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ORIGINAL ARTICLE

Changes in unhealthy exercise in outpatient eating disorder treatment: examining emotion avoidance mechanisms

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Abstract

Unhealthy exercise (UE) is a common symptom of eating disorders (EDs) associated with elevated chronicity and relapse. Previous research suggests that UE serves an emotion regulation function, especially to reduce negative affect. UE may be especially prevalent among individuals with anorexia nervosa (AN), as UE and restrictive eating may be used to avoid unpleasant emotions. It is unclear whether changes in emotion avoidance (EA) can lead to changes in UE over time. The current study examined whether month 1 EA mediated the relation between baseline UE and month 2 UE in a clinical sample of 127 patients (M_{age} = 22.52 [SD = 11.75], 92.9% female) recruited from an ED outpatient treatment program. The current study also explored whether this mediation was stronger for individuals with AN than other ED diagnoses. Findings revealed that changes in EA significantly mediated the relation between changes in UE from baseline to month 2 of treatment. ED diagnosis did not moderate this relation. Findings suggest that improvements in EA may reduce UE over time. Treatment should target UE through cultivation of increased emotion acceptance.
Level of evidence Level III: evidence obtained from well-designed cohort study.

Keywords Unhealthy exercise · Eating disorders · Emotion regulation

Introduction

Unhealthy exercise (UE) is a common symptom of eating disorders (EDs) that is associated with greater chronicity and relapse [1, 2]. Rates of UE have been found to be as high as 80% of individuals with anorexia nervosa (AN) and 40% of individuals with bulimia nervosa (BN; [1]). Although multiple terms have been used in the literature to label UE, including "excessive exercise," "compulsive exercise," and "exercise dependence" [3], most research has reached consensus that qualitative features (e.g., compulsive, rule-driven, rigid) define the relation between UE and eating pathology. Indeed, in a sample of women with and without EDs, experiencing guilt if exercise was not completed, exercising at inappropriate times, and adhering to strict exercise rules were features most characteristic of women with an ED [4]. The current study used the term UE, as it is broad enough to encompass the wide range of qualitative features that define UE (e.g., compulsive, rigid, rule-driven) and does not emphasize the quantitative features (e.g., excessive) that are less related to eating pathology. Given that UE can exacerbate complications from EDs [5], it is critical to understand the function of UE to inform treatment.

Recently, there has been an increase in both ED and non-clinical studies examining the role of UE in emotion processing [6–8]. Broadly, transdiagnostic conceptualizations of EDs have identified emotion regulation as a core function of ED symptoms [9]. For instance, in Haynos and Fruzzetti's [10] transactional model of emotion regulation in AN, UE is specified as a maladaptive coping mechanism used to prevent the onset of aversive emotions and elicit more positive emotions. Consistently, research on UE suggests this symptom may be negatively reinforced among those with an ED for its ability to experientially avoid negative affect [6, 7, 11]. For instance, ecological momentary assessment research indicates that exercising in response to perceived or anticipated negative affect is

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associated with greater levels of dietary restraint, weight/shape concerns, binge eating, and self-induced vomiting in the previous month [6]. Taranis and Meyer [12] also found that exercising to avoid negative emotions was positively correlated with severity of ED psychopathology and frequency of ED behaviors in a sample of college students. Thus, exercise to avoid aversive states may be a primary function underlying UE and its relation to EDs.

Although the relation between UE and emotion avoidance (EA) appears to be present across all ED diagnoses, there may be reason to believe this relation is stronger among individuals with AN than individuals with other ED diagnoses. First, higher rates of UE are typically found in individuals with AN than other EDs. Dalle Grave et al. [1] found that compulsive exercise was greater among individuals with AN (63.1%) than those with BN (39.3%), and eating disorder not otherwise specified ([EDNOS]; 31.9%). Similarly, in a large sample of women with EDs, the prevalence of UE ranged from 37.4 to 54.5% among individuals with AN and 20.2–24% among individuals with BN [13]. Although neither of these studies examined the mechanism underlying these differences in UE across ED diagnoses, the data suggest that individuals with AN may be more apt to use UE to serve some maladaptive function.

