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The association of vitamin D deficiency with depressive disorders in patients with eating disorders: a cross-sectional study

Rami Bou Khalil[1,2*], Hala Kerbage[3,4], Myriam Zarzour[1,2], Robertas Strumila[5,6,7], Maude Seneque[6,7], Ariane Sultan[8], Antoine Avignon[8], Philippe Courtet[6,7], Isabelle Jaussent[9] and Sebastien Guillaume[6,7]

Abstract

Background Vitamin D deficiency and depressive disorders are both common in patients with eating disorders (EDs). However, the association between vitamin D status and current depressive disorder (CDD) in EDs populations remains insufficiently explored.

Methods We conducted a cross-sectional study including 481 outpatients with EDs assessed at a specialized eating-disorders unit in Montpellier, France. Psychiatric diagnoses were established using the Mini-International Neuropsychiatric Interview (M.I.N.I.). Eating-disorder severity was assessed with the Eating Disorder Examination Questionnaire (EDE-Q). Serum 25-hydroxyvitamin D levels were measured as part of routine clinical assessment, and vitamin D deficiency was defined as < 20 ng/mL. Odds ratios (ORs) were estimated using logistic regression.

Results Vitamin D deficiency was significantly associated with higher odds of CDD (OR 1.98, 95% CI 1.20–3.28; $p = 0.008$). This association persisted after adjustment for EDs severity, psychiatric comorbidities, and season of assessment. No significant interactions were found between vitamin D deficiency and eating-disorder subtype, EDE-Q score, or season.

Conclusions Vitamin D deficiency was associated with the presence of a current depressive disorder in patients with eating disorders, independently of EDs subtype, severity, and seasonality. These findings support the clinical relevance of assessing vitamin D status in patients with EDs presenting with depressive symptoms. Longitudinal studies are needed to clarify the direction and clinical implications of this association.

Plain English summary

People with eating disorders often have low vitamin D levels, and depression is also common in this group. We studied 481 outpatients seen in a specialist eating disorder service in Montpellier, France, to find out whether vitamin D deficiency was more frequent in those who also had a current depressive disorder. Vitamin D deficiency was defined as a blood level below 20 ng/mL. We found that patients with vitamin D deficiency were almost twice as likely to have a current depressive disorder as those without deficiency. This relationship remained after taking into account eating disorder severity, other psychiatric conditions, and the season of assessment. It was also similar

*Correspondence:
Rami Bou Khalil
rami.boukhalil@usj.edu.lb

Full list of author information is available at the end of the article

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across different eating disorder subtypes. Because this was a cross-sectional study, we cannot determine whether low vitamin D contributes to depression, whether depression contributes to low vitamin D, or whether other factors influence both. Still, our findings support checking vitamin D status in patients with eating disorders who present with depressive symptoms.

Keywords Eating disorders, Vitamin D deficiency, Depression, Seasonality, Eating disorder subtype

Background

Vitamin D is a steroid hormone obtained from dietary sources or through the endogenous conversion of 7-dehydrocholesterol following exposure to solar ultraviolet B radiation [1, 2]. After hepatic and renal transformation, the biologically active form, calcitriol (1α,25-dihydroxyvitamin D or 1,25(OH)2D3), is produced [3]. Calcitriol regulates gene transcription and contributes to calcium and phosphorus homeostasis, metabolic functions, and bone health [4–8].

Vitamin D also has potential relevance for brain function because of its antioxidant, anti-inflammatory, proneurogenic, and neuromodulatory properties [8, 9]. In the general population, vitamin D deficiency has been associated with depression, particularly in older adults and during the postpartum period [10–13]. Low vitamin D levels have also been reported in several psychiatric conditions, including first-episode psychosis and fibromyalgia, especially when depressive symptoms are present [14, 15]. Meta-analytic evidence suggests that vitamin D supplementation may improve depressive symptoms in some settings [16, 17].

Patients with eating disorders may be especially vulnerable to vitamin D deficiency because of malnutrition, reduced dietary fat intake, limited sunlight exposure, disturbed daily routines, and medical comorbidities affecting vitamin D metabolism [18–27]. Previous studies have reported vitamin D deficiency rates ranging from 18% to 36% in eating-disorder populations, with possible variation across diagnostic subtypes [26, 28–31].

