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The Association of Body Mass Index and Serum Vitamin D level with Depression

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ABSTRACT

Background: Emerging evidence suggests that vitamin D deficiency and obesity may contribute to the pathophysiology of depression through neuroendocrine and inflammatory mechanisms.

Objective: To compare serum 25-hydroxyvitamin D and body mass index between patients with depression and healthy controls, and to assess their association with depression severity.

Subjects and Methods: A case-control study conducted at Al-Hakeem Hospital, Al-Najaf, Iraq, from February to August 2025. Included 120 participants; 70 patients diagnosed with depression and 50 age- and BMI-matched healthy controls. Serum 25(OH)D levels were measured using the Hipro AFS-1 immunoassay system. Statistical analysis was performed using SPSS v26, with $p < 0.05$ considered significant.

Results: A significant lower median value of serum 25(OH)D levels in patients with depression, compared to healthy controls; 17.50 vs. 22 ng/ml, respectively, (p =0.001). No significant differences in mean age and BMI values between patients and controls ($p > 0.05$). The median 25(OH)D level was significantly lower in patients with moderate and severe depression compared with both controls (p = 0.0043 and p = 0.0001, respectively) and the mild group (p = 0.0007 and p = 0.0001, respectively). The severe depression group showed the lowest median vitamin D levels, significantly lower than the moderate group (p = 0.0099). In contrast, the mild depression group did not differ significantly from controls. Correlation analysis revealed significant negative correlation between age and 25(OH)D levels in the mild group (r = –0.519, p = 0.0093) and significant negative correlation between BMI and 25(OH)D levels in the severe depression group (r = –0.473, p = 0.0263).

Conclusion: Depression is strongly associated with reduced serum vitamin D levels, and this association is more pronounced with increasing severity of depression.

Keywords: Obesity, Vitamin D, deficiency, Depression, Severity

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INTRODUCTION:

Depression is a leading cause of impairment and disability globally [1] and a major contributor to suicidal behavior. The prevalence of depression in Iraq is 3.6 % [3]. Epidemiological studies indicated that depression is two times in women than men. It involves depressed mood or loss of pleasure or interested in activity for long period of time. The main symptoms of depression include persistent sad, anxious, or "empty" mood, feelings of hopelessness, pessimism irritability, frustration, or restlessness, guilt, worthlessness, or helplessness, additionally depression can presented as loss of interest or pleasure in hobbies and activities, fatigue, lack of energy, or feeling slowed down, difficulty concentrating, remembering, or making decisions, difficulty sleeping, waking too early in the morning, or oversleeping and changes in appetite or unplanned weight changes [6].

There are several risk factors that may contribute to the development of depression; mostly psychological factors that associated with alcohol consumption,, tobacco smoking, maternal stress, low birth weight, physical inactivity, unhealthy eating style and low social support [7].

Depression is classified according to beck depression inventory- II (BDI-II) into mild, moderate and severe. Beck depression inventory-II is 21 self-reported inventory items used to assess severity of depression in adult. Each item consists of four statement (scored from 0 to 3) with a total score of 63. The total score determines the severity of depression into; where a score of 17-20 considered as mild depression, 21-30 moderate and 31-40 as severe depression [8].

The pathophysiology of depression is not well recognized, however, thirty years ago, a major hypothesis was formulated about development of depression, this hypothesis proposed that functional deficiency of the brain monoaminergic transmitters norepinephrine, serotonin and /or dopamine in the brain could be associated with the symptoms of depression while mania can be contributed to the excessive monoamines at the critical synapses. However, both the behavioral and functional abnormalities of depression or mania may occur as a result of changes in the synthesis, release or storage of these neurotransmitters in addition to the disturbances in the sensitivity of the receptors to these transmitters or abnormal function of the subcellular messengers [9].

Psychologically, depression may arise due to an interaction between psychological and sociologic factors such as life events like. Nonetheless, environmental factors often play a crucial role in depression. Moreover, other risk factors can enhance the development of depressive disorders such as loss of job, for instance, a person out of work has higher likelihood of having depression in about three times compared to those in employed in a work [10]. Nonetheless, the predisposing of a person to depression is multifactorial and no unique personality factors, type or trait that can be blamed. From other point of view, depression may affect any person of any gender at any age regardless the pattern of personality. Furthermore, certain personality disorders may contribute to the development of depression such as borderline and comorbid personality disorders where these patients at higher risk of developing depression compared to those with other psychological disorders like antisocial or paranoid personality disorders [10]. Regarding the treatment of mood disorders is mainly the duty of a psychiatrist. Antidepressant are currently available as a treatment of incident depression as well as a prophylactic to prevent depression[10].

