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BRIEF REPORT

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Relation between vitamin D and impulse behaviours in patients with eating disorder: a pilot observational study

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Abstract

Objective: There is growing evidence that vitamin D levels have a role not only in bone health and energy metabolism, but also for supporting nervous system and brain functions, including impulsivity. Impulsive behaviours are considered characteristics of great relevance in patients with Eating Disorders (ED) both for the course of the illness and for the treatment. The aim of this study is to examine the relationship between impulsive behaviours and vitamin D in patients with ED.

Method: 236 patients with a diagnosis of ED, consecutively recruited at an ED ward between 2014 and 2018, were enrolled. Patients were classified as impulsive or non-impulsive based on the presence of clinically relevant impulsive behaviours.

Results: Impulsive patients were found to have statistically significant lower levels of vitamin D than non-impulsive ($p = .007$). A threshold value of 20.4 ng/ml for discriminating impulsive from non-impulsive patients was found.

Discussion: This hypothesis generating study partially confirmed a relationship between vitamin D deficiency and impulsive behaviours in ED spectrum mediated by body weight, even if results were not confirmed after corrected by obesity. No definitive conclusion may be taken on whether the effect is reduced due to the loss of power. Future directions are discussed.

KEYWORDS

eating disorders, impulsive behaviour, impulsivity psychopathology, vitamin D

1 | INTRODUCTION

Eating disorders (ED) are severe biopsychosocial disorders characterized by specific psychopathological profiles (Solmi et al., 2018) and significant consequences on body health, mainly due to malnutrition: they range from undernutrition in Anorexia Nervosa (AN), to over nutrition in Binge Eating Disorder (BED) (Kaidar-Person, Person, Szomstein, & Rosenthal, 2008; Veronese et al., 2015). These disorders usually have their onset during adolescence, with a possible health and psychological lifetime impact on patients due to the insufficient intake of energy, proteins and vitamins or to the consequences of excessive intake for body mass (Golden et al., 2015). For example, there is increasing evidence that vitamin D levels play a role for supporting a healthy nervous system, brain function and energy metabolism, and adolescents with eating disorders have been shown to have a high prevalence of

vitamin D deficiency and insufficiency (Caprio, Infante, Calanchini, Mammi, & Fabbri, 2017; Modan-Moses et al., 2015; Malgorzata Wrzosek et al., 2013). Indeed, literature suggests that vitamin D has a distribution in the whole body and has a strong impact, not only in bone and calcium homeostasis, but also as a neuro-active steroid affecting neuronal differentiation, axonal connectivity and brain functions (Eyles, Burne, & McGrath, 2013). Recent literature on different clinical populations [e.g., attention-deficit hyperactivity disorder (ADHD), mood disorders and alcohol dependent patients] has shown a possible role of vitamin D deficiency in impulsivity through the increases of proinflammatory cytokines in the brain and a subsequent reduction of serotonin activity (Grudet, Malm, Westrin, & Brundin, 2014; Patrick & Ames, 2015; Wrzosek et al., 2013).

Therefore, in this study, we explore preliminary data on the relationship between impulsivity and 25-hydroxyvitamin D concentration in a sample of ED patients. This study is part of a larger ongoing longitudinal study.

2 | METHODS

2.1 | Sample

Patients with a diagnosis of eating disorders (ED) (n = 236) were consecutively enrolled at an inpatient Eating Disorders Unit of the Casa di Cura Villa Margherita (Arcugnano, Vicenza), between June 2014 and September 2018, and had data available for vitamin D. Participants were recruited as part of a larger observational longitudinal study. The main study, still ongoing, includes several assessments at baseline, discharge, 6-months, 1- and 5-years follow up, and it was designed to explore the course of ED in patients undergoing a multidisciplinary, patient-centred treatment. The inclusion criteria were: (a) a full diagnosis of any ED according to DSM-5 criteria (Frey, 2018) assessed by a trained and expert psychiatrist (PM); (b) age between 13 and 60; and (c) no severe medical comorbidity, neurological trauma or disorder. The sample included 236 subjects (96% female) with an average age of 29 years (SD 12.4). More than 50% of the sample had a diagnosis of Anorexia Nervosa [33.5% AN restricting (ANr) type, 22.5% AN binge purge (ANbp) type]; while 18.6% was affected by Bulimia Nervosa (BN), 14.4% by Binge Eating Disorder (BED), and 11% by Other Specified Feeding or Eating Disorder (OSFED).