Despite the high prevalence of both vitamin D deficiency and depressive disorders in people with eating disorders (EDs), the relationship between these conditions remains insufficiently explored in eating-disorder populations [31]. Existing studies were generally limited by small samples, a focus on anorexia nervosa, adolescent inpatient settings, or incomplete consideration of seasonal variation and metabolic confounders [28, 31]. Given the known seasonal variation in vitamin D status, season of assessment should be considered when examining associations involving vitamin D [32].

Accordingly, this study examined whether patients with eating disorders and a current depressive disorder had a higher prevalence of vitamin D deficiency than patients without depression. We also investigated whether any association between vitamin D deficiency and current depressive disorder varied according to EDs subtype, EDs severity, or season of assessment.

Methods

Participants

This cross-sectional study included outpatients aged 15–65 years with a diagnosis of an eating disorder according to the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) criteria [33]. Participants were assessed at a specialized eating-disorders day-care unit in Montpellier, France, between February 2012 and January 2020, before treatment initiation.

All participants provided informed written consent; for participants younger than 18 years, written consent was obtained from a parent or legal guardian. Exclusion criteria were: (1) lack of consent; (2) intellectual disability or psychotic disorder precluding valid consent; (3) serious physical comorbidity requiring full-time hospital admission; (4) ongoing vitamin D supplementation; and (5) missing serum vitamin D data.

The data used in this study were collected as part of two previous studies approved by CPP Sud-Méditerranée IV (reference 11-04-SC) and CPP Sud-Est 6 (reference AU13-13). The study was conducted in accordance with the Declaration of Helsinki.

Measures

Eating-disorder diagnosis: Eating-disorder diagnosis was established through an unstructured multidisciplinary clinical assessment conducted by psychiatrists, psychologists, and nutritionists according to DSM-5 criteria [33].

For descriptive subtype classification, participants with atypical anorexia nervosa were included in the anorexia nervosa category. Other DSM-5 presentations categorized as other specified feeding or eating disorder (OSFED) were grouped as other EDs.

Anthropometric measurements: Weight and height were measured in a standardized manner during the morning clinical examination, and body mass index (BMI) was calculated at the time of assessment.

Structured clinical interview: The Mini-International Neuropsychiatric Interview (M.I.N.I., version 5.0.0) was used to assess psychiatric comorbidities frequently encountered in EDs populations, including depressive disorders, anxiety disorders, obsessive-compulsive disorder (OCD), post-traumatic stress disorder (PTSD), and alcohol and/or other substance use disorders (SUD) [34].

Lifetime suicide attempts were also recorded. Current depressive disorder (CDD) was defined as the presence of a current major depressive episode according to the M.I.N.I.

Self-administered assessment: The 28-item Eating Disorder Examination Questionnaire (EDE-Q) version 6.0, validated in French, was used to assess eating-disorder psychopathology over the preceding 28 days. The instrument provides a global score and four subscales (restraint, eating concern, shape concern, and weight concern). Higher scores indicate greater eating-disorder severity; a global score ≥ 4 was considered indicative of severe eating-disorder psychopathology [35, 36]. In the present sample, Cronbach's alpha for the total EDE-Q score was 0.723.

Body composition: Bioelectrical impedance analysis was used to estimate fat mass (FM). Resistance and reactance were determined using a four-terminal impedance plethysmograph (BIACORPUS RX 4000, Lainate, Italy).

Resting energy expenditure: Resting energy expenditure (REE) was measured by open-circuit indirect calorimetry with the QUARK RMR system (COSMED, Rome, Italy). Measurements were obtained in the morning, after an overnight fast and smoking abstinence.

Laboratory evaluations: Fasting peripheral blood samples were collected between 8:00 and 9:00 a.m. Serum calcium, phosphate, and alkaline phosphatase were measured using a Cobas 8000 analyser (Roche Diagnostics, Meylan, France). Vitamin D status was assessed using serum 25-hydroxyvitamin D. In line with commonly used clinical thresholds, vitamin D deficiency was defined as < 20 ng/mL [37, 38].