Obesity is regarded as a global pandemic, or syndemic as it entails potentially disastrous consequences for human being [11]. Several factors and causes are contributed to increase snacking, grazing and loss of regular meals, increase energy dense 'added value ' food, increase affluence, increase car ownership, decrease walking to school and work, decrease sport in school, increase central heating, using washing machines, increase time spent on computer games and watching TV [11]. The world Health Organization classifies the body mass index (BMI) into 6 categories, where BMI of < 18.5 kg/m^{2}classified as underweight, 18.5-24.9 kg/m^2 normal, 25-29.9 kg/m^2 overweight and a BMI of ≥ 30 kg/m^2 as obesity which further classified as obesity grade I (BMI: 30-34.9 kg/m^2), grade II (BMI: 35-39.9 kg/m^2) and Grade III, morbid obesity (BMI (≥ 40 kg/m^2).

There is no doubt that anxiety and depression impose a significant negative effect on the individuals' health. The growing scientific researches as well as clinical studies showed varying degree of depressive symptoms in obese individuals. An intercorrelation between obesity and depressive disorders has been proposed by many authors[12]. This correlation can be attributed to physiological maladaptation [13]. Several studies and meta-analyses documented bidirectional and complicated multifactorial association between obesity and mood variation[14]. According to these studies, the likelihood of having depression is in obese individual is high by almost 55% in both men and women. While depressed individuals had almost 58% higher risk of developing obesity which reflects the inter-relation between these two disorders; obesity and depression [14,15], this is from one side, from the other side, there is a strong evidence that there is growing evidence that the levels of active signaling molecules (Adipokines and Lipokines) have a crucial role in depressive state; attenuating or promoting effect according to their levels [16]. this strength relationship is higher from obesity to depression. additionally, obesity affect mental health and cognitive health as it associated with increased risk of depression. obesity may also affect how individual's feels about themselves and what people living in society feel about them [17].

Vitamin D3 (cholecalciferol) is produced endogenously through exposure of skin to sunlight and is absorbed from foods containing or supplemented with vitamin D3 or D2 (ergocalciferol). Vitamin D2 and D3 are converted in the liver to 25 hydroxy vitamin D by vitamin D25 hydroxylase enzyme [18]. Biologically, the main function of vitamin D is in the control of calcium homeostasis [19]. Vitamin D deficiency results in inadequate calcium absorption from the intestine, which disrupts calcium and phosphate balance and affects bone health. Risk factors for deficiency include limited sunlight exposure, dietary insufficiency, malabsorption syndromes, and liver or kidney dysfunction, which impair the hydroxylation steps necessary for vitamin D activation [18].

With regards to the relationship between Vitamin D and depression, it had been postulated that Vitamin D has a role to play in development of depression [20]. It has numerous functions in the brain such as neuroimmunomodulation, regulation of neurotropic factors, neuroprotein, neuroplasticity, and brain development [21,22]. vitamin D is through to influence serotoninergic system and contribute to maintenance of circadian rhythm, both of which are associated with depressive symptoms [23].

With regards to the association between vitamin D levels and depression, several studies that investigated such association proposed that the risk of depression increases by up to 14% in individuals with deficient vitamin D levels [24] and that the risk of developning depression increases significantly with the lower levels of vitamin D reflecting a negative (inverse) direction of the correlation between vitamin D levels and depression where it has been documented that individuals with very low levels of vitamin D (<15 ng/ml) are about three times more likely to have depression while those with low levels are about two times more likely to have depression when compared to control group, depression patient has significantly lower level of vitamin D [25–28].

SUBJECTS AND METHODS:

A case control study conducted at the psychiatric consultant Clinics at Alhakeem General Hospital in Al Najaf city during period from February 2025 to August 2025.it included 120 subject, 70 subject with depression and 50 health subject.

