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Original Research Article

Vitamin D and sleep duration: a possible link

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ABSTRACT

Background: Normal-range sleep duration is an important factor for general health and metabolism. Insufficient or excessive sleep duration is associated with various chronic physical or mental illness, such as obesity, hypertension, diabetes, metabolic syndrome, depression, and other psychiatric diseases. Sleep duration is mainly affected by circadian rhythm, and circadian rhythm is affected by the melatonin level controlled by the light exposure. Therefore, sun exposure has an important role for determining sleep time. Aim and objective was to find an association between self-reported sleep duration with serum vitamin D levels in healthy adults.
Methods: A cross sectional study was carried out on 150 first professional MBBS students. They were asked about their sleep duration via a questionnaire and their morning samples of serum vitamin D was taken. All the data was put in SPSS software and analyzed.
Results: There was a definite positive correlation between sleep duration and the concentration of 25(OH)D in this study ($r=0.11$, $p<0.05$).
Conclusions: The present study revealed that vitamin D [25(OH)D] deficiency was an independent predictor of insufficient sleep among healthy adults.

Keywords: Sleep duration, Vitamin D

INTRODUCTION

Sleep is a complex physiological state, and although often thought as an inactivity period, it comprises a period of intense metabolic activity. Its function is still not fully understood, although it is known that this biological phenomenon participates in several processes, e.g., learning, consolidation of memory, inflammation, and metabolic disorders, being essential to good mental and physical health.[1]

Sleep, much like diet and physical activity, is a critical indicator of health and well-being in children and adolescents. Lack of sleep or short sleep duration may increase the risk of obesity, cardiovascular disease, diabetes mellitus, total mortality and other unhealthy conditions. Nowadays, insufficient sleep is a public health problem in children and adolescents worldwide.[2]

The recent interest in the role of vitamin D as a sleep modulator is based on studies in both animal models and humans, indicating the neuronal expression of CYP27B1 and VDR in different brain areas, especially in regions that also regulate the sleep-wake cycle, such as the hypothalamus.[3] Vitamin D is a steroid hormone which is mainly synthesized in the body through ultraviolet B (UVB) exposure on the skin or taken orally through food and/or supplements. Later studies revealed a much broader protective role of vitamin D in diseases related to the immune system, such as influenza, respiratory tract infections, cancer, and autoimmune diseases, as well as cardiovascular diseases.[4] In addition to these effects on physical health, there is also evidence that vitamin D status impacts cognitive, behavioural, and mood disorders.

In addition to increased time spent indoors, wearing clothes that cover most of the skin surface and/or

using sunscreen contribute to low levels of vitamin D in the general population. Moreover, at higher latitudes and during winter, vitamin D synthesis through sunlight exposure is almost impossible due to the large solar zenith angle which leads to absorption of UVB radiation in the ozone layer.

Recently, it has been proposed that vitamin D plays an important role in serotonin and melatonin regulation, which further indicates the relevance of vitamin D in mental health, especially the regulation of mood and sleep.[5]

Vitamin D is an essential micronutrient during the growth and development of the human body, and 25-hydroxyvitamin D [25(OH)D] is the most sensitive clinical marker for vitamin D status.

Pineal gland converts serotonin produced via TPH1 expression into melatonin during the evening and night time. The pineal gland, although located in the brain, belongs to these peripheral tissues as this is a circumventricular organ (i.e., lying outside of the blood-brain barrier).[6] During the evening and night time, it is suggested that TPH1 expression in the pineal gland is increased when serum 25(OH)D levels [as well as $1,25(OH)_2D$ levels] decrease, promoting serotonin production which can then be converted into melatonin. This process of increased peripheral TPH1 expression during the evening and night, stimulating melatonin production, and increased TPH2 expression during daytime enabling sufficient serotonin production in the brain, is optimal when serum 25(OH)D and $1,25(OH)_2D$ levels follow a 24-h pattern with higher levels during the day and lower levels in the evening and night. Research showed that both 25(OH)D as well as the active form $1,25(OH)_2D$ (measured every 2 hour) follow a 24-hour pattern with highest levels during midday and then decreasing to lower levels in the evening and night.[7,8]

Thus, the objective of our study was to find an association between self-reported sleep duration with serum vitamin D levels in healthy adults.