Seasonality: Samples obtained from autumn and winter were grouped as the low-light season, and samples obtained from spring and summer were grouped as the high-light season.

Statistical analysis

Categorical variables were described using counts and percentages, and continuous variables using medians and interquartile ranges because several variables were not normally distributed. Associations between demographic, clinical (including EDE-Q scores), and biological parameters (including vitamin D status) and CDD (dependent variable) were assessed using univariable logistic regression models and were quantified with odds ratios (ORs) and 95% confidence intervals (CIs). Categorical explanatory variables were recoded as dummy (indicator) variables before inclusion in the logistic regression models, with one category serving as the reference group. The linearity assumption for quantitative parameters was assessed using the likelihood ratio test.

Multivariable logistic regression models were used to estimate adjusted ORs for the association between vitamin D status and CDD, including potential confounders identified in univariable analysis. Sequential multivariable models were built: Model 1 for total EDE-Q score; Model 2 additionally for smoking status, anxiety disorders, suicidal attempts, OCD, SUD, and PTSD, and Model 3 further adjusted for season at time of assessment. Overall model fit for all multivariable logistic regression models was assessed using the likelihood ratio test for nested models. Interaction terms were tested using Wald χ^2 tests to assess potential effect modification for clinically relevant variables defined a priori, including EDs subtype, EDE-Q score, and season of assessment.

Correlations between continuous variables were assessed using Spearman's rank correlation coefficient.

The significance threshold was set at $p < 0.05$. Analyses were performed using SAS version 9.4 (SAS Institute, Cary, NC, USA).

Results

Sample characteristics

Of the 622 participants included in the two source cohorts, 481 (77.3%) had complete data on both CDD and vitamin D status and were therefore included in the present analysis. Median age was 24.56 years (interquartile range, 20.32–33.08), and 449 participants (93.35%) were female.

CDD was present in 146 participants (30.35%), and vitamin D deficiency was present in 76 participants (15.80%). A significant negative correlation was observed between BMI and serum vitamin D levels ($r = -0.32$, $p < 0.0001$). Participants with vitamin D deficiency had a higher mean BMI than those without deficiency (24.29 ± 8.80 vs. 19.77 ± 5.16 kg/m^2, $p < 0.01$), but BMI was not associated with the presence of a CDD.

Associations with current depressive disorder

Compared with participants without CDD, those with CDD had higher EDE-Q scores and were more likely to present with anxiety disorders, OCD, PTSD, substance use disorders, and a lifetime history of suicide attempt (all associations assessed using univariable logistic regression models; all $p < 0.05$). Assessment during the low-light season was also more frequent among participants with CDD (Table 1).

No significant associations with CDD were found for age, sex, BMI, fat mass, REE, smoking status, EDs subtype, or the biological parameters reported in the source manuscript.

Vitamin D deficiency and current depressive disorder

Vitamin D deficiency was associated with higher odds of CDD in the unadjusted model (OR 1.98, 95% CI 1.20–3.28; $p = 0.008$). The association remained significant

Table 1 Description of sociodemographic, clinical and biological parameters in patients with eating disorders (*N* = 481) and comparison between those with and without current depressive disorder