All ethical issues were approved by obtaining the official agreement from the Iraqi Council of Medical Specialization and the Psychiatric consultants of Alhakeem hospital in Al Najaf city. Additionally, verbal consent was obtained from each participant

The study included two groups, the Patients' group which included 70 patients with established diagnosed depression and they were later categorized according to the beck depression inventory- II into mild, moderate or severe and these subgroups included 24, 24 and 22, patients, respectively, On the other hand, patients were categorized according to their BMI as normal, overweight or obese.

The Control group involved 50 healthy subject and matched for age and obesity with patient group.

Inclusion criteria:

Cases of depression aged from 20 to 60 years of both sexes.

Exclusion criteria:

1. Patient with hypothyroidism.
2. Parkinson disease.
3. Rheumatoid arthritis.
4. Pulmonary disease.
5. Those taken vitamin D supplement within previous two months.
6. Chronic diseases such as liver and renal diseases, malabsorption syndrome, and other diseases that can affect the level of vitamin D

Data collection:

Data were collected through full history taking and a thorough physical examination, data included the patient's age, sex, history of chronic disease and taking vitamin D supplements, Depression was assessed according to the Beck Depression and Inventory-II

Data were collected through full history taking and a thorough physical examination, data included the patient's age, sex, history of chronic disease, taking vitamin D supplements, history, and other demographic characteristics

Body mass index was calculated as weight in kilogram divided by height in squared meter (14). The patients and controls were categorized according to their BMI into:

Normal weight (BMI: 18.6-24.9 Kg/m^2), Overweight (BMI: 25 – 29.9 kg/m^2) and Obese (BMI ≥ 30 kg/m^2)

Blood sampling:

Three millimeters of venous whole blood was aspirated from the vein of antecubital fossa of each study participant and collected into plain gel tube, left at room temperature for 20-30 minute and allowed to clot. Then, the gel tube was centrifuged at 2500 rpm for 10 minute to obtain serum sample for measurement of 25 hydroxyvitamin D level.

Vitamin D was tested using 25-hydroxy vitamin D test kit (immunofluorescence detection kit) by Hipro AFS 1 immunoassay system. The reference range for this kit is Vitamin D deficiency < 30 ng /ml and the preferred level of vitamin D >30 ng / ml

Statistical analysis:

All statistical analyses were performed using SPSS software version 26.0. Scale variables were initially assessed for normality using the Shapiro–Wilk test. Continuous variables with normal distribution (e.g., Age and BMI) were expressed as mean ± standard deviation (SD). Bivariate correlation analysis was performed to assess the pairwise correlation between age, BMI and vitamin D using Pearson's correlation test. The correlation coefficient (R) value was calculated which an estimator for the correlation strength and direction, the R value ranged between "0" which indicates complete no correlation and "1" for perfect correlation, the absolute R value close to "1" indicates the stronger correlation. The negative R value indicates an inverse correlation. Level of significance of $P \leq 0.05$ was considered significant.

RESULTS:

A total of 70 patients with different depression severity in addition to 50 health subjects as control group were enrolled in this study, both groups were almost matched for their demographic characteristics. The depression group (Patients) consisted of 24 patients with mild, 24 patients with moderate and 22 patients with severe depression. The mean (±SD) values of age and BMI of entire patients group did not differ significantly from those of control group ($p > 0.05$). The median and interquartile range (IQR) value of 25 hydroxyvitamin D of entire group of patients [17.50 (11.00- 22.00) ng/ml] was significantly decreased in comparison with that of controls [22.00 (18.00-30.00) ng/ml, $p < 0.001$], (**Table 1**)

non-significant differences in mean values of age and BMI among subgroups of depression patients (mild, moderate, severe) and against controls, (**Table 2**). The median value of 25 hydroxyvitamin D levels showed significant differences among the groups ($p < 0.0001$). The moderate subgroup of patients demonstrated significantly lower 25 hydroxyvitamin D levels compared with both the control ($p = 0.0043$) and mild group ($p = 0.0007$). The severe subgroup of patients had the lowest 25 hydroxyvitamin D levels which was significantly lower than the control ($p = 0.0001$), mild ($p = 0.0001$), and moderate subgroups ($p = 0.0099$). However, the mild subgroup of patients had median value of 25 hydroxyvitamin D level that did not differ significantly from that of controls ($p = 0.9999$), (**Table 3**).

