BRIEF REPORT



Differences in eating disorder symptoms and affect regulation for residential eating disorder patients with problematic substance use

Megan L. Michael¹ · Adrienne Juarascio²

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Abstract

Purpose The aim of the current study was to investigate differences in treatment outcomes for residential eating disorder (ED) treatment patients diagnosed with comorbid substance use disorders (SUDs), particularly differences in ED pathology and affect dysregulation.

Method Secondary data analysis was conducted on data from a previous study of 140 patients at a residential ED facility. SUD was diagnosed by a staff psychiatrist upon admission, and SUD diagnosis was extracted from electronic health records for the current study. Self-report measures of eating pathology and affect dysregulation from pre-treatment and post-treatment assessments were analyzed.

Results 20.1% of the sample (n=29) were diagnosed with a substance use disorder at the start of treatment. Contrary to hypotheses, those with comorbid SUD did not significantly differ in eating pathology severity, depression symptoms, emotion dysregulation, or psychological acceptance at baseline. Also contrary to hypotheses, individuals with comorbid SUD and ED evidenced slightly larger improvements in certain areas of eating pathology and affect dysregulation throughout treatment than those with ED diagnosis only.

Conclusions These findings suggest that residential ED treatment is an appropriate treatment choice for individuals with comorbid SUD. The observed improvements in affect dysregulation combined with a period of forced abstinence from maladaptive affect regulation behaviors may explain these positive results, though more research is needed to test the mechanisms of action of residential treatment for this population.

Level of evidence IV, multiple time series analysis.

Keywords Addiction · Eating disorder · Residential treatment · Substance use disorder

Introduction

Eating disorders (EDs) and substance use disorders (SUDs) have been found to be highly comorbid in both clinical and sub-clinical populations [1–3], and researchers have begun investigating theoretical models of shared risk factors.

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- Megan L. Michael mmichael1@crimson.ua.edu
- Center for Weight and Lifestyle Sciences, Drexel University, Philadelphia, PA 19104, USA
- Department of Psychology, Drexel University, Philadelphia, PA 19104, USA

Existing etiological models commonly hypothesize that affect dysregulation (i.e., deficits in awareness, acceptance, and regulation of negative emotional states; [4]) is a shared risk factor for both EDs and SUDs [5]. Affect dysregulation has been found to be associated with both disordered eating behaviors and problematic substance use [6, 7], and some studies have found that individuals with comorbid ED and SUD have difficulty accepting negative emotions [8, 9]. In addition, one study found that those with comorbid ED and SUD diagnoses are over five times more likely to have a concurrent depression diagnosis [10], a disorder characterized by difficulties identifying and utilizing effective affect regulation strategies [11]. When viewed collectively, these findings may lend support to etiological models wherein disordered eating and problematic substance use function



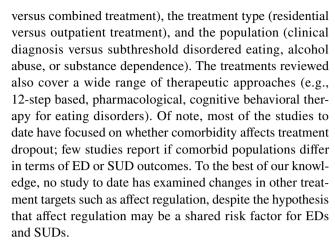
as maladaptive avoidant coping behaviors for regulating negative affect.

Clinically, comorbidity of these disorders is associated with increased severity of both ED and SUD symptoms [10, 12, 13]. A positive correlation has been noted between ED severity and number of adverse consequences related to substance use [10, 14], and comorbidity of these disorders has been linked to increased risk of overdose [15] and mortality [16, 17]. Since the evidence shows that this is a vulnerable at-risk population with worse clinical presentation and more severe consequences, it has been frequently hypothesized that comorbid SUD and ED would be associated with poor response to conventional treatments. However, there remain relatively few studies on treatment outcomes for this population.