METHODS

A medical institution-based cross-sectional study was conducted among MBBS students in Hind institute of medical sciences, Sitapur, during the period November 2022-January 2023 after approval by the institutional ethics committee.

The study population consisting of 150 MBBS students aged 17-28 years were enrolled for the study during the academic years 2022 and 2023. Prior consent of subjects was taken into account. All participants were in apparently good health. Exclusion criteria included hepatic or renal disease, ongoing use of anti-lipemic medications or growth hormone and any other medicine that could influence vitamin D status. After taking verbal consent and explaining the purpose of the study, self-reported sleep duration was recorded. Sleep duration was assessed by following question "On an average morning, what time do you get up?", "On an average evening, what time do you go to bed?" Accordingly sleep duration was calculated. Subjects were also asked about any physical activity they are engaged in (e.g., football, volleyball, cricket, badminton or any yoga). Then the participants were divided into three groups according to the sleep duration, first group having sleep duration <5 hours, second group having sleep duration 6-8 hours and the third one having duration >8 hours.

Serum sample collection (5 ml) was performed between 7:30 a.m. and 9:00 a.m. by a trained technician after 12 hours of fasting. Samples were allowed to clot and then were centrifuged, separated and stored until they were tested.

Serum 25(OH)D was measured by electrochemiluminescence immunoassay (ECLIA) method (Cobas E 411, India). Cut-off values for serum 25(OH)D levels include: >20 ng/ml for vitamin D sufficiency, and ≤20 ng/ml for insufficiency and deficiency. All of the intra- and inter-assay test coefficients of variation were <5%.

Statistical methods

The data were coded and entered in Microsoft Excel and analyzed using SPSS version 16. Continuous variables were summarized as means (standard deviations), and categorical variables were summarized as percentages. One way analysis of variance was used for continuous variable, whereas categorical variables were analyzed using a chi-square test. The relationship between 25(OH)D levels and sleep duration (dependent variable) was evaluated with multivariate logistic regression model. Statistical significance was set at $\alpha=0.05$.

RESULTS

The general characteristics of the study participants are shown in Table 1.

Table 1: Characteristics of the study subjects.

Variables	
Age (years) mean (SD)	11.15 (1.91)
Gender, n (%)	
Male	436 (54.50)
Female	364 (45.50)
Bedtimes, mean (SD)	21:12 (0:41)
Wake times, mean (SD)	6:23 (0:36)
Sleep duration, mean (SD) (hours)	9.17 (0.97)
25(OH)D, mean (SD) (ng/ml)	22.4 (6.0)

SD- standard deviation; data are presented as mean (SD), or numbers (%).

Of the 150 subjects included in our study, the mean age of the participants was 20.15±3.91 years, and 52.67% of participants were boys. The mean sleep duration of the subjects was 7.17±0.97 h, the mean serum 25(OH)D concentration was 22.38±6.03 ng/ml.

Table 2 shows the characteristics of the study participants according to sleep duration category. Nearly 1/3 (32.8%) of the study subjects were sleep insufficient (sleep duration <5 hours per day), 30.3% of the study subjects were vitamin D insufficient and deficient (serum 25(OH)D level ≤20 ng/ml). There were significant differences among three groups in age and 25(OH)D levels (p<0.001). No difference in gender or physical activities were found.

Table 2: Characteristics of the study subjects by sleep duration (n=150).

