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First do no harm: the impact of assessing for ultra-processed food addiction on dietary restraint in patients with and without eating disorders during residential treatment

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Abstract

Objective Ultra-processed food addiction (UPFA) is increasingly recognized but remains controversial among eating disorder (ED) clinicians, partly due to concerns that introducing food addiction concepts might intensify dietary restraint, a core feature of EDs. This study examined whether integrating UPFA assessment, psychoeducation and treatment into residential treatment (RT) impacts ED symptomatology, particularly dietary restraint.

Methods Adults (N = 132) admitted to RT completed the Eating Disorder Examination Questionnaire (EDE-Q) and the modified Yale Food Addiction Scale 2.0 (mYFAS2.0) at admission and discharge. Changes in EDE-Q global scores and EDE-Q restraint subscale scores were analyzed using repeated-measures analysis of variance (RANOVA) with ED diagnosis as a between-subjects factor and age, gender identity, sexual orientation, and admission BMI as covariates. Pearson correlations between mYFAS2.0 and EDE-Q scores were calculated at both time points to assess construct overlap.

Results Both EDE-Q global and EDE-Q restraint scores decreased significantly in the ED patients ($p \le 0.001$) and remained low in the non-ED patients. mYFAS2.0 scores were not significantly correlated with EDE-Q restraint scores at either admission or discharge, while they were weakly correlated with EDE-Q global scores at both time points.

Conclusions UPFA-informed assessment, psychoeducation and/or treatment did not increase EDE-Q restraint scores. Instead, they declined significantly in ED patients, with no worsening observed. Modest post-treatment correlations between UPFA and EDE-Q global scores but not EDE-Q restraint scores suggest partial but incomplete overlap between EDs and UPFA symptomatology, with caloric restraint confined to EDs alone. These results support their nosological distinction and the feasibility of concurrent treatment without worsening ED-related dietary restraint.

Plain Language Abstract

Ultra-processed food addiction has been gaining increased popularity and scientific study over the last two decades. Despite a large body of research examining how to characterize and identify food addiction, treatment studies remain scant. Whether it represents a valid clinical condition, related to yet distinct from eating disorders

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remains controversial, particularly among eating disorder professionals. In part this concern stems from concerns that introducing the concept of food addiction and its treatment might worsen elements of dietary restraint as a core feature of eating disorders. In this study, we examined dietary restraint and food addiction using standardized measures in a population of patients with and without eating disorders in a residential treatment center specializing in care for people with complex, co-occurring mental health, eating disorder and/or addiction problems. We asked 132 adults to complete questionnaires at the start and completion of residential treatment, including guestionnaires about dietary restraint and food addiction, to study whether introducing assessments for and treatment of food addiction in a residential treatment center worsened dietary restraint scores in people with eating disorders. We used statistical methods to determine whether there were significant changes in dietary restraint for patients with eating disorders introduced to food addiction concepts during residential treatment. We found significant decreases in dietary restraint for people with eating disorders in our setting from start to end of residential treatment. This study adds to a small body of research that has begun to examine the impact of food addiction treatment on people with eating disorders, a population that has been shown to have high rates of cooccurring food addiction. Further research including clinical trials of people with co-occurring eating disorders and food addiction will be important, since there remains a relatively large percentage of people with eating disorders who do not respond to current evidence-based treatments.

Keywords Ultra-processed foods, Food addiction, Restraint, Eating disorders

Introduction

Ultra-processed food addiction (UPFA) is a controversial clinical construct characterized by addiction-like patterns of consumption of ultra-processed foods (UPFs), such as pastries, packaged snacks, and sugar sweetened beverages [13, 18]. The severity of UPFA symptoms can be reliably measured using the modified Yale Food Addiction Scale 2.0 (mYFAS2.0) [6, 17, 24, 27]. Notably, the mYFAS2.0.is based on DSM-5 criteria for substance use disorders (SUDs), as defined by broad symptom categories of craving, tolerance, failure to fill role obligations, continued use despite negative consequences along with significant impairment and distress [8, 20, 23].