The study was approved by the internal revision committee of the Casa di Cura Villa Margherita as part of a clinical evaluation of ED patients hospitalized in the Unit, and it complies with the provisions of the Declaration of Helsinki. All patients included in the analysis accepted to participate to the study and signed an informed consent.

2.2 | Assessment

The assessment of the main study includes a semi-structured interview and several self-report questionnaires to evaluate general psychopathology and ED traits and behaviours. ED diagnoses were made according to DSM-5 criteria by a fully trained psychiatrist (PM) (American Psychiatric Association and others, 2013). Psychopathology was assessed by: the Clinical Impairment Assessment Questionnaire (CIA) for the evaluation of psychosocial functioning (Bohn et al., 2008); the Eating Disorder Examination Questionnaire (EDE-Q) for the assessment of specific eating disorder psychopathology (Fairburn & Beglin, 1994); the Body Uneasiness Test (BUT) for the estimation of body image disturbances (Cuzzolaro, Vetrone, Marano, & Garfinkel, 2006) and the Beck Depression Inventory (BDI) for the specific depression symptomatology evaluation (Beck, Steer, & Garbin, 1988).

The assessment of impulsive behaviours was derived from the semi-structured interview and covered specific clinical behavioural indicators, such as: current or past binge eating, current or past substance use, current or past alcoholism, past interventions for alcoholism or substance abuse, current or past self-harm behaviours, current or past pharmacological treatment for self-harm, current or past pharmacological treatment for other impulsive behaviours. The presence of each indicator was considered separately for current or past presence and the total number of impulsive behaviours could range from 0 to 13 in accordance to the included items. We decided to follow an approach based on clinical indicators of impulsivity, similar to the one used by other authors (Favaro et al., 2005; Waxman, 2009), because this was a pilot exploratory study derived from an ongoing follow-up study and no specific measure of impulsivity was administered.

Serum sample levels of 25(OH) vitamin D Total (D2 and D3) were determined by enzyme immunoassay (UniCel DxI 800 Immunoassay System, Beckman Coulter, Inc., Brea, CA), with a sensitivity of 2 ng/ml and intra- and inter-assay coefficients of variation <10%, which is considered a valid methodology of analysis (Akbas et al., 2014). Blood samples were drawn at the time of hospitalization.

2.3 | Statistical analysis

Mann–Whitney test was performed for the comparisons of all variables between the independent groups due to

the non-normal distribution of the vitamin D levels. A receiver operating characteristic (ROC) curve was then constructed to measure discrimination, that is, the ability of the vitamin D levels to correctly classify those with and without impulsivity. The cut-off value of vitamin D for the maximized accuracy of the prediction was calculated. The area under the curve was also measured, representing the percentage of randomly drawn pairs of people from the two groups which were correctly assigned.

3 | RESULTS

The impulsivity score, indicating the number of impulsive behaviours reported during the interview, were distributed as following: 31.4% (n = 74) individuals had score 0; 33.1% (n = 78) scored 1; 27.5% (n = 65) scored 2; 3.8% (n = 9) scored 3; 2.1% (n = 5) scored 4; 0.8% (n = 2) scored 5; 0.4% (n = 1) scored 6; 0.8% (n = 2) scored 7. No individuals reported more than 7 impulsive behaviours. See Table 1 for demographic characteristics of the sample.