Second, distinct forms of emotion dysregulation thought to characterize different EDs may also support the hypothesis that UE is more strongly related to EA in individuals with AN than individuals with other ED diagnoses. Previous research suggests that individuals with AN are characterized by primary emotion avoidance (i.e., prevent the onset of aversive emotions), whereas individuals with BN or BED are characterized by secondary emotion avoidance (i.e., alleviate distress after it has emerged; [14, 15]). Compulsive, repetitive and rule-driven UE may be a more effective form of primary emotion avoidance used to prevent the initial onset of aversive states, similar to dietary restriction. Indeed, as outlined by the dopamine model of UE in AN delineated in rodent models of activity-based AN [16], excessive dietary restriction increases vulnerability to UE through the presence of hyperdopaminergic activity and associated increased neural reward response. Thus, in the presence of severe dietary restriction, UE may be used to maintain certain baseline levels of positive emotions, while simultaneously preventing the onset of negative emotions. Conversely, as outlined by the dual-pathway model of BN, individuals with BN/binge eating may be more apt to engage in impulsive compensatory behaviors following the use of binge eating to reduce present negative emotions [17]. Thus, for individuals with an ED other than AN, UE may be more strongly linked with attempts to solely compensate for food that has been eaten, rather than to avoid emotions. Additional research exploring the relation between UE across ED diagnoses is needed to further elucidate potential differences.

Despite the apparent strong link between EA and UE, research has yet to examine whether changes in EA can lead to changes in UE over the course of ED treatment. Longitudinally, there is evidence to suggest maladaptive emotion regulation is predictive of UE. In a 12-month examination of adolescent girls, month 1 emotion repression predicted month 12 UE when controlling for baseline levels of the constructs [7]. However, this study did not examine the impact of changes in EA on changes in UE over the course of ED treatment. Other studies have demonstrated that UE improves over the course of cognitive-behavioral and family-based treatments for EDs and UE-specific treatment, but none elucidated the mechanism accounting for improvements in UE [18–22]. Given the emphasis on swift and meaningful behavioral change in evidence-based ED treatments [9], most of the studies examining UE over the course of treatment found improvements in UE at intervals of 7–12 weeks [18–22]. As such, the examination of EA and UE over the first 2 months of treatment is warranted.

Together, findings suggest that individuals with EDs, especially AN, may use UE to avoid emotions. Questions remain about whether improvements in EA during the first 2 months of treatment for EDs can lead to decreases in UE. The current study examined associations between UE and EA among patients enrolled in an outpatient ED treatment program ($N = 127$). It was hypothesized that baseline UE would predict month 2 UE, mediated via month 1 levels of EA. An exploratory analysis on whether this association was stronger for individuals with AN compared to other ED diagnoses was conducted, but no hypothesis was formulated based on limited research on these relations. To determine temporal precedence, the reverse model of baseline EA predicting month 2 EA, mediated via month 1 UE was examined. Changes in UE were not expected to mediate changes in EA.

Method

Participants

Participants were 127 adolescent and adult patients recruited from an ED outpatient treatment program in an Appalachian region. Participants were drawn from a longitudinal treatment outcome study examining symptom improvement over the course of a behaviorally based ED treatment. Recruited participants ranged in age from 13 to 68 (M (SD) = 22.52 (11.74)) years with body mass indexes (BMIs) from 14.64 to 66.34 (M (SD) = 24.04 (8.44)). 92.9% of participants were female. Most of the sample (85.9%) identified as White, with 5.2% identifying as Black/African American, 1.5% as Asian, 0.7% as Native Hawaiian/Other Pacific Islander, and 0.7% as bi- or multi-racial. Regarding ED diagnosis, 37.8% met

criteria for AN, 20% met criteria for BN, 9.6% met criteria for binge eating disorder (BED), and 32.6% met criteria for other-specified eating disorder (OSFED), as based on Diagnostic and Statistical Manual of Mental Disorders-5 [23]. Participants were excluded from the study if they met criteria for acute psychosis or mania, a lifetime history of intellectual disability, autism spectrum disorder, or dementia, or any medical condition causing visual, hearing, or motor impairments.