Neurobiological and clinical parallels between UPFA and other SUDs are well-documented including high rates of co-occurrence, shared patterns of neural dysregulation, and high rates of comorbid post-traumatic stress disorder (PTSD), mood disorders, anxiety disorders and eating disorders [4, 14, 15, 19, 25, 31]. However, the diagnosis remains particularly controversial among ED clinicians (Brewerton et al. 2024; Cassin et al. 2019). Despite studies showing frequent co-occurrence of binge-type EDs and UPFA, there are notable differences in each construct's diagnostic criteria (Kalan et al. 2024; Ratkovic et al. 2023; Schulte et al. 2020).

Despite substantial and growing peer-reviewed research indicating that UPFA is a valid clinical construct, it remains controversial, particularly among ED clinicians. Among the objections cited in the literature, eating disorder researchers have raised concerns about lack of evidence for a specific addictive nutrient in humans, risk of pathologizing normal appetite or dieting struggles, and potential harm from adopting abstinence-based addiction treatments in ED populations. Meule [21] outlines the potential risks of abstinence-based

models applied to BED or BN, and the contrast of such models to cognitive-behavioral therapy (CBT) as an evidence-based treatment. CBT targets reducing pathological dietary restraint to achieve flexible and moderate food consumption with no forbidden foods. Dietary restraint refers to the intentional and cognitive effort to restrict food intake for the purpose of controlling body weight or shape, rather than actual caloric restriction per se, and has been associated with eating disorder pathology and risk across diagnoses. Much has been written in the literature about the role of dietary restraint as a core transdiagnostic feature of eating disorders [11, 12]. UPFA as a construct often gets conflated with abstinence-based treatment models, despite the emergence of harm reduction treatment models in the substance use disorder and food addiction literature [16]. Abstinence-based models applied to UPFA have been hypothesized to be ineffective or even be harmful to individuals with BN and BED if they increase dietary restraint [21]. However, to our knowledge, this remains untested in clinical populations, and in fact, recent studies suggest that UPFA symptoms and dietary restraint are not associated and may reflect distinct psychosocial predictors and etiological pathways [26, 28, 29].

Dietary restraint can be measured using the restraint subscale of the Eating Disorder Examination Questionnaire (EDE-Q), a validated self-report tool. In the present study, we examined EDE-Q Global scores, EDE-Q restraint subscale scores, and mYFAS2.0 scores at admission and discharge among patients receiving residential treatment for eating disorders, SUDs, mood disorders, and/or trauma and stressor related disorders. We hypothesize that administering the mYFAS2.0 and providing psychoeducation about UPFA would not worsen dietary restraint for patients with ED.

Methods

Setting: The study was conducted at SunCloud Health in Northbrook, IL, USA, a free-standing residential treatment center for adults with complex co-occurring eating disorders, SUDs, mood disorders and trauma and stressor related disorders. This residential program specializes in integrated treatment for patients with multiple disorders happening simultaneously, rather than treating patients based on one "primary" diagnosis.

Ethics: This study was approved by the Brany Institutional Review Board. All participants gave written informed consent for the use of deidentified data from their assessments.

Table 1. Demographic and baseline clinical characteristics of patients with (n = 47) and without (n = 85) an eating disorder (ED) diagnosis using analyses of covariance with age, BMI, gender, and sexual orientation. (* = p < 0.05, ** p < 0.01, ***p < 0.001)