We first compared vitamin D levels among three groups: no impulsivity (no impulsive behaviour reported), low impulsivity (only one impulsive behaviour reported), high impulsivity (two or more behaviours reported) and data showed that there was no difference between low and high impulsivity groups in terms of vitamin D ($p = .86$). Based on these results and on literature about impulsivity in ED, showing that multi-impulsive patients are not clinically different from patients with only one indicator of impulsivity as they have similar severity and outcome (Fahy & Eisler, 1993; Lavender & Mitchell, 2015), we used a categorization of impulsive versus non-impulsive patients for the purpose of our analysis. The "no impulsive behaviours" group was defined by the absence of any impulsive behaviour during the patient's lifetime, and the "impulsive" group was defined by the presence of at least one of the specified impulsive characteristics.

Thus, we proceeded to compare no impulsivity versus impulsivity groups, which showed significantly different levels of vitamin D (31.59 ± 18.72 ng/ml and 25.85 ± 15.06 ng/ml respectively; $p = .007$; Cohen's $d = 0.34$).

The ROC curve test was significant (area under the curve = 0.61, $p = .004$) and yield a value of 20.4 ng/ml as best discriminating between impulsive and non-impulsive group (sensitivity 0.45, specificity 0.78) (see Figure 1).

Frequencies of diagnostic categories in the impulsive behaviours group were: 22 out of 79 ANr (27.8%), 43 out of 53 ANbp (81.1%), 44 out of 44 BN (100%), 34 out of 34 BED (100%) and 19 out of 26 OSFED (73.1%). Thus, the non-impulsive group was composed of individuals with ANr (n = 57), ANbp (n = 10) and OSFED (n = 7). The difference in the distribution of diagnostic categories between the two groups was significant ($p < .001$). The diagnostic distribution is partially driven by the inclusion of binge eating among the behavioural indicators of impulsivity.

TABLE 1 Description of Impulsive and Non-impulsive groups

	Non impulsive group (n = 74)	Impulsive group (n = 162)
Age (mean [SD])	26.08 (11.94)	30.33 (11.05)
Gender (%)	Females: 94.9 Males: 4.1	Females: 96.3 Males: 3.7
BMI (mean [SD])	15,64 (3,68)	24,04 (11,21)
Obesity (% BMI > 30 kg/m^2)	1.4	24.1
Diagnoses (%)	ANr (n = 57): 77.03 ANbp (n = 10): 13.51 OSFED (n = 7): 9.46	ANr (n = 22): 13.58 ANbp (n = 43): 26.54 BN (n = 44): 27.16 BED (n = 34): 20.99 OSFED (n = 19): 11.73

Abbreviations: ANbp, Anorexia Nervosa binge purge type; ANr, Anorexia Nervosa restricting type; BMI, body mass index; BED, binge eating disorder; BN: Bulimia Nervosa; OSFED, other specified feeding or eating disorder.

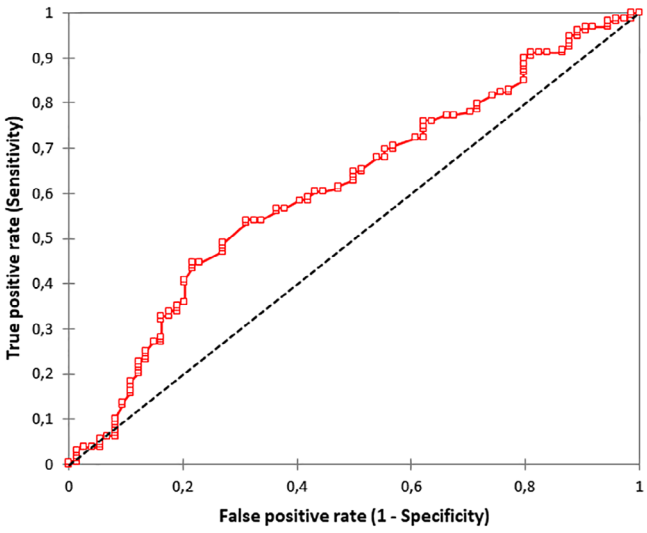

FIGURE 1 The ROC Curve represents the optimal cut-off score between subjects with impulsive behaviours and without. AUC: 0.6115, 95% CI [0.5366–0.6865]. ROC, receiver operating characteristics; AUC, area under the curve; CI, confidence interval