Variables	Sleep duration (hours)			P value
	<5	6-8	>8	
	N=19	N=116	N=15	
Gender Male n (%)	3 (20)	112 (91.05)	8 (66.67)	0.070
Bed times	21:45 (0:39)	21:15(0:23)	20:35 (0:36)	<0.001
Wake times	5:35 (0:24)	6:35 (0:25)	07:15 (0:36)	<0.001
25-OH-D mean (SD) (ng/ml)	20.8 (5.7)	25.4(5.7)	24.8(6.4)	<0.001
25(OH)D status, n%				
Sufficiency (>20 ng/ml)	5 (26.32)	97 (83.62)	8 (53.33)	<0.001
Deficiency/insufficiency (≤20 ng/ml)	14 (73.68)	19 (16.38)	7 (46.67)	
Physical activity n%	35 (25)	90 (77.59)	15 (10.71)	0.74

The relationship between vitamin D levels and sleep duration was examined by multivariate logistic regression analyses (Table 3). Insufficiency and deficiency of the vitamin [25(OH)D≤20 ng/ml] was significantly associated with the increased probability of insufficient sleep (95% CI =1.13-2.47).

Table 3: Odds ratio and 95% confidence interval for insufficient sleep (<5 hours/day) by categories of 25(OH)D levels.

	25-(OH) vitamin D levels		P value
Sleep duration insufficiency	≤20 ng/ml	>20 ng/ml	
<5 hours/day	1.67 (1.13-2.47)	1	0.009

DISCUSSION

To date, the mechanisms for a link between sleep duration and vitamin D are not well understood. There might be some possible reasons for explaining the relationship between vitamin D status and sleep duration. For instance, lower 25(OH)D levels might lead to sleep disorders through affecting sleep regulating substance, such as melatonin, tumor necrosis factor alpha (TNF-α), interleukin-1 (IL-1) and prostaglandin D2 (PD2).[9] Another possible explanation is that vitamin D receptors have been found in specific regions in the central nervous system involved in regulating sleep, which include the anterior and posterior hypothalamus, the raphe nuclei, the midbrain central grey, and the nucleus reticularis pontis caudalis and oralis.[10] This explanation was supported by some intervention studies in humans, which found improved sleep duration and sleep latency with higher levels of vitamin D supplementation.[11-19] However, sleep disorders might be linked to changes in dietary habits and outdoor activity's patterns, which could affect 25(OH)D levels. Thus, these relationships between sleep duration and 25(OH)D levels may be bidirectional.[20]

In our study we found direct association between sleep duration and serum vitamin D levels. Same findings were reported by Bertisch et al who did their study on 1721 adults and found that vitamin D deficient participants had the shortest sleep duration.[3]

Same findings were seen by Kim et al who did their study on 1614 elderly adults and divided them among 4 groups according to sleep duration and subsequently reported that groups having longer sleep duration were having significantly higher vitamin D levels as compared to the group with shorter sleep duration.[4]

In contrast to our findings, Liu et al found that both long and short sleep duration was associated with vitamin D deficiency in older Chinese women.[5] Almost same findings were found by Choi et al who stated that differences in log-transformed vitamin D status between the sleep insufficient or excessive sleep groups and the normal range sleep groups were not significant.[6]

Though Abboud et al did systematic review and included 19 studies where they got the other way round and found that vitamin D status is promising in improving sleep quality.[8] Muscogiuri et al also found a positive association between the two.[10] In our study also, we found that insufficiency and deficiency of the

vitamin [25(OH)D≤20 ng/ml] was significantly associated with the increased probability of insufficient sleep taken as <5 hours/day (95%CI =1.13-2.47).

The limitation of our study was that we took only one batch of 150 subjects, so the sample size was small. More studies with larger sample size would be indicative of the result.

CONCLUSION

The present study revealed that vitamin D [25(OH)D] deficiency was an independent predictor of insufficient sleep among the participants. The results implied that adequate vitamin D in healthy young adults might curb sleep insufficiency problems. As the present study only highlighted the association between vitamin D deficiency and insufficient sleep duration, prospective studies and randomized controlled trials are required to establish the causality between serum vitamin D and sleep outcomes.

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