In mild depression group, there was a statistically significant negative correlation between age and vitamin D levels ($r=-0.519$, $p=0.009$), (**Table 4 & Figure 2**)

In patients with severe depression, there was a statistically significant negative correlation between BMI and vitamin D level ($r=-0.473$, $p=0.026$), (**Table 5 & Figure 3**)

In patients with moderate depression and the control group there was no significant correlation between age and BMI from one side against vitamin D levels on the other side, in both correlations, R value is very small and the P. value >0.05. From other point of view, in both of moderate depression and the control groups, BMI was significantly correlated with age, (P. value <0.05). These findings are shown in (**Table 6 and Table 7**)

Table 1. Demographic characteristics and 25 hydroxyvitamin D of entire patients group and control group.

Parameters	Entire patient group(n=70)	Control group (n=50)	P. value
Age (year), (mean ±SD)	35.21 ±11.12	35.04 ±11.67	0.934
BMI (kg/m²), (mean ±SD)	26.88 ±6.31	26.94 ±6.45	0.944
25 Hydroxy vitamin D, Median (IQR)	17.50 (11.00-22.00)	22.00 (18.00-30.00)	0.001

SD: standard deviation, IQR: interquartile range

Table 3.2: Comparison of age and body mass index of sub groups of depression patients and healthy controls.

	Age (Year)		BMI (Kg/m2)	
	Mean	SD	Mean	SD
Mild depression (n=24)	33.17	10.44	24.00	5.58
Moderate depression (n=24)	37.96	12.36	28.54	6.45
Severe Depression (n=22)	34.45	10.27	28.14	6.09
Control group (n=50)	35.04	11.67	26.94	6.45
P value	0.516		0.056	

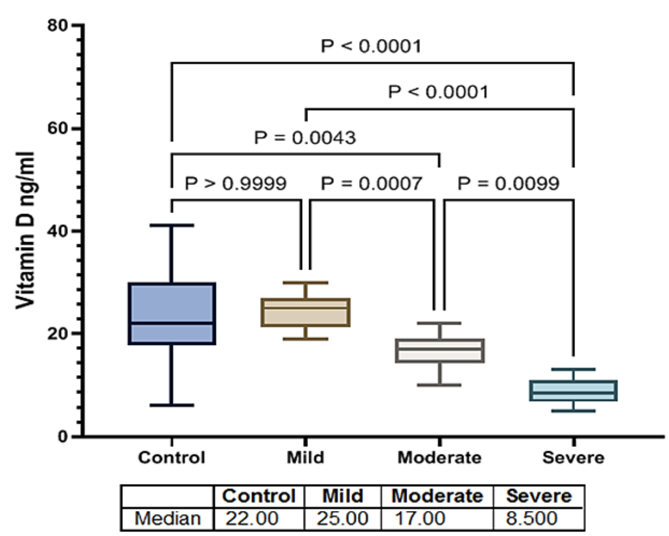

Figure 1. Comparison of differences among and between subgroups of patients and controls

Table 4. Results of bivariate correlation analysis between age, vitamin D and BMI in patients with mild depression (n=24)

Variables	Correlation parameters	Variables	
		Age (year)	BMI (kg/m²)
BMI (kg/m²)	R	0.774	-
	P. value	**<0.001**	-
vitamin D (ng/ml)	R	-0.519	-0.254
	P. value	**0.009**	0.231