Procedure

All patients enrolled in treatment at the outpatient ED treatment center who met inclusion criteria were invited to participate in the study during their psychological intake assessment. Participants (or their legal guardians) provided written informed consent/assent. Patients who did not wish to participate in the study received the same care/treatment for the ED; over the course of data collection for this study, less then 10 patients decided not to participate. After an unstructured clinical interview, participants completed a series of paper questionnaires on eating pathology, mental health, and emotion regulation. Measures were repeated monthly after psychotherapy treatment sessions. Treatment consisted of either family-based therapy (FBT), cognitive-behavioral therapy for EDs (CBT-E), or dialectical behavioral therapy (DBT), all which encourage swift behavioral change. Treatment was provided by a psychologist with speciality training in these evidence-based therapies.

Measures

The eating disorder examination-questionnaire-6 (EDE-Q; [24]). The EDE-Q is a 36-item measure of disordered eating behaviors and cognitions on a scale from 0 (no days) to 6 (every day) based on the past 28 days. Item #18 on the EDE-Q was used to assess the frequency of UE over the past 28 days ("Over the past 28 days, how many times have you exercised in a "driven" or "compulsive" way as a means of controlling your weight, shape or amount of fat or to burn off calories?"). Baseline UE represented UE in the 28 days prior to the start of treatment, month 1 UE represented UE over the first 28 days of treatment, and month 2 UE represented UE between days 28 and 56 of treatment. Item #18 on the EDE-Q has demonstrated convergent validity with other multi-item measures of UE, including the Compulsive Exercise Test and Commitment to Exercise Scale [25].

The acceptance and action questionnaire-II (AAQ-II; [26]). The AAQ-II is a 7-item measure of psychological inflexibility and experiential avoidance. Items assess an unwillingness to experience unwanted emotions ("I'm afraid of my feelings"), the ability to be present in the moment ("Worries get in the way of my success"), and a commitment to value-driven actions when experiencing stressful psychological events ("My painful experiences and memories make it difficult for me to live a life that I would value") on a scale from 1 (never true) to 7 (always true). In a large diverse sample, the AAQ-II demonstrated adequate internal consistency (αs = 0.78–0.88) and test–retest reliability over 3 (r = 0.81) and 12 months (r = 0.79; [26]). Convergent validity has also been established [26]. Internal consistency estimates for the AAQ-II in the current sample were excellent at baseline (α = 0.92), month 1 (α = 0.90), and month 2 (α = 0.96).

Data analytic plan

Mplus version 8.3 [27] was used to examine whether EA mediated the effects of treatment-related reductions in UE. Violations of data normality were examined prior to conducting analyses. Ratings of UE across all timepoints demonstrated skew and kurtosis values greater than values found to be problematic in SEM simulation studies (i.e., skew values >|2|, kurtosis values >|7|; [28]). To determine the appropriate distribution to model these variables, an iterative approach was used in which Bayesian information criteria (BIC) values were compared for models with progressively more complex ways of modeling nonlinear data (i.e., linear, Poisson, zero-inflated Poisson, negative binomial, zero-inflated negative binomial; [29]). BIC values were lowest when modeling UE using zero-inflated negative binomial regression. However, results did not differ substantively when using the zero-inflated negative binomial distribution and the linear distribution. Therefore, results of the models using a linear distribution are reported to ease interpretation.

