Associations between serum 25-hydroxyvitamin D and sleep, as estimated by actigraphy and the Pittsburgh Sleep Quality Index (PSQI)

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It is unknown whether vitamin D status affects sleep health, but recent studies suggest vitamin D deficiency is associated with shorter sleep duration¹ and lower sleep efficiency². This study investigated whether there is a relationship between vitamin D status and sleep-wake cycles in UK dwelling South Asian (SA) and Caucasian (C) women, using ambulatory actigraphic data and self-reported sleep quality data from the D-FINES II (Vitamin D, Food Intake, Nutrition and Exposure to Sunlight in Southern England II) study. In June-August 2010, serum 25-hydroxyvitamin D [25(OH)D] and data on self-reported musculoskeletal pain were collected from n = 47 women. In September-October 2010, participants wore Actiwatch-L (AWL, Cambridge Neurotechnology) monitors on their wrists for 24 h/day, over 14 consecutive days to measure sleep-wake activity as well as completing the PSQI (self-reported sleep quality) once. A subset of n = 37 women also wore an AWL on a neckband during the daylight hours to measure environmental light exposure. Each subject’s actigraphic data (including light exposure) were eligible to be included in the statistical analysis if they had ≥ 7 days of valid data and a 25(OH)D measurement. Relationships between 25(OH)D and actigraphic measures were analysed by Pearson’s bivariate correlations, as well as by partial correlations to control for potential confounders. PSQI scores are ordinal data so relationships were analysed by Spearman’s correlations only.

There was a significant negative relationship between 25(OH)D concentration and actigraphic sleep latency in SA (r = −0.562, P = 0.036), and a significant positive relationship between 25(OH)D and both overall PSQI score (r = 0.385 P = 0.047) and PSQI sleep latency subscale (r = 0.439, P = 0.02) in C (see Table). Partial correlations controlling for bone pain (n = 23 C, n = 11 SA) found a statistically significant positive relationship between 25(OH)D and actigraphic sleep latency (r = 0.426, P = 0.048, n = 23) in C only. However, when adjusting for muscle pain (n = 21 C, n = 8 SA), there were no significant associations between 25(OH)D and actigraphic sleep parameters in either ethnic group (P > 0.05). Finally, there were no significant correlations between 25(OH)D and actigraphic sleep parameters when adjusting for outdoor light exposure (mins/d > 1000 lux) (P > 0.05, n = 20 C, n = 8 SA).

These findings suggest that higher 25(OH)D levels are associated with shorter actigraphic sleep latency in SA, but not when controlling for the confounders of musculoskeletal pain or light exposure. In C, it is unclear why controlling for bone pain leads to the appearance of a relationship between 25(OH)D and sleep latency, and why participants with worse self-reported sleep (using PSQI) have better 25(OH)D status. These results are not easy to interpret, but suggest a potential mediating effect of ethnicity, musculoskeletal pain and light exposure in the relationship between 25(OH)D and both sleep latency and self-reported sleep quality. This interpretation is supported by previous studies showing a link between sleep problems and musculoskeletal pain³ and the fact that daytime light exposure is important to sleep health. Actigraphy, however, has known limitations in measuring sleep latency, and the number of subjects with valid light data were small. Further research in this area is warranted.

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