Variable	ED (Mean + SD)	No ED (Mean + SD)	t-score	Hedg- es' g
Age (years)	29.23 + 9.253	35.05 + 13.262	2.666***	0.485
Admit BMI	29.96 + 9.64	25.97 + 5.97	- 2.958***	- 0.535
	ED (n)	No ED (n)	Chi-square	Cram- er's V
Gender			16.193***	0.350
-Man	8	44		
-Woman	32	35		
–Transman	1	2		
–Non-binary	6	4		
Sexual Orientation			12.193*	0.304
-Heterosexual	16	49		
-Queer	20	26		
–Bisexual	5	3		
–Gay or Lesbian	4	1		
Pansexual	0	1		
Prefer not to say	2	5		
Race			5.229	0.199
-White	39	64		
–Black	0	6		
–Hispanic	2	4		
–Asian	3	3		
–Multi-racial	3	6		
-Prefer not to say	0	2		
Eating Disorder Diagnosis				
-AN-R	7			
-AN-BP	3			
-BN	4			
-BED	12			
OSFED	21			

Abbreviations: AN-BP = anorexia nervosa, binge-purge type; AN-R = anorexia nervosa, restricting type; BMI = body mass index; BED = binge eating disorder; BN = bulimia nervosa; OSFED = Other specified feeding and eating disorder

Participants: The demographic and clinical characteristics of the total group are shown in Table 1. Participants included a total of 132 patients entering a residential level of care setting, including those diagnosed with [N=85] and without [N=47] a current ED, which was determined by admitting psychiatrists using a structured interview based on DSM-5 criteria (American Psychiatric Association 2013).

Assessments: Patients completed validated self-report assessments at admission and discharge, including the Eating Disorder Examination Questionnaire (EDE-Q) [3] and the Modified Yale Food Addiction Scale 2.0 (mYFAS2.0) [27]. We used the diagnostic scoring method to interpret the mYFAS2.0 in this study [27].

Statistics: We used repeated-measures general linear models to evaluate whether EDE-Q Global and restraint subscale scores changed from admission to discharge, with ED diagnosis as a between-subjects factor and age, gender identity, sexual orientation, and admission BMI included as covariates. We also calculated Pearson "r" correlation coefficients between EDE-Q Global scores, EDE-Q restraint subscale scores, and mYFAS2.0 total scores at admission and discharge. For these correlation coefficients, the probability cutoff used was alpha ≤ 0.003 to correct for multiple comparisons. All statistical procedures were performed using SPSS version 29.0.

Procedure: We tested whether assessment with the mYFAS2.0 and exposure to a treatment environment including psychoeducation and individualized treatment for UPFA impacted EDE-Q global and dietary restraint subscale scores from admission to discharge in patients with and without diagnosed eating disorders. The average length of stay for participants in residential treatment was 33.6 days (SD = 14.8 days). Psychoeducation regarding eating, substance use, mood, and trauma and stressor related disorders was provided by a multidisciplinary team of therapists, psychiatrists, nurses and registered dieticians in a combination of group and individual sessions. Treatments were individualized, matched to each patient's needs based on their current DSM-5 and mYFAS2.0 UPFA diagnoses. Care consisted of the following modalities: psychotropic medication management, active management of medical comorbidity, CBT, dialectical behavior therapy (DBT), cognitive processing therapy (CPT), nutritional counseling, psychoeducation and mindfulness-based therapy. All patients received psychoeducation about size-inclusivity, weight stigma and the principles of Health at Every Size [1]. All approaches emphasized autonomy, flexibility, attunement to internal cues, and avoidance of calorie restriction. Every patient with ED, UPFA or both was provided with a meal plan in collaboration with a registered dietician sufficient to meet their estimated daily caloric needs. Frequency of vital signs, weight, and laboratory testing was specified

by the attending psychiatrist for each patient. Patients who had an ED and no UPFA were treated with nutritional plans aligned with an "All Foods Fit" model of care, incorporating 3 meals and 3 snacks a day. Patients meeting criteria for UPFA (with or without ED) could choose among three nutritional approaches: (1) an "All Foods Fit" approach, (2) a harm reduction approach (reducing the proportion of UPFs consumed while maintaining overall caloric intake), or (3) an abstinence-based approach (eliminating all identified problematic UPFs while maintaining overall caloric intake) [9]. Every person who met criteria for an ED, UPFA or both attended meal support group for all meals and snacks, nutrition group therapy, body image group therapy, and 1-2 individual meetings with an ED dietitian weekly. The patients with UPFA who opted for a harm reduction or abstinence approach had additional individual nutritional counseling and psychoeducation about UPFA as an emerging clinical construct, how to identify UPFs, and the impact of UPFs on reward circuitry and eating behaviors as reported in research studies involving human subjects (Ifland et al., 2025).