The majority of the existing studies on treatment outcomes for comorbid SUD and ED have focused on how SUD treatment impacts comorbid ED symptoms. The general consensus from these studies is that comorbid ED symptoms are associated with SUD treatment dropout [18–20] and decreased SUD improvements [21], yet one study did find that outpatient alcohol abuse treatment was effective in improving comorbid ED symptoms [22]. The literature on ED treatment outcomes for individuals with comorbid SUD is even more limited. One study of outpatient ED treatment found that comorbidity predicted dropout [23], while another study found that outpatient ED treatment may have a positive effect on reducing drinking frequency for those with high alcohol consumption [24]. Only one study has examined outcomes from a residential ED treatment [25]. This study found that even though comorbid SUD was associated with higher eating pathology severity at baseline and end of treatment, those with comorbid SUD actually had significantly faster rates of symptom improvement over the first 4 weeks compared to those with ED diagnosis only [25]. These results markedly differ from the other studies reviewed, suggesting that residential ED treatment may not only be effective for individuals with a comorbid ED and SUD, but potentially even more effective than for individuals with an ED alone.

In addition to the studies described above that tested outcomes from either an SUD or ED-focused treatment, two studies have examined outcomes from treatment approaches that were tailored to directly target both substance use and disordered eating. Both these studies found positive outcomes, with participants improving in ED attitudes and behaviors, SUD severity, and affect regulation [26, 27]. However, it is difficult to know if resources should be dedicated to further development of concurrent treatment approaches, without a better understanding of clinical outcomes from existing focused treatment modalities.

In sum, clinical outcomes appear to vary depending on the disorder targeted (SUD treatment versus ED treatment



The primary aim of the current study was to examine differences in ED pathology and affect dysregulation treatment outcomes at a residential ED treatment center between patients with comorbid SUD and those with ED diagnosis only. We hypothesized that individuals with comorbid SUD would present with more severe eating pathology and affect dysregulation at the start of treatment. We further predicted that individuals with comorbid SUD would evidence less improvements in eating pathology and affect dysregulation throughout treatment.

Methods

Study setting and population

We conducted secondary analyses on parent study data previously collected by Juarascio and colleagues from a residential ED facility [28]. The parent study was approved by the Institutional Review Board at Drexel University and by the Core Research Committee at the Renfrew Center. All participants signed written consent to participate in the study. Treatment for all patients included individual therapy, group therapy, family therapy, and nutritional counseling. The treatment provided covered a wide range of therapeutic types (e.g., feminist relational therapy, family systems therapy, dialectical behavior therapy, cognitive behavior therapy for eating disorders). As part of the original study, half of the patients received twice weekly acceptance and commitment therapy (ACT) groups and the rest received treatment as usual.

A total of 159 women with a diagnosis of anorexia, bulimia, or eating disorder not otherwise specified were admitted to the residential ED facility during the period of data collection, of which 140 consented to take part in the study. Comorbid psychiatric diagnoses were assessed by psychiatrics upon admission to the residential program. To examine differences in treatment outcomes for those with comorbid problematic substance use, we examined



electronic health records for comorbid diagnoses and created an SUD group that included any comorbid diagnosis of alcohol or substance abuse or dependence. The age range of the sample was 18-55 with an average age of 26.74 (SD=9.19). The sample was primarily Caucasian (89.3%, n = 125), with small proportions of other racial groups (African American = 3.6%, Asian = 2.1%, Hispanic = 2.9%, Other = 1.4%). The average length of ED was 10.75 years (SD = 9.08) with an average age of onset at 16.43 years old (SD=5.5). Participants had an average length of stay at the current residential facility for 28.5 days (SD = 14.01). The sample was relatively evenly divided between AN spectrum diagnoses (i.e., AN restrictive subtype, AN binge-purge subtype, subthreshold AN; 47.1%, n = 66) and BN spectrum diagnoses (i.e., BN purging subtype, BN non-purging subtype, subthreshold BN, and BED; 52.9%, n = 74).

Measures

Eating Disorder Examination Questionnaire (EDE-Q) assessed overall eating pathology as well as four core features of EDs: Restraint, Weight Concern, Shape Concern, and Eating Concern [29]. Internal consistency and test–retest reliability are both excellent [30], and the EDEQ is highly correlated with the EDE interview. Cronbach's alpha for the current study was .91 for the Global subscale.