		Current depressive disorder			
	Whole population ***N*** **= 481**	**No** ***N*** **= 335**	**Yes** ***N*** **= 146**		
Variable	n (%)	n (%)	n (%)	OR [95%CI][1]	p-value
Gender, Female	449 (93.35)	310 (92.54)	139 (95.21)	1.01 [0.99;1.03]	0.32
Age (years)[2]	481 ; 24.56 (20.32; 33.08)	335; 24.27 (20.16; 32.63)	146; 25.33 (20.75; 34.50)	1.60 [0.68;3.79]	0.28
Body mass index (kg/m^2)[2]	481 ; 18.55 (16.58; 22.10)	335; 18.60 (16.54; 22.02)	146; 18.36 (16.60; 23.09)	1.01 [0.97;1.04]	0.70
Smoking status, Current smoker	185 (39.11)	119 (36.39)	66 (45.21)	1.44 [0.97;2.14]	0.07
Resting energy expenditure (Kcal/day)[2,3]	459; 1236.00 (1075.00; 1402.00)	322; 1226.50 (1071.00; 1380.00)	137; 1253.00 (1080.00; 1427.00)	1.20 [0.59 ;2.42]	0.61
Fatty mass (kg)[2]	454; 12.87 (8.36; 19.78)	317; 12.48 (8.09; 18.91)	137; 13.16 (9.06; 22.07)	1.01 [0.99;1.03]	0.26
Eating disorder					0.40
Anorexia Nervosa	267 (55.51)	182 (54.33)	85 (58.22)	1	
Bulimia nervosa	115 (23.91)	83 (24.78)	32 (21.92)	0.83 [0.51;1.34]	
Binge-eating disorder	53 (11.02)	34 (10.15)	19 (13.01)	1.20 [0.65;2.22]	
Other EDs[4]	46 (9.56)	36 (10.75)	10 (6.85)	0.60 [0.28;1.26]	
Total EDE-Q score[2]	448; 3.71 (2.27; 4.65)	315; 3.31 (1.83; 4.29)	133; 4.32 (3.38; 5.09)	1.67 [1.42;1.97]	< 0.0001
Total EDE-Q score, ≥4	187 (41.74)	107 (33.97)	80 (60.15)	2.93 [1.93;4.46]	< 0.0001
Anxiety disorder, Yes	244 (51.05)	142 (42.51)	102 (70.83)	3.28 [2.16;5.00]	< 0.0001
Suicide attempt, Yes	115 (24.06)	64 (19.28)	51 (34.93)	2.25 [1.45;3.48]	0.0003
Obsessive-compulsive disorder, Yes	61 (12.76)	35 (10.45)	26 (18.18)	1.91 [1.10;3.30]	0.02
Substance use disorder, Yes	40 (8.32)	22 (6.57)	18 (12.33)	2.00 [1.04;3.86]	0.04
PTSD, Yes	35 (7.35)	10 (3.00)	25 (17.48)	6.84 [3.19;14.7]	< 0.0001
Season at time of assessment, High-light	265 (55.09)	196 (58.51)	69 (47.26)	0.64 [0.43;0.94]	0.02
Biological parameters					
Prothrombin time (%)[2,5]	455; 100.00 (94.00; 100.00)	315; 100.00 (94.00; 100.00)	140; 100.00 (93.00; 100.00)	0.99 [0.97;1.02]	0.67
Vitamin B12 (pg/mL) [2]	468; 298.50 (217.50; 391.00)	328; 299.50 (210.00; 386.50)	140; 293.00 (229.00; 402.50)	0.95 [0.75 ;1.21]	0.87
C-reactive protein (mg/L) [2]	452; 0.40 (0.30; 1.65)	319; 0.50 (0.30; 1.60)	133; 0.40 (0.30; 1.70)	1.00 [0.94;1.06]	0.99
Haemoglobin (g/dl) [2]	474; 13.30 (12.70; 14.10)	331; 13.40 (12.70; 14.00)	143; 13.20 (12.60; 14.30)	1.06 [0.91;1.24]	0.45
Ferritin (Microg/mL) [2,5]	459; 46.00 (24.00; 83.00)	321; 46.00 (25.00; 81.00)	138; 45.00 (23.00; 86.00)	1.01 [0.98;1.04]	0.76
TSH (mUI/L) [2]	442; 1.86 (1.28; 2.65)	313; 1.81 (1.25; 2.60)	129; 2.00 (1.30; 2.79)	1.09 [0.93;1.28]	0.27
Albumin (g/L) [2]	443; 46.00 (44.00; 49.00)	306; 46.00 (44.00; 49.00)	137; 46.00 (44.00; 48.00)	0.98 [0.92;1.04]	0.51
Creatinine (µmol/L) [2]	475; 68.00 (60.00; 76.00)	330; 69.00 (61.00; 76.00)	145; 67.00 (59.00; 75.00)	0.99 [0.97;1.01]	0.24

[1]Values in square brackets in the OR columns are 95% confidence intervals

[2]Continuous variable was expressed as a number, median (First quartile; Third quartile)

[3]OR for a 1000-unit increase

[4]Other EDs include UFED and OSFED presentations other than atypical anorexia nervosa. Subthreshold forms related to bulimia nervosa and binge-eating disorder were grouped within their respective broader diagnostic categories when applicable

[5]OR for a 10-unit increase

CI Confidence intervals, *EDE-Q* Eating Disorder Examination Questionnaire, *OR* Odds-ratio, *PTSD* Post-Traumatic Stress Disorder

after adjustment for EDE-Q total score and after additional adjustment for smoking status, anxiety disorders, lifetime suicide attempts, OCD, SUD, PTSD, and season of assessment (Table 2).