Results

Patients diagnosed with an ED (with or without UPFA) at admission tended to be younger and have a higher BMI than those without ED (with or without UPFA) (see Table 1). On measures of eating disorder severity, patients with EDs scored the highest on both EDE-Q Global and Restraint subscales. Patients with UPFA but no ED had elevated scores compared to patients with neither ED nor UPFA, but not as high as those with ED. The

highest symptom levels overall were seen in patients with comorbid ED and UPFA.

EDE-Q Scores: There was a significant overall reduction in EDE-Q restraint subscale scores across the full sample (both ED and non-ED patients) (F = 3.95, $p \le 0.05$, $\eta^2 = 0.03$). As seen in Fig. 1, ED patients showed a significantly greater reduction than non-ED patients, as reflected in a time \times ED diagnosis interaction (F = 11.57, $p \le 0.001$, $\eta^2 = 0.084$). Among ED patients, EDE-Q restraint scores decreased from 2.9 ± 1.9 to 2.0 ± 2.0 $(F = 23.295, p \le 0.001, \eta^2 = 0.156)$, while scores for non-ED patients remained low and stable without significant change $(0.4 \pm 0.8 \text{ to } 0.3 \pm 0.8, \text{ NS}, \eta^2 = 0.003)$. There was a reduction in EDE-O Global scores in the total sample of both ED and non-ED patients between admission and discharge, but this did not reach statistical significance $(F=2.8, p \le 0.1, \eta^2=0.022)$. There was also no significant time x ED diagnosis interaction for EDE-Q Global scores (F = 0.885, NS, η^2 = 0.007). Independently, patients with ED showed a statistically significant reduction from 3.5 ± 1.5 to 2.9 ± 1.4 (F = 10.128, p \le 0.002, η^2 = 0.074), while non-ED patients showed no significant change between admission and discharge $(0.8 \pm 1.0 \text{ to } 0.6 \pm 0.8;$ F = 0.417, NS, $\eta^2 = 0.003$).

Correlation Between UPFA and ED Symptoms: As shown in Table 2, Pearson correlations indicated no statistically significant relationship between dietary restraint (EDE-Q restraint subscale) and food addiction symptoms (mYFAS2.0) at admission (r = 0.139, NS) and no significant relationship between EDE-Q global scores and mYFAS2.0 total score (r = 0.297, NS). By discharge, mYFAS2.0 total scores were significantly correlated with

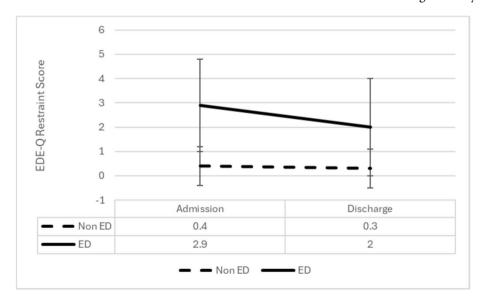


Fig. 1 Repeated measures analysis of covariance (RANCOVA) of EDE-Q restraint scores at admission and discharge for patients with an eating disorder diagnosis (n = 47) and without an eating disorder diagnosis (n = 85). Admission score \times ED patients (M = 2.9, SD = 1.9). Discharge score \times ED patients (M = 2.0, SD = 2.0). Admission score \times non-ED patients (M = 0.4, SD = 0.8). Discharge score \times non-ED patients (M = 0.3, SD = 0.8). Time: F = 3.95, p < 0.05; Time \times Diagnosis: F = 11.57, p < 0.001