Goldberg Depression Scale (GDS) measured depression symptoms [31]. This measure has demonstrated acceptable internal and external validity and adequate internal consistency [32]. Cronbach's alpha for the current study was .92.

Drexel Defusion Scale (DDS) is a self-report assessing an individual's ability to defuse or distance themselves from negative thoughts, feelings, and physiological reactions [33]. DDS has acceptable reliability [33]. Cronbach's alpha for the current study was .83.

Philadelphia Mindfulness Scale (PHLMS) assesses mindfulness, specifically present-moment awareness and non-judgemental acceptance [34]. Exploratory and confirmatory factor analyses support the two-factor structure. Good internal consistency and reliability were demonstrated in both clinical and non-clinical samples [34]. The current study used the Acceptance subscale as a measure of psychological acceptance. Cronbach's alpha for the current study was 0.82.

Acceptance and Action Questionnaire-II (AAQ-II) is a measure of psychological flexibility and assessed experiential avoidance [35]. It has demonstrated adequate reliability and validity [35]. Cronbach's alpha for the current study was .92.

Dimensions of Emotion Regulation Scale (DERS) assessed overall emotion regulation abilities and six dimensions of emotion regulation: nonacceptance of emotional responses, difficulties engaging in goal-directed behavior, impulse control difficulties, lack of emotional awareness,

limited access to emotion regulation strategies, and lack of emotional clarity [4]. It has demonstrated excellent internal consistency and good construct validity [36]. Cronbach's alpha for the current study ranged from 0.91 to 0.95 for the subscales.

Data analysis plan

Independent samples *t* tests were run to examine differences between groups at baseline, with comorbid SUD as the independent variable, and eating pathology and affect regulation variables (depression symptoms, emotion regulation, and psychological acceptance) as the dependent variables. Mixed-factorial ANOVAs were conducted to compare the main effects of SUD comorbidity and change in eating pathology and affect regulation throughout treatment. Since the original study noted trend level differences in eating disorder outcomes between conditions, we also ran the same analyses with treatment condition as an additional factor to assess for any potential impact of treatment condition.

Results

Over a fifth (20.1%, n = 29) of the participants were diagnosed with either comorbid alcohol (n = 22) or substance (i.e., amphetamines, cannabis, benzodiazepenes, cocaine, or polysubstance; n=7) abuse or dependence upon the start of treatment and were classified in the problematic substance use group (SUD) for the current study. Overall, the sample had high eating severity at baseline (M = 4.312, SD = 1.29). Contrary to our original hypothesis, those with comorbid SUD did not significantly differ in eating pathology severity than the ED-only group. Additionally, those with comorbid SUD did not significantly differ on affect regulation factors of depression symptoms, emotion dysregulation, and psychological acceptance. At a trend level, those with comorbid SUD scored higher on the DERS Impulsivity subscale (M=19.440, SD=4.457) compared to those with ED-only (M = 17.255, SD = 5.343) t(123) = -1.886, p = 0.062.Table 1 presents results from an independent samples t test comparing group differences at baseline.

In terms of change in symptoms over the course of treatment, all participants significantly improved in eating pathology and affect regulation. However, contrary to our original hypothesis, the SUD group improved slightly more than the ED-only group on overall eating pathology and other subscales of eating pathology and affect regulation. The comorbid SUD group exhibited significantly greater improvements in weight concern (p=0.038) and depression symptoms (p=0.038). At trend level significance, those with comorbid SUD reported larger decreases in overall eating pathology (p=0.087), eating concern (.064), and lack of



