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Do End of Treatment Assessments Predict Outcome at Follow-up in Eating Disorders?

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ABSTRACT

Objective: To examine the predictive value of end of treatment (EOT) outcomes for longer term recovery status.

Method: We used signal detection analysis to identify the best predictors of recovery based on outcome at EOT using five different eating disorder samples from randomized clinical treatment trials. We utilized a trans-diagnostic definition of recovery that included normalization of weight and eating related psychopathology.

Results: Achieving a body weight of 95.2% of expected body weight by EOT is the best predictor of recovery for adolescents with anorexia nervosa (AN). For adults with AN, the most efficient predictor of weight recovery (BMI > 19) was weight gain to greater than 85.8% of ideal body weight. In addition, for adults with AN, the most efficient predictor of psychological recovery was achievement of an eating disorder examination (EDE) weight concerns score below 1.8. The best predictor of recovery for adults with Bulimia Nervosa (BN) was a frequency of compensatory behaviors less than two times a month. For adolescents with BN, abstinence from purging and reduction in the EDE restraint score of more than 3.4 from baseline to EOT were good predictors of recovery. For adults with binge eating disorder, reduction of the Global EDE score to within the normal range (<1.58) was the best predictor of recovery.

Discussion: The relationship between EOT response and recovery remains understudied. Utilizing a transdiagnostic definition of recovery, no uniform predictors were identified across all eating disorder diagnostic groups.

Keywords: recovery; transdiagnostic; predictors

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rates from 24 to 78%. In an exploratory study, Couturier and Lock determined that maintenance of recovery at 4-year follow-up in a sample of adolescents treated for AN was best predicted by a combination of attaining a post-treatment weight greater than 90% IBW and an EDE restraint subscale within 1 standard deviation of normal. As noted, recovery implies the maintenance of improvements over a period of time. Suggestions for the length of time needed for maintenance of symptomatic improvement to constitute recovery include 4 weeks, 8 weeks, 3 months, and 1 year, but none of these have been established empirically.

Recovery from BN has also been defined in a variety of ways. Earlier studies most commonly defined treatment response as a significant (e.g., 80%) reduction in binge eating and purging from baseline to EOT. In contrast, more recent studies use a more stringent definition of recovery; that is, abstinence from binge eating and purging for at least 8 weeks, or more commonly abstinence for 4 weeks. Less strict definitions of recovery in BN sometimes utilized include the presence of 1 or less weekly symptom episode over the prior 2 to 4 weeks. For adults with BN, studies report estimated relapse rates from 30 to 63%, depending on the definitions of remission and relapse, suggesting that EOT outcomes may be poor predictors of longer term recovery. There are only two published RCTs of outpatient psychosocial treatments for adolescents. Each of these studies used the same marker for recovery, i.e., abstinence rates from binge eating and purging over the previous four weeks. As is the case for adult BN studies, only behavioral criteria were used in defining recovery for adolescents (that is, eating related psychological recovery was not included in the definition).

Researchers in BED also have not established a common definition for recovery from this disorder. Indeed, recovery is rarely chosen as a primary treatment outcome in BED. The most recent Cochrane review (2009), for example, identified its primary outcomes allowing comparisons among the reviewed psychological interventions for BED as “abstinence from binge eating at the end of the study” or “remission”. In some studies, references to abstinence from binge eating are used interchangeably with “recovery.” The definition of abstinence varies across studies. For example, abstinence has been defined as a lack of binge episodes (determined by reviews of patient food records) for the prior 1 week, no binges for the last 2 weeks of treatment, or no binges for the prior 28 days. Some studies require abstinence from objective eating episodes as defined by the EDE and others from both objective and subjective eating episodes. An emerging standard threshold in outcome studies of BED defines abstinence as the absence of objective binge episodes (as determined by the EDE guidelines) over the prior 28 days.

