25(OH)D concentration and primary sleep outcomes at 12 or at 24 months, except that child's 25(OH)D ≥ 75 nmol/L at 12 months was associated with lower SDSC total scores at 24 months in a fully adjusted model including mother's sleep (Model 3, p = 0.046) (Table 4). Child's sex did not moderate the association (sex × 25(OH)D interaction p-values >0.12, see Supplementary Table 3).

Table 2
Follow-up characteristics.

	400-IU group (n = 421) n (%)/mean(SD)	n	1200-IU group (n = 424) n (%)/mean(SD)	n	p[a]
12-month follow-up					
Child's 25(OH)D	82.94 (19.61)	388	115.21 (27.53)	398	**<0.001**
<50 nmol/L	8 (2.06)		0 (0.00)		
50–74 nmol/L	146 (37.63)		21 (5.28)		
≥75 nmol/L	234 (58.94)		377 (94.72)		
Poor sleep, yes	61 (16.31)	374	75 (19.69)	381	0.81
24-month follow-up					
Child's 25(OH)D	86.74 (19.64)	397	117.75 (26.25)	406	**<0.001**
<50 nmol/L	5 (1.26)		0 (0.00)		
50–74 nmol/L	115 (28.97)		19 (4.68)		
≥75 nmol/L	277 (69.77)		387 (95.32)		
Breastfed, months	10.51 (5.69)	413	10.86 (5.50)	419	0.38
Parental education level, high	334 (80.29)	416	354 (83.49)	424	0.23
Non-smokers	355 (85.34)	410	358 (85.65)	418	0.76
Poor sleep, yes	74 (25.08)	295	75 (24.51)	306	0.91
SDSC total score	39.40 (6.77)	329	39.06 (6.37)	336	0.51

SD=Standard Deviation, 25(OH)D = 25-hydroxyvitamin D, BMI=Body-mass-index, BNSQ=Basic Nordic Sleep Questionnaire, Poor sleep = in the 10th highest percentile in nocturnal awakening times or wakefulness or total sleep at least two standard deviations less than the mean, SDSC=Sleep Disturbance Scale for Children.

[a] Group differences calculated with *t*-test or $\chi 2$ -test.

Table 3
Associations between vitamin D_3 intervention group (400-IU vs 1200-IU) and sleep problems (poor sleep and SDSC total score).

	Model 1 (n = 601–755)		Model 2 (n = 601–755)		Model 3 (n = 524–658)	
	OR/B(95% CI)	p	OR/B(95% CI)	p	OR/B(95% CI)	p
Poor sleep at 12 mo	1.26 (0.87; 1.83)	0.23	1.23 (0.84; 1.80)	0.30	1.12 (0.74; 1.71)	0.59
Poor sleep at 24 mo	0.97 (0.67; 1.40)	0.87	1.00 (0.68; 1.46)	0.99	0.97 (0.64; 1.47)	0.88
SDSC total score at 24 mo	−0.34 (−1.34; 0.66)	0.51	−0.39 (−1.39; 0.61)	0.45	−0.38 (−1.42; 0.67)	0.48

Model 1: crude, Model 2: season of birth, parental education, breastfeeding duration, gestational age, mother's body-mass-index, mother's age, mother's smoking controlled, Model 3: Model 2 + mother's sleep problems and depressive symptoms controlled. OR in poor sleep outcomes. Bs in SDSC total score outcome, Bs are unstandardized. Poor sleep = in the 10th highest percentile in nocturnal awakening times or wakefulness or total sleep at least two standard deviations less than the mean, SDSC=Sleep Disturbance Scale for Children.

Table 4
Associations between child's 25(OH)D concentration (<75 nmol/L vs ≥ 75 nmol/L) and primary sleep outcomes (poor sleep at 12 and 24 months and SDSC total score at 24 months).

	Model 1		Model 2		Model 3	
	OR/B(95% CI)	p	OR/B(95% CI)	p	OR/B(95% CI)	p
Child's 25(OH)D concentration, 12 mo						
Poor sleep at 12 mo	1.13 (0.70; 1.83)	0.62	1.13 (0.69; 1.86)	0.63	1.01 (0.58; 1.77)	0.96
Poor sleep at 24 mo	0.93 (0.58; 1.49)	0.75	0.99 (0.60; 1.63)	0.98	0.85 (0.48; 1.49)	0.57
SDSC total score at 24 mo	−1.22 (−2.51; 0.08)	0.07	−1.12 (−2.44; 0.20)	0.10	−1.44 (−2.85; −0.03)	**0.046**
Child's 25(OH)D concentration, 24 mo						
Poor sleep at 24 mo	0.79 (0.48; 1.29)	0.34	0.79 (0.47; 1.30)	0.35	0.77 (0.44; 1.34)	0.35
SDSC total score at 24 mo	−0.33 (−1.72; 1.06)	0.64	−0.33 (−1.74; 1.08)	0.65	−0.18 (−1.66; 1.30)	0.82

Model 1: crude, Model 2: season of birth, parental education, breastfeeding duration, gestational age, mother's body-mass-index, mother's age, mother's smoking controlled, Model 3: Model 2 + mother's sleep problems controlled. Bs in SDSC total score outcome, Bs are unstandardized. Poor sleep = in the 10th highest percentile in nocturnal awakening times or wakefulness or total sleep at least two standard deviations less than the mean, SDSC=Sleep Disturbance Scale for Children.