An indirect effects model was estimated to examine whether baseline UE predicted month 2 UE through reductions in month 1 EA. The cross-lagged effects of EA on subsequent UE were also modeled to examine if the hypothesized effects were specific to UE reductions. All models were estimated after controlling for the effects of UE and EA at the prior timepoint. After estimating the main indirect effects model, sensitivity analyses were conducted to determine if diagnostic status (AN diagnosis versus other ED diagnosis) moderated the observed effects. All variables were treated as manifest variables. Given the skewed nature of UE in these data, robust maximum estimation (MLR) was used to estimate all models. The final mediation models were estimated with maximum likelihood estimation (ML) and 5000 bootstrap samples to provide stable and replicable asymmetric confidence intervals for the indirect effects [30]. Full information maximum likelihood estimation [31] was used to handle missing data in these analyses. Full information maximum likelihood estimation provides less biased parameter estimates compared to listwise deletion, pairwise deletion, and mean imputation [32].

Several indices were used to assess overall model fit, including the Chi-square value obtained using the likelihood ratio test (LRT), the comparative fit index (CFI), the standardized root mean square residual (SRMR), and the root mean square error of approximation (RMSEA). A nonsignificant Chi-square value indicates excellent model fit [33]. CFI values greater than 0.95 and SRMR values less than 0.08 indicate good fit. Further, RMSEA values less than 0.05 indicate good fit [32, 33]. RMSEA confidence intervals (CIs) were also reported, with a 90% lower bound CI value less than 0.05 suggesting good model fit cannot be ruled out, and an upper bound CI value greater than 0.10 suggesting poor model fit cannot be ruled out [33, 34]. When evaluating model fit, preference was given to CFI values, given evidence that RMSEA values and SRMR values can be inflated when samples sizes are small and variables are not normally distributed [35, 36].

Results

Pearson's correlations and descriptive statistics are presented in Tables 1 and 2. More than half of the sample endorsed UE at baseline (53.7%), with episodes ranging from 1 to 28 over the past 28 days. At baseline, 11 participants were missing UE scores and 68 participants were missing EA scores. At month 1, 73 participants were missing UE scores and 104 participants were missing EA scores. At month 2, 87 participants were missing UE scores and 115 participants were missing EA scores.

The mediation model examining baseline UE as a predictor of month 2 UE through month 1 EA demonstrated adequate fit to the data ($\chi^2 = 9.70$, df = 4, $p = 0.05$, CFI = 0.94, SRMR = 0.11, RMSEA = 0.11, 90% CI [0.01–0.19]; see Fig. 1). There was a significant indirect effect of baseline UE on month 2 UE through month 1 EA (β = 0.14, 95% CI [0.02–0.32]). However, there was no significant indirect effect of baseline EA on month 2 EA through month 1 UE (β = 0.04, 95% CI [−0.09 to 0.29]). Diagnostic status (AN diagnosis versus other ED diagnosis) did not significantly

Table 1 Pearson correlations among variables

Variables	1	2	3	4	5	6
Baseline UE	–	0.64***	0.64***	0.10	0.09	−0.04
Month 1 UE	–	–	0.80***	0.20	0.21	−0.12
Month 2 UE	–	–	–	0.31	0.25	0.49*
Baseline EA	–	–	–	–	0.87***	0.75**
Month 1 EA	–	–	–	–	–	0.92***
Month 2 EA	–	–	–	–	–	–

T scores reflect mean differences between AN and other ED diagnosis groups. No significant differences on any of the variables were found at any time point between individuals with AN and individuals with another ED diagnosis