Table 1 Group differences at baseline

Variable	Pre-	treatment	Group differences						
	Sub	st. + ED		ED-o	nly				
	N	М	SD	n	М	SD	t	df	p
AAQ total	24	33.083	7.978	102	32.897	9.883	- 0.086	124	0.932
PHLMS accept	20	39.000	5.938	99	36.985	6.629	-1.260	117	0.210
DDS total	23	20.696	9.187	97	24.330	10.488	1.528	118	0.129
DERS nonaccept	25	20.660	6.688	101	18.797	6.908	- 1.215	124	0.227
DERS goals	25	17.960	3.568	101	17.505	3.953	-0.525	124	0.601
DERS impulse	25	19.440	4.457	100	17.255	5.343	- 1.886	123	0.062†
DERS aware	24	15.3958	5.548	100	17.510	5.855	1.604	122	0.111
DERS strategy	25	27.940	5.731	101	25.782	7.415	- 1.357	124	0.177
DERS clarity	25	14.840	2.794	102	14.250	2.968	-0.901	125	0.370
Goldberg total	21	53.381	17.597	92	48.400	19.871	-1.057	111	0.293
EDEQ restraint	23	3.939	1.799	99	4.063	1.769	0.301	120	0.764
EDEQ eating concern	23	3.896	1.337	99	3.806	1.361	-0.285	120	0.776
EDEQ shape concern	23	4.842	1.313	99	4.854	1.414	0.034	120	0.973
EDEQ weight concern	23	4.591	1.393	99	4.406	1.660	-0.495	120	0.622
EDEQ global	23	4.317	1.182	99	4.311	1.320	- 0.021	120	983

^{*}Correlation is significant at the 0.05 level (p < 0.05)

emotional clarity (p = 0.091), and greater improvements in psychological acceptance (p = 0.086). When we re-ran the mixed-factorial ANOVA including condition as a factor, there were no significant interaction effects, indicating

that these results were not impacted by treatment condition. Table 2 presents group means from the mixed-factorial ANOVA examining difference between groups for symptom change throughout treatment.

Table 2 Group differences from pre- to post-treatment

Variable	Pre-treatment							t-treatme	nt	Group × time differences					
	Subst. + ED			ED-only			Subst. + ED			ED-only					
	n	М	SD	n	М	SD	\overline{n}	М	SD	n	М	SD	\overline{F}	P	Effect size
AAQ Total	15	35.600	5.501	75	33.067	9.897	15	32.767	6.592	75	28.993	10.494	0.273	0.602	0.003
PHLMS accept	11	39.364	4.864	69	36.906	6.813	11	32.955	2.779	69	34.587	6.868	3.018	0. 086 †	0.037
DDS total	15	19.667	9.409	71	23.155	8.843	15	26.933	13.176	71	28.338	10.344	0.584	0.447	0.007
DERS nonaccept	14	20.750	6.142	76	18.849	6.709	14	17.357	4.971	76	15.901	6.598	0.045	0.832	0.001
DERS goals	14	19.286	2.562	76	17.184	4.039	14	16.214	3.191	76	15.618	4.277	1.170	0.282	0.013
DERS impulse	14	19.929	3.368	74	16.696	5.450	14	15.607	3.443	74	14.716	5.152	1.940	0.167	0.022
DERS aware	13	13.808	5.134	75	17.113	5.633	13	17.769	5.525	75	20.027	6.074	0.250	0.618	0.003
DERS strategy	14	29.643	4.199	76	25.743	7.587	14	23.357	5.372	76	22.467	7.845	1.832	0.179	0.020
DERS clarity	15	16.000	2.903	77	14.325	2.917	15	13.633	1.950	77	13.760	2.858	2.923	0.091†	0.031
Goldberg total	18	55.167	14.972	81	48.819	19.433	18	28.111	14.864	81	32.556	18.442	4.304	0.041*	0.042
EDEQ restraint	20	3.880	1.686	92	4.102	1.772	20	.820	.645	92	1.487	1.303	0.782	0.378	0.007
EDEQ eating concern	20	3.860	1.420	92	3.900	1.335	20	1.430	.735	92	2.115	1.279	3.493	0.064†	0.031
EDEQ shape concern	20	4.788	1.375	92	4.940	1.300	20	3.266	1.283	92	3.838	1.748	1.336	0.250	0.012
EDEQ weight concern	20	4.570	1.451	92	4.516	1.559	20	2.800	1.336	92	3.509	1.788	4.394	0.038*	0.038
EDEQ global	20	4.274	1.232	92	4.395	1.248	20	2.079	.858	92	2.740	1.380	2.986	0.087†	0.026