TABLE 2 Binary logistic regression predicting the presence of clinical indicators of impulsivity from vitamin D after adjusting for age

Source	Value	SE	*p* value	OR	OR lower 95%CI	OR upper 95%CI
Vitamin D	−0.0201	0.0086	.0195[a]	0.9801	0.9638	0.9968
				1.0203	1.0032	1.0376
Age	0.0318	0.0127	.0127[a]	1.0323	1.0068	1.0584

Abbreviations: CI, confidence interval; OR, Odds ratio; SE, standard error.
[a]Significant at 0.05 level.

There was no difference in terms of gender distribution between impulsivity (males = 3.7%) and non-impulsivity (males = 4.1%) groups ($p = .896$). However, impulsive individuals shown to have a higher average age than non-impulsive (respectively: 30.33 ± 11.05 years and 26.08 ± 11.94 years; $p < .001$; Cohen's $d = 0.37$). As expected, based on the distribution of diagnoses, we found a significantly higher rates of obesity (BMI ≥ 30 kg/m^2) in the impulsive group versus non-impulsive (respectively 24.1% and 1.4%, $p < .001$). Thus, we performed a binary logistic regression twice, the first time including only age and the second time including both age and obesity. We did not include gender as a covariate since there was no difference in the two groups. The association between impulsivity and lower levels of vitamin D was confirmed after adjusting for age (see Table 2). When adjusting also for obesity, the relationship between impulsivity and lower level of vitamin D was no longer significant although maintained a positive trend (OR: 0.9995–1.0338, $p = .0710$), see Table 3.

4 | DISCUSSION

To the best of our knowledge, this exploratory study represents the first report of an association between vitamin D and impulsive behaviours in ED patients. We only know of a recent study reporting the scores on the Non-planning Subscale of the Barratt Impulsiveness Scale to be related to particularly low levels of vitamin D in bariatric surgery candidates with emotional eating (Wrzosek, Sawicka, Tałałaj, Wojnar, & Nowicka, 2018). These findings were observed also in animal models (Turner, Young, McGrath, Eyles, & Burne, 2013). Data from different studies showed a possible role of vitamin D deficit, both in adult and developing brains (Groves, McGrath, & Burne, 2014). Data arising from animal models support the neurochemical role of vitamin D, acting in the excitatory/inhibitory brain system with an impact on impulsive behaviours regulation (Groves et al., 2014; Turner et al., 2013). As shown in other clinical populations, such as patients with ADHD or depression, our results indicate that a deficit of vitamin D is linked with increased impulsive behaviours (Goksugur et al., 2014; Patrick & Ames, 2015). Literature also reports a causative link between obesity and low levels of vitamin D (Vimaleswaran et al., 2013). Our data confirm this association, however they cannot prove a direct link between impulsivity and low levels of vitamin D. When we adjusted for obesity in the regression model, the association was no longer significant ($p = .07$), although this could be at least partially due to lack of statistical power. Further studies should be designed to disentangle the effect of impulsivity and obesity on vitamin D.

To date, vitamin D has been studied only for its impact in bones mineralization of ED patients (Veronese et al., 2015), however, present data highlight a possible role also in their psychopathology. If confirmed, these results may have relevant implication in the therapeutic approach and introduce a new way to think about vitamin D and its relevance in the management of patients with ED. Indeed, impulsivity is considered a multidimensional construct implicated in the onset as well as in the outcome of different ED (i.e., BED more than restrictive AN) (Schag, Schönleber, Teufel, Zipfel, & Giel, 2013; Waxman, 2009; Wonderlich, Connolly, & Stice, 2004). Impulsive behaviours have been proposed to have a core role in the ED spectrum due to the neuroimaging evidences of an imbalance of the frontal and mesolimbic area (Brooks, Rask-Andersen, Benedict, & Schiöth, 2012) and our data can show a possible link between this relationship. Supplementation with vitamin D could be considered as part of the therapeutic approach to reach symptoms control and relapse prevention, in individuals with ED, as already tested in other groups of patients with an effect on impulsivity, inattention and hyperactivity both in adults and children (Patrick & Ames, 2015).