UE unhealthy exercise, *EA* emotion avoidance

*$p < 0.10$

**$p < 0.01$

***$p < 0.001$

Table 2 Descriptive statistics

Variables	Total sample		Anorexia nervosa		Other ED diagnosis		*T*
	M (SD)	Range	M (SD)	Range	M (SD)	Range	
Baseline UE	5.22 (8.32)	0.00–28.00	4.83 (8.12)	0.00–28.00	5.46 (8.48)	0.00–28.00	−0.52
Month 1 UE	3.16 (6.36)	0.00–28.00	2.74 (3.61)	0.00–14.00	4.0 (8.07)	0.00–28.00	−1.93
Month 2 UE	2.32 (5.92)	0.00–28.00	1.40 (3.44)	0.00–14.00	3.33 (7.74)	0.00–28.00	−1.25
Baseline EA	27.59 (11.00)	7.00–48.00	24.87 (10.09)	7.00–39.00	29.14 (11.27)	8.00–48.00	−1.27
Month 1 EA	28.67 (11.29)	7.00–46.00	21.18 (10.10)	7.00–39.00	35.00 (8.03)	14.00–46.00	−2.02
Month 2 EA	22.94 (13.94)	7.00–49.00	21.20 (13.89)	7.00–43.00	25.43 (14.70)	7.00–49.00	0.06

T scores reflect mean differences between AN and other ED diagnosis groups. No significant differences on any of the variables were found at any time point between individuals with AN and individuals with another ED diagnosis

UE unhealthy exercise, *EA* emotion avoidance

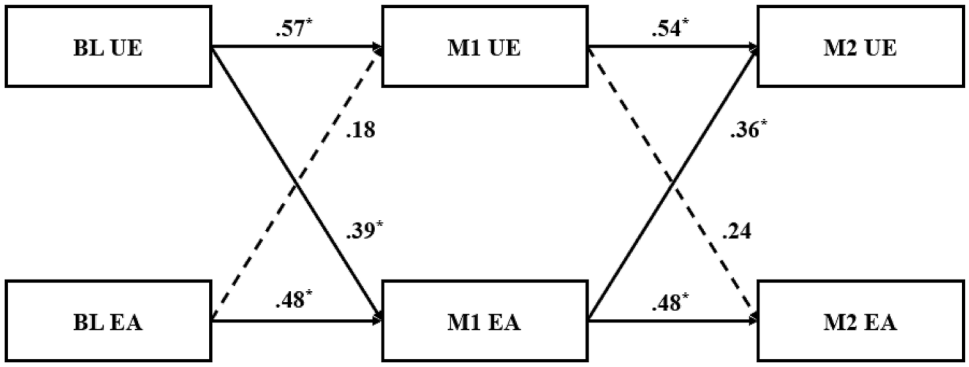

Fig. 1 Mediation model for the indirect effect of baseline unhealthy exercise on month 2 unhealthy exercise through month 1 emotion avoidance. *BL UE* baseline unhealthy exercise, *M1 UE* month 1 unhealthy exercise, *M2 UE* month 2 unhealthy exercise, *BL EA* baseline emotion avoidance, *M1 EA* month 1 emotion avoidance, *M2 EA* month 2 emotion avoidance. Standardized effects are reported. Cross-sectional covariances omitted for clarity. Solid lines indicate significant paths. $^{*}p < 0.05$

moderate the observed indirect effect ($\beta = 0.08$, $p = 0.83$); therefore, the indirect effect model without ED diagnosis was selected as the most parsimonious model. The final model accounted for 60.9% of the variance in UE and 40.0% of the variance in EA at month 2.

Discussion

This study examined the impact of changes in EA on changes in UE over the course of a behaviorally based outpatient ED treatment. Month 1 EA mediated the relation between baseline UE and month 2 UE. This relation did not differ among those with AN versus one of the other ED diagnoses (BN, BED, and OSFED). Month 1 UE did not mediate the relation between baseline EA and month 2 EA. Findings reveal that EA may be a core function underlying the symptom of UE among those with EDs. Targeting EA in ED treatment may result in UE symptom improvement, whereas changes in UE alone may not impact EA.