^{*}Correlation is significant at the 0.05 level (p < 0.05)

[†]Correlation is significant at the 0.10 level (p < 0.10)



[†]Correlation is significant at the 0.10 level (p < 0.10)

Discussion

The aim of the current study was to examine treatment outcome in the areas of eating pathology and affect regulation for ED patients diagnosed with comorbid SUD. At baseline, we found no significant differences in eating pathology or any of the affect dysregulation variables (albeit a trend level difference in impulsivity) between the two groups. This contradicted our original hypotheses as, based on the previous literature, we had expected that those with comorbid diagnoses would exhibit more severe eating pathology and dysregulated affect.

Also contrary to our hypotheses, we found that those with comorbid SUD improved more than individuals with only an ED in some domains of eating pathology and affect dysregulation. These findings, while opposite to outcomes typically observed in studies of outpatient ED treatment [24], are consistent with the results from the one other study of residential ED treatment [25]. Collectively, our findings suggest that individuals with comorbid ED and SUD respond increasingly well to treatments that target shared underlying pathology, such as affect dysregulation, and enforce abstinence from maladaptive coping behaviors (i.e., disordered eating and problematic substance use) through residential treatment supervision.

Given the evidence that both SUD and EDs can be maintained by affect dysregulation, one might expect that a treatment approach focused on improving affect dysregulation would be uniquely beneficial for this population. In the current study, approximately half of the patients did receive an additional group that included a focus on affect dysregulation (ACT) as part of the clinical trial that comprised the parent study. However, our current study did not find differences in outcomes due to treatment condition for ED patients with comorbid SUD. Of note, our study was underpowered for test of a three-way interaction, so the lack of significance could simply reflect low power. Additionally, the parent study tested a relatively low dose of ACT (twice weekly ACT groups plus treatment as usual at the residential facility compared to treatment as usual alone), which may have also reduced the ability to observe an effect of targeting affect dysregulation directly. However, another interpretation of these results may be that it is the forced abstinence inherent in a residential treatment program itself that is the key mechanism underlying the notable improvements observed in the SUD group. Future research is needed to test the mechanisms of action of residential treatment for this population and to evaluate the utility of emotion-focused treatments.

In addition to the limitations described above, there are other important limitations to note for the current study. Because our study was a secondary data analysis,

we were limited in the use of a small comorbid sample as well as the lack of measures pertaining to substance use severity and illness course. Although the length of stay at the residential treatment facility is consistent with other residential eating disorder treatment programs [25], future research should utilize longitudinal study designs to examine changes in treatment outcomes over the course of a longer residential program. Future research should also examine a larger comorbid ED and SUD sample, and a sample with a higher severity of substance abuse, to see if there are significant differences in eating pathology and affect regulation severity than an ED-only group. Perhaps further investigation may confirm the current study's findings and suggest that ED patients with comorbid SUD are not necessarily a more pathological sample, just one that requires specialized treatment to target underlying pathology.

Overall, the current study adds to the field of the literature on treatment outcomes for comorbid ED and SUD populations and lends support for the role of residential ED treatment for individuals with comorbid ED and SUD. The study also contributes novel findings by examining treatment outcome differences in affect dysregulation and finding large improvements in these constructs in individuals with comorbid ED and SUD. As individuals with comorbid eating disorder and substance use disorder are at a high risk for dropout [18–20, 23], it is especially important to investigate treatment approaches that produce successful outcomes for both diagnoses. Future research is needed to further clarify the mechanisms of action contributing to improvements in eating pathology and affect dysregulation and whether therapies that target affect regulation are beneficial for this population.

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Compliance with ethical standards

Conflict of interest The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Ethical approval The current study was a secondary data analysis of data from a previous study [25]. The previous study was approved by the Institutional Review Board at Drexel University and by the Core Research Committee at the Renfrew Center.

Informed consent Informed consent was obtained from all individuals included in the study.

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