R: Correlation coefficient

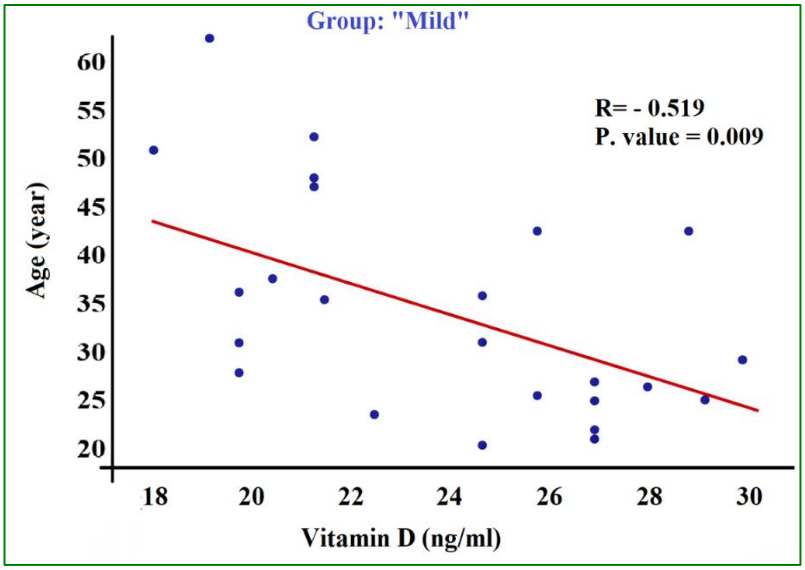

Figure 2. Scatter-Plot showing the inverse significant correlation between age and 25 hydroxyvitamin D in patients with Mild depression (N=24).

Table 5. Results of bivariate correlation analysis between age, vitamin D and BMI in patients with severe depression (n=22)

Variables	Correlation parameters	Variables	
		Age (year)	BMI (kg/m²)
BMI (kg/m²)	R	0.684	-
	P. value	**0.0004**	-
vitamin D (ng/ml)	R	-0.087	-0.473
	P. value	0.700	**0.0263**

R: Correlation coefficient

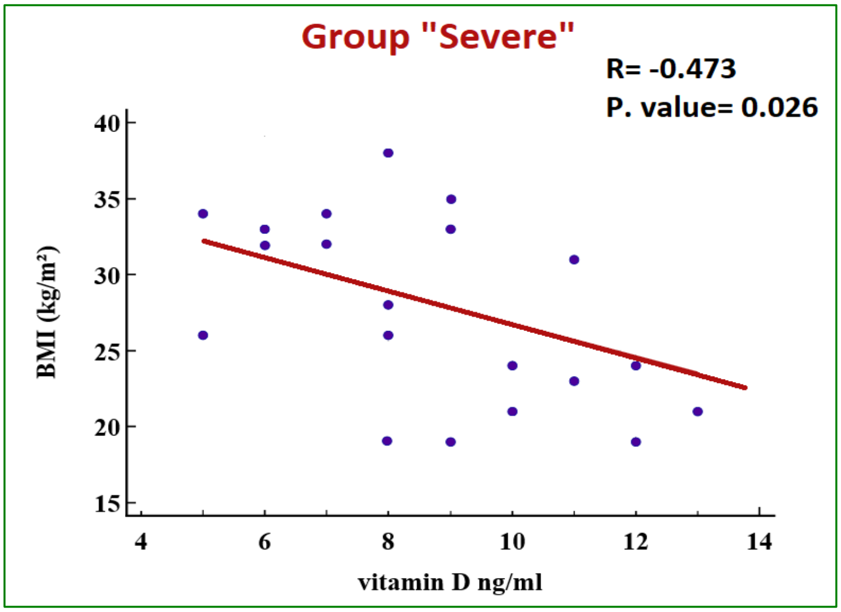

Figure 3. Scatter-Plot showing the inverse significant correlation between BMI and 25 hydroxyvitamin D in severe group of patients (N=22)

Table 6. Results of bivariate correlation analysis between age, vitamin D and BMI in control group (n=50)

Variables	Correlation parameters	Variables	
		Age (year)	BMI (kg/m^2)
BMI (kg/m^2)	R	0.703	-
	P. value	**<0.001**	-
vitamin D (ng/ml)	R	-0.092	-0.134
	P. value	0.5273	0.3519

R: Correlation coefficient

Table 7. Results of bivariate correlation analysis between age, vitamin D and BMI in patients with moderate depression (n=24)

Variables	Correlation parameters	Variables	
		Age (year)	BMI (kg/m^2)
BMI (kg/m^2)	R	**0.640**	-
	P. value	**0.0008**	-
vitamin D (ng/ml)	R	-0.047	0.201
	P. value	0.827	0.347

R: Correlation coefficient

DISCUSSION:

The mean value of BMI of patients was 26.8 kg/m², which may indicate borderline effect of obesity on the incidence of depression. It has been revealed that obesity play a role but doesn't fully mediate the relationship with depression suggested that presence of other contributing factors [29].