Table 2 Pearson "r" correlation coefficients for admission and discharge variables, including the EDE-Q Global score, EDE-Q Restraint score and mY-FAS symptom count score. Given the number of statistical comparisons, alpha was set at p < 0.003 (**p < 0.003, ***p < 0.001)

Variable	n	1	2	3	4	5	6
1. EDE-Q Global Score (ADM)	47	-	-	_	-	_	_
2. EDE-Q Restraint subscale (ADM)	47	0.805**	_	_	_	_	-
3. mYFAS 2.0 (ADM)	45	0.297	0.139	-	-		-
4. EDE-Q Global Score (DC)	47	0.509**	0.619**	0.152	-		-
5. EDE-Q Restraint subscale (DC)	47	0.331	0.591**	0.030	0.809***		-
6. mYFAS 2.0 (DC)	46	- 0.017	0.040	0.518***	0.429**	0.300	_

Abbreviations: ADM = Admission; DC = Discharge; EDE-Q = Eating Disorder Examination - Questionnaire; mYFAS 2.0 = modified Yale Food Addiction Scale Score 2.0

EDE-Q Global scores (r = 0.429, p = 0.003) but not with EDE-Q restraint subscale scores (r = 0.3, NS).

Covariate Effects: No significant moderation effects were found for age, gender identity, sexual orientation, or admission BMI on changes in EDE-Q global or restraint subscale scores.

Discussion

To our knowledge, this is the first study to evaluate the impact of assessing for UPFA using the mYFAS2.0, UPFAfocused psychoeducation, and treatment in a milieu including a variety of nutritional approaches-all foods fit, reduced UPF and abstinence-based approaches-on a measure of dietary restraint for patients with and without ED in a residential treatment setting. This is clinically relevant since dietary restraint has been studied extensively as a transdiagnostic feature that increases risk for and maintains ED pathology. Our findings show that assessing for and addressing UPFA did not increase dietary restraint, as measured by the EDE-Q restraint subscale, in patients with ED or ED/UPFA. In fact, dietary restraint significantly decreased in both groups, as would be expected with eating disorder treatment. This may reflect our combined approach in which we incorporated food addiction assessment and education into traditional eating disorder treatment (which typically excludes it) and incorporated eating disorder education and treatment, including avoiding caloric restriction and weight neutrality, into food addiction treatment (which often includes focus on weight loss and calorie restriction). Treatment studies for UPFA are limited to date, but emerging results are promising [28, 30]. Given the overlap between ED and UPFA, and well documented risks associated with calorie restriction for patients with ED, we underscore the importance of avoiding caloric restriction in the provision of care for patients with co-occurring ED and UPFA. It is important to note that nutritional plans tailored to the needs or preferences of a patient with ED/UPFA cooccurring could include reduction of UPF intake without reducing overall caloric intake, to minimize risks associated with calorie restriction in the ED population.

Our study found no significant correlations between mYFAS2.0 scores and EDE-Q restraint subscale scores at either admission or discharge. This would support the notion that UPFA and EDs are distinct with regards to the absence or presence of dietary restraint. Not surprisingly, modest but statistically significant correlations were found between mYFAS2.0 and EDE-Q Global scores at both admission and discharge, which is not unexpected given the overlap between UPFA and EDs, particularly binge-type EDs [5, 7, 16] (Muele 2024). Overall, our results support a model that the two conditions often co-occur yet remain nosologically distinct with respect to dietary restraint, and that concurrent treatment in a residential setting does not increase dietary restraint for patients with ED.

Our study adds to the emerging literature documenting that despite much overlap between ED and UPFA diagnoses, there are areas of clinical distinction [29]. Modest post-treatment correlations between UPFA and ED symptoms suggest partial but incomplete overlap between the two, with dietary restraint being distinct to EDs alone. These results support their nosological differences.