When vitamin D was analyzed as a continuous variable, no significant association with CDD was observed.

Interaction analyses

No significant interactions were detected between vitamin D deficiency and EDs subtype, EDE-Q score, or season of assessment in relation to the presence of CDD. The association between vitamin D deficiency and CDD was therefore not meaningfully modified by diagnostic subtype, EDs severity, or seasonality in this sample.

Table 2 Association Between Vitamin D Status and the Presence of Current Depressive Disorder

Variable		Current depressive disorder									
	Whole population *N* = 481	No *N* = 335	Yes *N* = 146	Model 0[2]		Model 1[3]		Model 2[4]		Model 3[5]	
Variable	n (%)	n (%)	n (%)	OR [95%CI][6]	p	OR [95%CI][6]	p	OR [95%CI][6]	p	OR [95%CI][6]	p
Vitamin D deficiency					0.008		0.02		0.02		0.04
≥ 20 ng/ml	405 (84.20)	292 (87.16)	113 (77.40)	1		1		1		1	
< 20 ng/ml	76 (15.80)	43 (12.84)	33 (22.60)	1.98 [1.20; 3.28]		1.90 [1.09; 3.29]		2.03 [1.12; 3.68]		1.89 [1.03; 3.46]	
Vitamin D serum concentration (ng/ml)[1] OR for a 20-units decrease	481; 33.00 (24.00; 43.00)	335; 33.60 (24.50; 43.30)	146; 31.75 (21.00; 40.00)	1.33 [1.00; 1.77]	0.049	1.30 [0.96; 1.76]	0.09	1.24 [0.89; 1.74]	0.21	1.18 [0.84; 1.66]	0.35

[1]Continuous variable was expressed as a number, median (First quartile; Third quartile)

[2]Crude association

[3]Model 1 was adjusted for the Total EDE-Q score

[4]Model 2 was adjusted for Total EDE-Q score plus smoking status, anxiety disorders, suicidal attempts, obsessive-compulsive disorder, substance use disorders, and PTSD

[5]Model 3 was adjusted for all the covariates in Model 2, plus season at time of assessment

[6] Values in square brackets in the OR columns are 95% confidence intervals

Overall model fit for each logistic regression model was assessed using the likelihood ratio test

EDE-Q Eating Disorder Examination Questionnaire, *OR* Odds ratio, *CI* Confidence Intervals, *PTSD* Post-Traumatic Stress Disorder

Discussion

In this relatively large outpatient sample of patients with EDs, vitamin D deficiency was associated with nearly twice the odds of a CDD. This relationship remained significant after adjustment for total EDE-Q score, smoking status, anxiety disorders, lifetime suicide attempts, OCD, SUD, PTSD, and season of assessment, and it did not differ according to EDs subtype, EDE-Q score, or season.

The fact that vitamin D deficiency, but not serum 25-hydroxyvitamin D concentration analyzed as a continuous variable, was associated with CDD suggests that the relationship may reflect a threshold effect rather than a dose-dependent association across the full range of vitamin D values. In other words, the clinically deficient state may be more relevant to depression than incremental variations in serum vitamin D concentration among individuals without a vitamin D deficiency. This interpretation is consistent with the use of < 20 ng/mL as a clinically meaningful threshold for vitamin D deficiency and with previous evidence linking vitamin D deficiency to depression. However, given the cross-sectional design of the present study, this finding should be interpreted cautiously and does not allow causal inference [37–39].