The present study documented that the levels of vitamin D was significantly among depressed individuals compared to controls, and this decline was more pronounced with increasing severity of depression. These finding agreed that reported in a study conducted in UK which suggested that both vitamin D deficiency and insufficiency might be risk factors for the development of new onset depression in middle age adult. Moreover, that study concluded that vitamin D deficiency and to lesser extent insufficiency might be a predictor of sustained depressive symptoms in patients who were already depressed [30]. A narrative review published by Parker, et al.[31] in 2017 and included multiple empirical papers published from 2011 onward, documented the potential benefits of vitamin D supplementation and higher serum vitamin D levels in reducing the development and symptoms of depression [31].

A meta-analysis conducted by Park et al. [32] revealed that vitamin D supplementation had significantly ameliorating effect on depression. Nonetheless, there is heterogenous results among various studies [32]. It had been widely postulated that microglial inflammatory function is caused by vitamin D deficiency, which may leads to infection of the brain [33].

Hence, in a study conducted by Zittermann et al. in 2003 a significant association was found between brain dysfunction and vitamin D deficiency. These findings also supported by a later study conducted in 2008 by McCann et al.[34,35]. In fact, the role of vitamin D is so crucial for the normal development of the brain and its deficiency has been connected multiple morphological changes in the brain like ventricles enlargement and lower thickness of the cerebral cortex [36]. However, since almost 30 years, a complicated correlation was proposed among the immune activation, brain changes, neuroinflammation and each of depression and anxiety [37], because vitamin D plays a significant role in the immunity [38] and through this mechanism, vitamin D influences immune responses in both the peripheral and central nervous systems [39]. Initially recognized for its antimicrobial properties, vitamin D is also involved in modulating both innate and adaptive immune reactions, also depression and anxiety are frequently associated with low-grade inflammation and elevated levels of acute-phase proteins and inflammatory cytokines [40].

Moreover, vitamin D regulates the gene expression of tyrosine hydroxylase, a key enzyme in the synthesis of dopamine and norepinephrine, neurotransmitters that play a central role in mood regulation and depression [41,42].

Hoogendijk et al. proposed a possible role for vitamin D in brain function [43]. Vitamin D is a secosteroid hormone capable of crossing the blood–brain barrier, and its receptors are widely distributed in several brain regions, including the cortex, cerebellum, and limbic system. It has been suggested that low vitamin D levels may lead to elevated parathyroid hormone (PTH), which has been linked to depressive symptoms. Interestingly, addressing hyperparathyroidism has been reported to improve depressive symptoms, indicating a potential therapeutic pathway through the correction of vitamin D deficiency [43].

However, in patients with severe depression, BMI was negatively correlated with 25(OH) D levels suggesting that adiposity may exacerbate vitamin D insufficiency. This finding is in line with previous reports found that level of adipose tissues was elevated the inflammatory cytokine CRP and IL 6 [44]. In other words, adipose tissue increases the risk of inflammation and inflammatory disease. Because of anti-inflammatory effect of vitamin D, vitamin D deficiency and insufficiency increase risk of inflammation and inflammatory disease [44]. Obese and overweight people have weaker bone so their requirement might be greater than others [45]. Also, obese people have low physical activity and prefer to cover their body with more clothes so may not have adequate sun exposure [46]. Vitamin D have greater tendency to store in adipose tissue so the bioavailability of vitamin D decrease, this exacerbating cycle may reduce circulating vitamin D [47].

CONCLUSION:

Body mass index may have borderline effect on depression incidence. However, adiposity may worsen vitamin D deficiency in severe depression. A Significant reduction in serum 25(OH)D levels among depressed individuals, and this reduction was more pronounced with increasing depression severity. We recommend regular screening for vitamin D deficiency in individuals with depression, particularly those with obesity, to allow for early detection and timely intervention. Implementation of vitamin D supplementation programs and dietary modifications to improve vitamin D status may help reduce the burden of depression and its complications. Encouragement of outdoor physical activity and healthy weight management strategies may contribute to improving both vitamin D levels and mental health outcomes. Longitudinal studies are recommended to clarify the causal relationship between vitamin D deficiency, obesity, and depression, as well as to evaluate the impact of vitamin D supplementation on mood disorders. Health authorities should raise public awareness about the importance of adequate vitamin D intake through sunlight exposure, fortified foods, and supplements where necessary.

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