Child's 25(OH)D concentration at 12 or 24 months of age was not associated with any BISQ items (pFDR-values >0.16) (Supplementary Table 6).

Child's 25(OH)D concentration at 12 or 24 months was not significantly associated with SDSC indices (p-values >0.05), with one exception. Child's 25(OH)D ≥ 75 nmol/L at 12 months was associated with lower Disorders of Arousal -index score at 24 months (Model 2, B = −0.30, 95% CI: 0.52; −0.08, p = 0.009 and Model 3, p = 0.002, see Supplementary Fig. 1 and Supplementary Table 7).

3.4. Maternal 25(OH)D concentration during pregnancy and child's sleep

No associations were found between maternal 25(OH)D concentration during pregnancy and primary sleep outcomes, i.e., poor sleep or

SDSC total score, at 12 or at 24 months (Table 5). Child's sex did not moderate the association (sex × 25(OH)D interaction p-values >0.12, see Supplementary Table 3).

The associations between maternal 25(OH)D concentrations during pregnancy and BISQ items are presented in Supplementary Table 6. After Model 2 adjustments maternal 25(OH)D ≥ 75 nmol/L during pregnancy was associated with child's longer total (B = 20.53 min, 95 % CI: 10.12; 30.94, pFDR<0.001), night (B = 11.22 95 % CI: 1.93; 20.50, pFDR = 0.03), and day sleeping time (B = 9.62, 95 % CI: 3.14; 16.09, pFDR = 0.01), and with shorter sleep latency (B = −2.99 min, 95 % CI: 4.94; −1.04, pFDR = 0.01) and earlier bedtime (B = −9.13 min, 95 % CI: 16.84; −1.41, pFDR = 0.03) at 12 months. Higher maternal 25(OH)D during pregnancy was also associated with a lower risk of perceived problems in child's sleep at 12 months (Model 2: OR = 0.67, 95 % CI: 0.48; 0.94, pFDR = 0.03). These results remained significant, after further adjustment for mother's sleep problems (Model 3). In addition, maternal 25(OH)D ≥ 75 nmol/L was significantly associated with a lower risk of problematic nocturnal awakenings in the model adjusting for mother's sleep (Model 3: OR = 0.37, 95% CI: 0.16; 0.87, pFDR = 0.03). Problematic nocturnal wakefulness was the only 12-month BISQ item not associated with maternal 25(OH)D concentration during pregnancy (pFDR-values in all models>0.12).

At 24 months, after Model 2 adjustments, maternal 25(OH)D ≥ 75 nmol/L during pregnancy was also associated with child's longer total (B = 13.12, 95% CI: 3.57; 22.68, pFDR = 0.03) and night sleeping time (B = 10.97, 95% CI: 2.61; 19.33, pFDR = 0.03) and shorter sleep latency (B = −4.91, 95% CI: 8.13;-1.68, pFDR = 0.02). In models further adjusting for mother's sleep problems (Model 3) FDR corrections attenuated the associations to non-significant (pFDR>0.10).

The associations between 25(OH)D concentrations and SDSC indices are presented in Supplementary Table 7. Maternal 25(OH)D ≥ 75 nmol/L during pregnancy was associated with Disorders of Initiating and Maintaining Sleep (B = −0.56, 95% CI: 1.10;-0.02, p = 0.04) in Model 2 but the association was not significant in Model 3 (p = 0.08). There were no other associations between maternal 25(OH)D concentration during pregnancy and SDSC indices (p-values >0.35).

4. Discussion

In this randomized clinical trial of 845 healthy term-born children, we found that high-dose (1200-IU) versus standard-dose (400-IU) daily vitamin D_3 did not affect any of our primary– i.e., poor sleep and sleep disturbance total score – or secondary sleep outcomes up to 24 months of age. However, when examining the role of child's blood 25(OH)D concentrations, we found that children with 25(OH)D ≥ 75 nmol/L at 12 months had less sleep disturbances and especially disorders of arousal at 24 months of age. Moreover, we also found that even if maternal 25(OH)D ≥ 75 nmol/L during pregnancy was not systematically associated with primary sleep outcomes, it was associated with child's longer sleep duration (day, night and total), shorter sleep latency, earlier bedtime and a lower risk of problematic nocturnal awakenings or parental perception of child's problematic sleep at 12 months of age. The associations between maternal 25(OH)D concentration during pregnancy and the child's longer sleep duration and shorter sleep latency were apparent also at 24 months of age.