As far as the serum levels of vitamin D and impulsive behaviours, a first analysis was performed based on suggested clinical levels for bone metabolism regulation (Modan-Moses et al., 2015) but no result was found with that cut offs. Instead, our data showed a concentration of 20 ng/mL as discriminating level for impulsivity, which is lower than suggested by international guidelines for bones health (Patrick & Ames, 2015). These results are in

TABLE 3 Binary logistic regression predicting the presence of clinical indicators of impulsivity from vitamin D after adjusting for age and Obesity

Source	Value	SE	*p* value	OR	OR lower 95%CI	OR upper 95%CI
Vitamin D	−0.0158	0.0089	.0710	0.9843	0.9673	1.0005
				1.0159	1.0338	0.9995
Age	0.0160	0.0141	.2546	1.0162	0.9885	1.0446
Obesity	2.8369	1.0378	.0063[a]	17.063	2.2320	130.449

Abbreviations: CI, Confidence Interval; OR, Odds Ratio; SE, standard error.
[a]Significant at 0.05 level.

agreement with previous studies indicating similar serum level of 25-OH vitamin D in children with ADHD and high impulsivity levels as well as in adults with suicidal behaviours (Goksugur et al., 2014; Villagomez & Ramtekkar, 2014). Thus, the vitamin D concentration reference for bone health may not be valid when studying the neurochemical role of vitamin D and in particular its concentration levels implicated in impulsivity regulation.

4.1 | Limitations

Our study has several limitations. First, data analysed in this study come from a single time point, thus they cannot give any information on the nature of the association between vitamin D and impulsive behaviours. Second, other factors, such as seasonality, nutritional data and body composition that could influence vitamin D levels, were not assessed. Another possible limitation could be the use of a clinical approach to assess impulsivity. The present study was inspired by clinical observations and data are part of a wider observational longitudinal study, not specifically designed for the purpose of the present study. Given the absence of a validated measure of impulsivity in this pilot exploratory study we decided to follow an approach similar to the one used by other authors (Favaro et al., 2005; Waxman, 2009) and based on clinical indicators of impulsive behaviours.

5 | CONCLUSIONS

With this study, we aimed to generate preliminary data to support the hypothesis that vitamin D may be implicated in the regulation of impulsive symptoms in ED patients, as already found in other groups of patients. Although statistical significance is achieved when not considering obesity, this is lost when it is considered as a covariate, with a minor reduction in effect size that caused the *p*-value to marginally not pass the threshold. Therefore, we cannot derive any definitive conclusion on whether the effect is reduced when adjusting for obesity due to the loss of power of adding a covariate, or whether the result is spurious. We therefore conclude that there are strong indications that further research is needed.

Our results partially confirmed that the relationship between impulsivity and low vitamin D levels could be explained by obesity/body composition of subjects. Based on the findings from this preliminary work, we designed a protocol to specifically investigate the relation between impulsivity and vitamin D levels with specific psychological assessment of impulsivity and a larger sample size, in order to reach a higher statistical power: this includes a validated questionnaire and neuropsychological tasks for impulsivity assessment and the determination of vitamin D at two time points (admission and discharge from the ED Unit). A deeper understanding of the role of vitamin D in behavioural symptoms, and in particular in impulsive behaviours, may inform the use of vitamin D supplementation in symptoms management of ED patients.

CONFLICT OF INTEREST

The authors declare no potential conflict of interest.

ETHICS STATEMENT

All patients gave written informed consent before the evaluation and the study was conducted in accordance with the Declaration of Helsinki.

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