Recently, Bardone-Cone et al. proposed that full recovery can be defined transdiagnostically for all individuals with an eating disorder, i.e., when they are indistinguishable from healthy controls on indices reflecting behavioral and psychological aspects of eating disorders. These authors operationalized their definition of full recovery as follows: (1) no longer meeting diagnostic criteria for an eating disorder (AN, BN, or EDNOS); (2) no binge eating, purging (e.g., vomiting and laxative use), or fasting in the past 3 months; (3) a body mass index (BMI) of at least 18.5 kg/m² (a BMI of 18.5–24.9 is considered normal by the World Health Organization); and (4) scores within 1 SD of age-matched community norms on all the subscales of the EDE-Q. In this study, we examined predictors of transdiagnostic recovery status at follow-up based on behavioral, psychological, and weight measures gathered at the EOT in samples of patients treated in RCTs for adolescent AN and BN, and adult AN, BN and BED.

**Method**

**Participants**

Analyses are based on data from five randomized clinical trials treating eating disorders in either an adult or adolescent population. In all studies, the
analysis required that a participant have both EOT and follow-up data (FU). We decided to use data on all female patients receiving active treatment as long as we had sufficient follow-up data for the analysis. Table 1 outlines the participant samples used for analysis in this study (Adolescent AN N = 83; Adult AN N = 28; Adolescent BN N = 47; Adult BN N = 101; Adult BED N = 97).

**End of Treatment Predictors of Recovery**

Predictor variables for recovery at follow-up were measured in two ways: (1) at EOT and (2) as a change score calculated as the difference between measurements at EOT and baseline. Predictor variables used were the following:

**Physical.** BMI was computed as weight in kilograms divided by height in meters squared. Percent ideal body weight for age, sex, and height was determined by Centers for Disease Control and Prevention growth charts (http://www.cdc.gov/growthcharts/percentile_data_files.htm).

**Psychological.** The EDE is a standardized, validated investigator-based interview that measures the severity of the psychopathology of eating disorders.37,38 There are four subscales in addition to a global score.

**Behavioral.** In addition to psychopathology, the EDE measures frequency of key behaviors: objective binging and compensatory behaviors.

**Definition of Recovery Utilized as the Outcome in the Predictor Analysis**

We based our thresholds for recovery largely on the global and transdiagnostic definition described by Bardone-Cone above.32 Participants (except adults with AN) were considered recovered if they met the following standards for physical, psychological, and behavioral recovery at follow-up:

1. Weight: percent IBW >95% based on age, height, and gender using Centers for Disease Control standards;39
2. Psychological-Global Score on the EDE less than community norms + 1 SD37,38 and
3. No reported binges or compensatory behaviors as measured by EDE.

In the adult AN sample, so few participants (N = 8) met all three criteria that the analysis was done separately for each of the three standards. Similarly, a cut-point of BMI >19 was used for the definition of physical recovery rather than the more stringent %IBW>95% to allow a sufficient number of recovered for the analysis. Because the analysis of the adult AN sample was done with more flexible standards, these results should be considered preliminary.

**Data Analysis**

Independent sample t-tests and chi-square tests of independence were used to check for selection bias as not all participants in the original studies met the assessment criteria for this study. Prediction of recovery was determined using signal detection analysis (SDA). SDA is nonparametric process that uses recursive partitioning to determine subgroups that are more or less likely to have a specific binary outcome. SDA is an ideal technique to make medical decisions by evaluating the performance of diagnostic tests.40,41 Recursive partitioning, using the QROC program available at http://www.stanford.edu/~yesavage/ROC.html, independently selects the best cut-point for each predictor variable based on the weighted kappa values. Kraemer cites > .8 = almost perfect, .6-.8 = substantial, .4-.6 = moderate, and .2-.4 = fair, < .2 slight or poor.41 We consider only predictors with Kappa values > .4 (moderate) as clinically significant in our results. We used SDA to identify the most efficient predictors (behavioral, physical, and psychological) of recovery in each eating disorder sample. Efficient tests balance the two extremes of sensitivity (maximizing true positives) and specificity (maximizing true negatives).