Table 5
Associations between maternal 25(OH)D concentration (<75 nmol/L vs ≥ 75 nmol/L) during pregnancy and primary sleep outcomes (poor sleep at 12 and 24 months and SDSC total score at 24 months).

	Model 1		Model 2		Model 3	
	OR/B(95% CI)	p	OR/B(95% CI)	p	OR/B(95% CI)	p
Maternal 25(OH)D concentration during pregnancy						
Poor sleep at 12 mo	0.83 (0.55; 1.27)	0.39	0.73 (0.47; 1.14)	0.17	0.64 (0.39; 1.05)	0.08
Poor sleep at 24 mo	0.93 (0.61; 1.42)	0.73	0.77 (0.49; 1.21)	0.26	0.92 (0.56; 1.52)	0.75
SDSC total score at 24 mo	−0.45 (−1.56; 0.67)	0.43	−0.62 (−1.77; 0.52)	0.29	−0.39 (−1.58; 0.81)	0.53

Model 1: crude, Model 2: season of birth, parental education, breastfeeding duration, gestational age, mother's body-mass-index, mother's age, mother's smoking and gestational week and season of the blood sampling controlled, Model 3: Model 2 + mother's sleep problems and depressive symptoms controlled. Bs in SDSC total score outcome, Bs are unstandardized. Poor sleep = in the 10th highest percentile in nocturnal awakening times or wakefulness or total sleep at least two standard deviations less than the mean, SDSC=Sleep Disturbance Scale for Children.

To our knowledge this is the first randomized trial evaluating the impact of higher-versus-standard vitamin D_3 supplementation on sleep in childhood. Our results of no differences in vitamin D_3 intervention groups in sleep problems or characteristics are partly in line with trials conducted in adult populations [39]. A recent systematic review [39] concluded that among adults vitamin D_3 supplementation might potentially improve sleep quality (e.g. nocturnal wakefulness), but the evidence on its effect on sleep quantity and disorders is scarce and contradictory.

The found association between child's 25(OH)D concentration at 12 months and less total sleep disturbances at 24 months of age is in line with some earlier observational studies in children [25–27]. Our finding was driven by the disorders of arousal index including child's sleep-walking, nightmares, nighttime screaming and confused arousal, occurring during partial arousal from non-rapid-eye-movement sleep [34]. This association was observed from the concentrations at 12 months but not at 24 months, suggesting that 25(OH)D may play a more critical role in development of sleep regulation during the first year, a period of rapid brain maturation. Further, disorders of arousal have been shown to be associated with emotional and behavioral problems [40], which are also linked to lower blood 25(OH)D concentrations [41], suggesting vitamin D as a potential shared etiological factor.

We did not find associations between child's 25(OH)D and poor sleep or sleep characteristics and thus could not replicate the previously reported significant association between lower 25(OH)D concentrations and shorter sleep duration in early childhood [28,29]. In our study, all children received vitamin D_3 supplementation and only approximately 1% of children had blood 25(OH)D concentration under 50 nmol/L at 12 and 24 months of age. Previous significant associations were found in populations including approximately 30% children with 25(OH)D concentrations <50 nmol/L [28,29] limiting the comparability of the results. It is possible that the association with sleep would be more evident in lower 25(OH)D concentrations. However, also in other earlier studies mainly small effect sizes of the association between 25(OH)D and sleep disorders and characteristics are reported [25].

Although we did not find any vitamin D_3 supplementation effects, we found correlation between blood 25(OH)D concentration and sleep disorders. Thus, our results indicate biomarker associations but do not provide causal evidence. The results might reflect presence of another factor contributing to both vitamin D metabolism and sleep problems, such as healthier lifestyle patterns, or a reverse causality so that sleep problems would affect vitamin D metabolism [29,42]. As no supplementation effect with higher-versus-standard vitamin D_3 supplementation was observed in this vitamin D sufficient population, the potential benefits of vitamin D_3 supplementation could be limited to individuals with 25(OH)D concentration <50 nmol/L.

It is important to note that vitamin D is important for normal brain development, including regions involved in sleep regulation, starting from the fetal period [9,10]. In early pregnancy metabolism of vitamin D changes substantially and maternal 25(OH)D concentrations tend to increase [43–46]. Placenta actively takes up and metabolizes maternal blood 25(OH)D [46] - the only vitamin D source for the fetus. Yet, to our knowledge our study is the first to examine maternal 25(OH)D concentrations during early/mid pregnancy and child's sleep problems and

characteristics. Our findings suggest that maternal 25(OH)D concentrations during pregnancy may shape the development of the offspring sleep regulation up to 24 months of age and are in line with earlier results showing an association between low cord blood 25(OH)D and child's shorter sleep duration [47]. However, the specific underlying mechanism remains unclear and there is yet no evidence of the causality of the association.