These findings extend previous work showing that vitamin D deficiency is common in EDs populations [26, 28–31, 40] and that depressive disorders are frequent comorbidities in EDs [41–43]. Our results differ from earlier studies in adolescent inpatient samples, which did not identify a clear relationship between vitamin D status and depression [28, 31]. Compared with those studies, the present work included a larger sample, a broader range of EDs subtypes, and explicit consideration of seasonality and clinical confounders. The present sample ($n = 481$) was approximately five times larger than the samples of 87 and 93 adolescent patients included in previous studies specifically assessing vitamin D status and depression in eating-disorder populations [28, 31].

Several biological mechanisms could plausibly link vitamin D deficiency and depression. Vitamin D may influence inflammatory pathways, oxidative stress, and neurotrophic signaling, all of which have been implicated in affective disorders [9, 39]. In patients with EDs, these pathways may interact with malnutrition, circadian disruption, and reduced sunlight exposure [26, 27, 44–46]. However, the present cross-sectional design does not allow conclusions about causality, and interventional studies are needed before any therapeutic implications can be established in EDs populations.

From a clinical perspective, the findings support systematic assessment of depressive symptoms in patients with eating disorders and consideration of vitamin D testing when depressive comorbidity is present. The absence of significant interaction by season suggests that this issue should not be viewed as relevant only in winter. Similarly, the absence of interaction by EDs subtype suggests that attention to vitamin D status should not be restricted to anorexia nervosa alone.

This study has several limitations. First, its cross-sectional design precludes any inference about directionality. Second, the sample was recruited from a single specialist center in Montpellier, France, which may limit generalizability. Third, the study took place in Southern France, where ambient sunlight is relatively abundant, and seasonal findings may differ at higher latitudes. Finally, the binge eating disorders subgroup likely underrepresented patients with obesity because local referral pathways more often direct such patients to nutrition or endocrinology services rather than psychiatric EDs units. Despite these limitations, the study contributes clinically relevant evidence from a sizeable sample assessed with standardized psychiatric and EDs measures.

Conclusions

Vitamin D deficiency was associated with the presence of a CDD in patients with EDs, independently of EDs subtype, EDs severity, and season of assessment.

These findings support the clinical relevance of assessing vitamin D status in patients with eating disorders who present with depressive symptoms. Longitudinal and interventional studies are needed to clarify directionality and to determine whether correcting vitamin D deficiency can improve clinical outcomes in this population.

Abbreviations

BMI	Body mass index
CDD	Current depressive disorder
CI	Confidence interval
DSM-5	Diagnostic and statistical manual of mental disorders, fifth edition
EDs	Eating disorders
EDE-Q	Eating disorder examination questionnaire
FM	Fat mass
M.I.N.I.	Mini-International neuropsychiatric interview
OCD	Obsessive-compulsive disorder
OR	Odds ratio
PTSD	Post-traumatic stress disorder
REE	Resting energy expenditure
SUD	Substance use disorder

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Author contributions

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Data availability

The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request, subject to institutional and ethical constraints.

Declarations

Ethics approval and consent to participate

The data used in this study were collected as part of two previous studies approved by CPP Sud-Méditerranée IV (reference 11-04-SC) and CPP Sud-Est 6 (reference AU13-13). All participants provided informed written consent; for participants younger than 18 years, written consent was obtained from a parent or legal guardian.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

Author details

[1]Psychiatry Department, Saint Joseph University, Beirut, Lebanon
[2]Psychiatry Department, Hôtel-Dieu de France, PO box: 166830, Boulevard Alfred Naccache, Achrafieh, Beirut, Lebanon
[3]Child and Adolescent Psychiatry Department, Saint Eloi University Hospital, Montpellier, France
[4]4. Epidemiology and Population Health Centre (CESP), INSERM U1018, Developmental Psychiatry, Paris-Saclay University, Paris, France
[5]Faculty of Medicine, Institute of Clinical Medicine, Psychiatric Clinic, Vilnius University, Vilnius, Lithuania
[6]Emergency Psychiatry and Acute Care Department, Lapeyronie Hospital, Montpellier University Hospital Centre, Montpellier, France
[7]IGF, Montpellier University, CNRS, INSERM, Montpellier, France
[8]Nutrition-Diabetes Department, Lapeyronie University Hospital Centre, Montpellier, France
[9]Institute for Neurosciences of Montpellier (INM), Montpellier University, Inserm, Montpellier, France

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