**Results**

Table 2 displays descriptive statistics for the five samples. Three of the five samples used in this analysis: adolescent BN (N = 49), adolescent AN (N = 83), adult AN (N = 28), did not differ in terms of baseline characteristics from the samples in the original study. However, the sample used in
the adult BN SDA sample \((N = 101)\) differed from the original adult BN study sample because they were older \((t(145) = -2.9 p = .005)\), better educated \((\chi^2(1) = 5.8 p = .02)\), had a lower BMI \((t(107) = -3.1 p = .002)\), and reported less eating psychopathology as measured by the Global Score on the EDE \((t(145) = 2.5 p = .02)\), Eating Concerns subscale \((t(145) = 2.1 p = .04)\) and Shape Concerns subscale \((t(145) = 2.6 p = .01)\). The sample used for BED had lower eating psychopathology as measured by EDE shape concerns \(t(116) = 2.3 p = .02\).

Descriptive statistics for the predictor variables are presented in Table 3. Table 4 displays the results for the most efficient predictors for each diagnosis from the SDA analysis and Figure 1 illustrates the summary results for significant EOT predictors by age and diagnostic groups.

**Anorexia Nervosa**

The most efficient predictor of recovery for adolescent AN was weight attainment to 95.2\% of expected IBW (Kappa ES = .47). For adults with AN, the most efficient predictor of weight recovery (for adults this was a BMI > 19) was weight gain to greater than 85.8\% of IBW (Kappa ES = .78). In addition, for adults with AN, the most efficient predictor of psychological recovery was achievement of an EDE weight concerns score below 1.8 (Kappa ES = .70). There were no efficient predictors of cessation of compensatory behaviors with medium or large Kappa ES.

**Bulimia Nervosa**

For adolescent BN, the most efficient predictor was the absence of compensatory behaviors (Kappa ES = .52) and a reduction in EDE restraint score of more than 3.4 from baseline to EOT (Kappa ES = .52). Seventy-two percent of the participants who met both criteria were recovered, whereas none of the participants who did not meet either criterion were considered recovered. Thirty-five percent of those with one of the criteria met definition of full recovery. For adult BN, the best predictor was frequency of compensatory behaviors less than two times a month at EOT (Kappa ES = .41).
### TABLE 4. Results from SDA efficiency tests