Strengths in our study include the double-blind randomized clinical trial design, well-characterized sample, and use of standardized and validated questionnaires [32]. However, there are limitations. We assessed sleep outcomes with parent-reported questionnaires, and in all sleep outcomes they are not interchangeable to more objective assessment methods, such as actigraphy [48], even if they are highly correlated [49]. Subjective assessment methods may be subject to reporting bias and insufficiently sensitive to detect small effects [48]. In addition, although one of the questionnaires (SDSC) was not originally designed to assess sleeping disorders in 24-month-old children, it has been validated in this age group [35,36]. We were able to follow 85.6% of the participants of the original sample (67.8% remaining up to the 24 months' follow-up), but the participating mothers were more often non-smokers, had higher 25(OH)D concentration and the participating parents had higher education than non-participants. Hence, a possible selection bias may have influenced particularly the findings of the observational analyses of 25(OH)D concentrations. The observational analyses of maternal and child's 25(OH)D concentrations are also subject to residual confounding of factors such as diet quality [50], physical activity [51], outdoor exposure [52], parenting practices [53] or chronic conditions (e.g. asthma [54]) and reverse causality, i.e. sleep affecting lifestyle behaviors that influence 25(OH)D concentration. Furthermore, our sample had only few participants with 25(OH)D concentration <50 nmol/L, which might have limited the power of our analysis. However, in turn, we were able to test associations with sleep problems and characteristics in a vitamin D sufficient cohort [55]. Our results also show that the association between child's 25(OH)D concentration and sleep problems is evident even with the 75 nmol/L cutoff for 25(OH)D concentration. In addition, the generalizability of the results might be affected by national food fortification as part of the public health efforts in Finland [56,57] and high ethnic homogeneity in our sample. However, recent studies suggest that there is no moderating effect of skin color in the association between vitamin D and health outcomes, even if there are lower rates of 25(OH)D concentration <50 nmol/L in white populations compared to more diverse populations [58,59].

5. Conclusion

In this randomized clinical trial of 845 healthy term-born children, we found that raising daily vitamin D_3 within an already sufficient range, high-dose (1200-IU) versus standard-dose (400-IU), provides no benefits for child's sleep problems or characteristics up to 2 years of age. However, we found that early childhood 25(OH)D concentrations were associated with children's sleep disorders. Maternal 25(OH)D concentrations during pregnancy were also associated with children's sleep characteristics pointing to a possible role of vitamin D during fetal development. The found biomarker associations do not provide causal evidence. In future, studies in diverse settings are still needed to examine the effect of vitamin D_3 on sleep in early childhood.

CRediT authorship contribution statement

Vilja Seppälä: Writing – review & editing, Writing – original draft, Formal analysis, Conceptualization. **Elisa Holmlund-Suila:** Writing – review & editing, Project administration, Investigation. **Helena Hauta-alus:** Writing – review & editing, Data curation. **Samuel Sandboge:** Writing – review & editing. **Eero Kajantie:** Writing – review & editing, Investigation. **Jenni Rosendahl:** Data curation. **Maria Enlund-Cerullo:** Data curation. **Saara Valkama:** Data curation. **Outi Mäkitie:** Writing – review & editing, Project administration, Investigation. **Sture Andersson:** Writing – review & editing, Project administration, Investigation. **Katri Räikkönen:** Writing – review & editing, Supervision. **Kati Heinonen:** Writing – review & editing, Supervision, Conceptualization.

Contributors statement

Vilja Seppälä contributed to conceptualization of the study, conducted the analyses, drafted the initial manuscript and revised the manuscript. Elisa Holmlund-Suila, Eero Kajantie, Outi Mäkitie and Sture Andersson contributed to the study design and reviewed and edited the text. Jenni Rosendahl, Maria Enlund-Cerullo and Saara Valkama contributed to the study design and data collection. Helena Hauta-alus contributed to data cleaning and reviewed and edited the text. Samuel Sandboge reviewed and edited the text. Katri Räikkönen reviewed and edited the text and supervised this study. Kati Heinonen contributed to the conceptualization of the study, supervised this study and reviewed and edited the text. All authors approved the final manuscript as submitted and agree to be accountable for all aspects of the work.

Data sharing

The data and codes supporting the findings are available on request from the corresponding author. The data are not publicly available due to privacy restrictions. Data requests may be subject to review by the Finnish national register authorities and ethical committees.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.sleep.2026.108913.

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