This study was novel in its longitudinal examination of associations among EA and UE in a clinical sample receiving outpatient ED treatment. The significant model of month 1 EA mediating the relation between baseline UE and month 2 UE is consistent with past research suggesting that UE serves an emotion regulation function [6, 7, 10], as well as extant models of ED symptom functions [9, 10]. As such, increases in emotion acceptance, awareness, and clarity through evidence-based treatment may be an important pathway to alleviating this often treatment-resistant symptom. Importantly, improvements in UE through improvements in EA were demonstrated at month 2 of treatment in the current study, which may be explained by the use of evidence-based treatments (i.e., FBT, CBT-E, and DBT) that emphasize fast behavioral change. This early symptom improvement is also consistent with past research finding improvements in UE at 7 weeks of treatment [20], and provides initial support that UE can be malleable within the initial months of treatment.

Previous research suggests that emotion-specific ED treatments (i.e., emotion acceptance behavior therapy) result in improvements in weight, disordered eating, and EA at treatment termination and 6-month follow-up, likely through increased knowledge and use of adaptive emotion regulation skills that decrease reliance on emotional suppression [37]. Our study adds to this literature by providing initial evidence that improvements in UE can be demonstrated over the course of a behavioral based therapy for EDs. Although FBT, CBT-E, and DBT are not exclusively emotion-specific treatments, they do provide skills training on emotion regulation to aid in successful completion of behavioral goals. Indeed, FBT incorporates parental validation and support of a child's ability to develop adaptive coping mechanisms for negative emotions at mealtime, CBT-E emphasizes cognitive-affective strategies to prevent disordered eating/exercise behaviors, and DBT incorporates both emotion regulation and distress tolerance skills training to decrease impulsivity and life-worth-living interfering behaviors. Thus, behaviorally based treatments that incorporate cognitive-affective components may decrease EA, which may improve UE and other aspects of disordered eating. However, research examining the utility of specific behavioral based therapies on UE uniquely is still needed.

The lack of significant indirect effect of month 1 UE on baseline EA and month 2 EA suggests that decreases in UE alone are not enough to significantly decrease EA. These findings are consistent with the results of Racine and Wildes [38] who found that emotion dysregulation predicted changes in AN symptom severity in the year following intensive treatment, but AN symptom severity did not predict changes in emotion dysregulation. Similarly, in Haynos and Fruzzetti's [10] transactional model of emotion regulation in AN, there is a strong emphasis on

the need to address emotion dysregulation even following weight restoration and a remittance of ED behaviors. One interpretation for this pattern of results is that behavioral treatment for UE may help with initial symptom interruption but may not sufficiently address the underlying function of the behavior. Effective treatment for UE may require cognitive-affective techniques that help patients acknowledge, identify, and adaptively cope with distressing emotions in alternative ways then UE. Indeed, a recent randomized-controlled trial of the healthy exercise behavior group therapy program, one of the few UE-specific treatments that tailors cognitive-behavioral principles and affective training specifically to UE, demonstrated enhanced UE improvement in adolescent and adult inpatients with an ED compared to treatment as usual [18]. Addressing EA specifically may also prevent the development of symptom substitution, or the use of other maladaptive EA behaviors (e.g., substance use, purging) after UE is no longer used.

Despite previous research suggesting a strong link between EA and AN [10, 14, 15], as well as higher rates of UE in AN than other ED populations [1, 16], the mediation effect was not moderated by diagnostic group. These results may be explained by the selected measure of UE, as previous research has demonstrated inconsistencies in differences in UE across ED diagnoses depending on the measure of assessment. In general, measures assessing more compensatory features of UE, such as exercise to control weight/shape and/or get rid of food that was just eaten, may be endorsed more significantly among individuals with BN and BED who engage in more frequent binge-compensatory behaviors; contrasting this, compulsive exercise to avoid guilt associated with missing regular exercise sessions may be endorsed at higher rates among individuals with AN who are characterized by habit-based mechanisms [39]. Since the measure of UE used in the present study was broad and unspecific, there may have been few differences in UE to result in a moderated effect.