<table>
<thead>
<tr>
<th>Sample</th>
<th>Predictor</th>
<th>N</th>
<th>P</th>
<th>Q</th>
<th>Sensitivity</th>
<th>Specificity</th>
<th>Efficiency</th>
<th>Kappa</th>
<th>PVP</th>
<th>PVN</th>
<th>FP</th>
<th>FN</th>
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</thead>
<tbody>
<tr>
<td><strong>Adolescent AN</strong></td>
<td>% IBW EOT &gt;=95.2</td>
<td>82</td>
<td>0.317</td>
<td>0.329</td>
<td>0.654</td>
<td>0.821</td>
<td>0.768</td>
<td>0.470</td>
<td>0.63</td>
<td>0.84</td>
<td>0.22</td>
<td>0.21</td>
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<td>Change in EDE shape concerns</td>
<td>83</td>
<td>0.313</td>
<td>0.699</td>
<td>0.846</td>
<td>0.368</td>
<td>0.518</td>
<td>0.161</td>
<td>0.38</td>
<td>0.84</td>
<td>0.48</td>
<td>0.09</td>
</tr>
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<td></td>
<td>Compensatory Behaviors PT &gt;=-1.13</td>
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<td>0.305</td>
<td>0.146</td>
<td>0.24</td>
<td>0.895</td>
<td>0.695</td>
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<td>0.5</td>
<td>0.73</td>
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<td><strong>Adult AN physical</strong></td>
<td>%IBW EOT &gt;=85.75</td>
<td>28</td>
<td>0.536</td>
<td>0.571</td>
<td>0.933</td>
<td>0.846</td>
<td>0.893</td>
<td>0.784</td>
<td>0.88</td>
<td>0.92</td>
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<td>EDE eating concerns EOT &lt;0.2</td>
<td>28</td>
<td>0.536</td>
<td>0.429</td>
<td>0.6</td>
<td>0.769</td>
<td>0.679</td>
<td>0.364</td>
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<td>0.429</td>
<td>0.467</td>
<td>0.615</td>
<td>0.536</td>
<td>0.081</td>
<td>0.58</td>
<td>0.5</td>
<td>0.18</td>
<td>0.29</td>
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<td>EDE weight concerns EOT &lt;1.8</td>
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<td>0.607</td>
<td>0.607</td>
<td>0.882</td>
<td>0.818</td>
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<td>BMI EOT &lt;20.12</td>
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<td>0.607</td>
<td>0.571</td>
<td>0.706</td>
<td>0.636</td>
<td>0.679</td>
<td>0.337</td>
<td>0.75</td>
<td>0.58</td>
<td>0.14</td>
<td>0.18</td>
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<td>%IBW EOT &lt;93.16</td>
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<td>0.636</td>
<td>0.679</td>
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<td>0.75</td>
<td>0.58</td>
<td>0.14</td>
<td>0.18</td>
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<tr>
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<td>Change in compensatory EOT &gt;0</td>
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<td>0.647</td>
<td>0.545</td>
<td>0.607</td>
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<td><strong>Adult AN behavioral</strong></td>
<td>Change in Global EDE &gt;-1.88</td>
<td>28</td>
<td>0.643</td>
<td>0.643</td>
<td>0.778</td>
<td>0.714</td>
<td>0.378</td>
<td>0.78</td>
<td>0.6</td>
<td>0.14</td>
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<tr>
<td></td>
<td>Change in BMI &gt;= 0.74</td>
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<td>0.643</td>
<td>0.571</td>
<td>0.667</td>
<td>0.6</td>
<td>0.643</td>
<td>0.255</td>
<td>0.75</td>
<td>0.5</td>
<td>0.14</td>
<td>0.21</td>
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<tr>
<td></td>
<td>Change in % IBW &gt;=3.66</td>
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<td>0.643</td>
<td>0.571</td>
<td>0.667</td>
<td>0.6</td>
<td>0.643</td>
<td>0.255</td>
<td>0.75</td>
<td>0.5</td>
<td>0.14</td>
<td>0.21</td>
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<tr>
<td></td>
<td>Change in compensatory behaviors &gt;=-1</td>
<td>28</td>
<td>0.643</td>
<td>0.643</td>
<td>0.722</td>
<td>0.5</td>
<td>0.643</td>
<td>0.222</td>
<td>0.72</td>
<td>0.5</td>
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<td><strong>Adolescent BN</strong></td>
<td>Compensatory behaviors EOT &lt;1</td>
<td>49</td>
<td>0.286</td>
<td>0.429</td>
<td>0.857</td>
<td>0.743</td>
<td>0.776</td>
<td>0.522</td>
<td>0.57</td>
<td>0.93</td>
<td>0.49</td>
<td>0.06</td>
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<td>Change in EDE Restraint &lt; -3.4</td>
<td>49</td>
<td>0.286</td>
<td>0.327</td>
<td>0.714</td>
<td>0.829</td>
<td>0.796</td>
<td>0.521</td>
<td>0.63</td>
<td>0.88</td>
<td>0.48</td>
<td>0.09</td>
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<tr>
<td></td>
<td>% IBW EOT &gt;=107.28</td>
<td>47</td>
<td>0.298</td>
<td>0.574</td>
<td>0.929</td>
<td>0.576</td>
<td>0.681</td>
<td>0.398</td>
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<td>0.95</td>
<td>0.40</td>
<td>0.04</td>
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<td><strong>Adult BN</strong></td>
<td>Compensatory behaviors EOT &lt;2</td>
<td>98</td>
<td>0.143</td>
<td>0.286</td>
<td>0.786</td>
<td>0.798</td>
<td>0.796</td>
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<td>0.96</td>
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<td>Change in global EDE &gt;-2.02</td>
<td>101</td>
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<td>0.317</td>
<td>0.688</td>
<td>0.753</td>
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<td>BMI EOT &gt;=21.68</td>
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<td>0.158</td>
<td>0.525</td>
<td>1</td>
<td>0.565</td>
<td>0.634</td>
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<td>0.3</td>
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<tr>
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<td>101</td>
<td>0.158</td>
<td>0.525</td>
<td>1</td>
<td>0.565</td>
<td>0.634</td>
<td>0.291</td>
<td>0.3</td>
<td>1</td>
<td>0.37</td>
<td>0.00</td>
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<tr>
<td><strong>Adult BED</strong></td>
<td>Global EDE EOT &lt;1.58</td>
<td>97</td>
<td>0.464</td>
<td>0.443</td>
<td>0.756</td>
<td>0.827</td>
<td>0.794</td>
<td>0.584</td>
<td>0.79</td>
<td>0.8</td>
<td>0.24</td>
<td>0.26</td>
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<tr>
<td></td>
<td>BMI EOT &lt;38.15</td>
<td>94</td>
<td>0.479</td>
<td>0.638</td>
<td>0.733</td>
<td>0.449</td>
<td>0.585</td>
<td>0.18</td>
<td>0.55</td>
<td>0.65</td>
<td>0.33</td>
<td>0.17</td>
</tr>
<tr>
<td></td>
<td>% IBW EOT &lt;175.61</td>
<td>94</td>
<td>0.479</td>
<td>0.638</td>
<td>0.733</td>
<td>0.449</td>
<td>0.585</td>
<td>0.18</td>
<td>0.55</td>
<td>0.65</td>
<td>0.33</td>
<td>0.17</td>
</tr>
<tr>
<td></td>
<td>EDE objective binges &lt;1</td>
<td>97</td>
<td>0.464</td>
<td>0.732</td>
<td>0.8</td>
<td>0.327</td>
<td>0.546</td>
<td>0.122</td>
<td>0.51</td>
<td>0.65</td>
<td>0.39</td>
<td>0.12</td>
</tr>
</tbody>
</table>