Despite concern about potential for harm of including UPFA assessment and treatment in ED populations, our study in a RT center for complex co-occurring disorders showed that dietary restraint significantly decreased for both patients with ED alone and those with UPFA/ED. Treatment studies for UPFA are largely lacking, and for the few that have been published, patients with ED are typically excluded (Skinner et al. 2024). Therefore, understanding the impact of diagnosing and treating UPFA among ED treatment seeking populations is crucial, given the high percentage of patients with binge-type EDs who also meet criteria for UPFA [7, 10, 22]. We posit that overlooking this comorbid condition in ED treatment contributes to the high rates of non-response and relapse observed in standard evidence-based care for binge-type EDs (Brewerton et al. 2024) [16]. Assessment of UPFA in patients in an ED RT can enhance diagnostic accuracy without worsening dietary restraint. Incorporating UPFA assessment may improve clinicians' capacity to design

individualized care plans and improve treatment outcomes, particularly in patients who do not improve after standard ED treatment protocols.

This study has several limitations that are worthy of consideration. The sample size was relatively small and consisted of a large percentage of white, privately insured patients. Therefore, our findings may not be generalizable, and further research with patients from more diverse racial backgrounds is warranted. Secondly, our nutritional treatment interventions were not randomized (or blinded) but driven by patient preference in collaboration with medical nutritional therapy provided by ED dieticians [9]. Randomized treatment trials are needed. In addition, long term treatment outcomes are needed to assess outcomes at 6- and 12-months post-discharge and beyond. This study also highlights the limits of our current ED assessment tools with regards to distinguishing pathological versus adaptive dietary restraint, which have been echoed by other researchers [2]. Dietary restriction is a well-evidenced maintenance factor for eating disorder pathology initially recognized in Fairburn's transdiagnostic model of EDs [11, 12]. While the EDE-Q is a valid and reliable measure of eating disorder related pathological dietary restraint, we are lacking in more nuanced measurements of dietary restraint that is potentially adaptive. For example, while skipping full meals may be a form of pathological restraint, maintaining overall meal structure, and overall caloric intake while reducing the amount of UPF intake may be an example of adaptive restraint for a person with UPFA and BED. Future research should focus on the development and study of measures which can distinguish between pathological dietary restriction and nonpathological dietary restraint in the clinical context of treating patients with EDs, ED risk factors and/or UPFA. This distinction would be a critical aid in mitigating risk of harm and guiding appropriate nutritional interventions when working with patients exhibiting disordered eating behaviors. As other researchers have noted, our current ED assessment tools are incapable of distinguishing between pathological and adaptive dietary restriction[2, 26].

In conclusion, we present evidence that in the context of UPFA-informed evaluation, psychoeducation and/or treatment, EDE-Q restraint scores decreased in both ED and non-ED patients. The lack of significant correlations between symptomatic measures of UPFA and ED-related restraint adds to the data supporting the nosological distinction between UPFA and EDs. Although the symptomatology of UPFA and EDs partially overlap, caloric restraint appears to be confined to EDs alone. These results support their nosological distinction and the feasibility of concurrent treatment without worsening ED-related dietary restraint.

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Author contributions

Conceptualization, K.D., T.B., and N.B.; methodology, K.D., N.B., and T.B.; software, C.N. and D.B.; validation, C.N., T.B., N.B. and K.D.; formal analysis, K.D., N.B., C.N., D.B. and T.B.; investigation, K.D. and T.B.; resources, K.D. and T.B.; data curation, C.N., D.B. and T.B.; writing—original draft preparation, K.D.; writing—review and editing, K.D., N.B., D.B. and T.B.; supervision, K.D. and T.B.; project administration, K.D.; funding acquisition, K.D. All authors have read and agreed to the published version of the manuscript.

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Data availability

The data presented in this article are not readily available because they are part of an ongoing study.

Declarations

Ethics approval and consent to participate

The study was conducted in accordance with the Declaration of Helsinki and approved by the Brany Institutional Review Board (File # 23-12-396-1474, approved 6 June 2023) for studies involving humans. Informed consent was obtained from all subjects involved in the study.

Competing interests

The authors declare no competing interests.

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