Alternatively, it may be that emotion dysregulation underlies UE among individuals across the ED diagnostic spectrum and differences in distinct forms of emotion dysregulation do not impact the function of UE. Consistently, the current study found no significant differences in EA at any time point between individuals with AN and individuals with one of the other ED diagnoses, suggesting that EA was high and relevant to UE across the entire sample. Although extant models have established the role of emotion dysregulation in EDs broadly [9, 14, 15], additional research exploring differences in emotion dysregulation across diagnostic groups is needed to further elucidate symptom function and presentation among distinct EDs.

Limitations

Some limitations should be considered when interpreting the results of this study. First, UE was measured by a single item, which does limit the reliability of this measured construct. However, other studies have demonstrated that using one or two items to assess ED-related variables, including item #18 on the EDE-Q, is a reliable approach [40]. Regardless, results should be replicated using a multi-item measure of UE, such as the Compulsive Exercise Test [41]. Second, baseline episodes of UE in this sample were lower than what is typically seen in other ED patient populations. This may be due to an Appalachian cultural influence where physical activity is not as engendered, and exercise levels are typically lower than other urban regions [42]. Examining the link between EA and UE in a sample with higher levels of UE will be an important next step in prevention and intervention efforts for this symptom. Further, there was a large degree of dropout in the current study. Thus, some of the nonsignificant paths may be due to a lack of power. Finally, although the present study represented a true clinical sample seeking care in a rural medical center with broad inclusion criteria, most of the sample was White and ethnically homogenous. Examining these constructs in samples that differ in terms of sex, race, and ethnicity will be essential for generalizing findings.

Implications

Findings from this study have implications for future research and clinical practice. Given the detrimental outcomes associated with UE [1, 5], continuing to examine the functions of UE in ED populations is essential to combat this specific symptom and inform treatment options. Future research should delineate additional forms of emotion dysregulation apart from EA (e.g., impulsivity) that may contribute to the onset/maintenance of UE to inform intervention efforts. Consideration of other functions of UE outside of emotion regulation, such as personality traits, is also needed.

ED treatment should also work to deliberately incorporate UE interventions that are based on improving awareness and acceptance of emotions. Clinicians may consider tailoring existing evidence-based treatment protocols for improving emotion acceptance among patients with EDs (e.g., Emotion Acceptance Behavioral Therapy for AN; [37]) to specifically target the symptom of UE. Additional treatments created specifically for UE that work to improve acceptance of emotions and cultivate intentional, mindful movement for purposes other than to avoid aversive states and/or alter weight/shape should be developed. For instance, ecological momentary interventions that offer real-time emotion regulation training and coaching when the urge to engage in UE to

regulate emotions is high may increase emotion awareness, acceptance, and adaptive regulation. Through continued research and functional analysis of key ED symptoms, such as UE, evidence-based therapies for EDs will improve and provide relief for those impacted by these mental illnesses.

What is already known on this subject

UE may function to avoid negative emotions. Although UE has been found to improve over the course of evidence-based ED treatment, no study has identified the mechanism underlying changes in UE across treatment. Research examining the impact of changes in EA on UE over the course of ED treatment is needed to inform intervention.

What this study adds

This study demonstrates that reductions in EA over the course of ED treatment may result in reductions in UE across ED diagnoses. Emotion regulation may be a primary intervention effective for the reduction of UE.

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Declarations

Conflict of interest The author Shelby Martin has no conflict of interest. The author Jessica Luzier has no conflict of interest. The author Kevin Saulnier has no conflict of interest.

Ethical approval All the procedures performed in this study that involved human participants were in accordance with the ethical standards of hospital Institutional Review Board and with the 1964 Helsinki Declaration.

Informed consent All participants provided informed consent prior to participating in the study.

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