n, sample size; P, prevalence (percent of sample with a positive dx); Q, level of test (percent of sample with a positive test); PVP, predictive value of positive test (probability of having a positive diagnosis among those patients having a positive test); PVN, predictive value of negative test (probability of having a negative diagnosis among those patients having a negative test); FP, false positive rate (percent having a negative diagnosis and positive test); FN, false negative rate (percent having a positive diagnosis and negative test).

**FIGURE 1.** Predictors of transdiagnostic recovery. [Color figure can be viewed in the online issue, which is available at wileyonlinelibrary.com.]
**Binge Eating Disorder**

For BED, which was examined only in adults, attainment of an EDE global score of < 1.58 was the most efficient predictor (Kappa ES = .58). Binge eating levels per se were not a clinically significant predictor of recovery.

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**Discussion**

As noted in the introduction, there is little agreement about the definition of recovery from eating disorders. However, a number of recent studies have used a defined threshold for recovery.\(^4,15,24,36,42\) For the most part, these studies use definitions that are broadly transdiagnostic, often utilizing community norms on weight, eating related cognitions, and behaviors as the bases for defining recovery. Nonetheless, the relationship between the EOT response and recovery remains understudied;\(^4,5\) however, both clinicians and patients share an interest in having treatment outcome predict future recovery. Hence, it is important to understand the probability that a patient’s clinical status at the EOT leads to longer-term recovery. Because many eating disorders are subject to relapse after treatment,\(^16,18,43,44\) this information is needed to determine which patients likely require additional treatment.

Significant weight gain to a particular criterion by the EOT appears to be the best predictor of recovery for adolescents with AN. This finding is in accordance with previous studies.\(^4,8\) Unfortunately, because so few adults with AN in the study used for this report met even a lower threshold for recovery, it was not possible to ascertain any EOT predictors associated with a broad transdiagnostic conceptualization of recovery in this group. This finding is consistent with the observation that for persistently ill patients with AN a definition of recovery may not be helpful; instead, considerations of outcomes related to harm minimization, improvements in quality of life, and reduction of high risk behaviors might be more useful.\(^34,45\)

Despite the common use of change in binge eating and purging rates as an outcome measure for studies of BN and BED,\(^15,46\) based on our study, binge eating rates at the EOT do not appear to be strongly related to recovery at follow-up for these disorders in adults. Instead, compensatory behaviors, (more broadly defined to include: purging, excessive exercise, diuretic use, and fasting) reduced to fewer than two per month at EOT, predicted transdiagnostic recovery in adult BN. Although abstinence from binge eating and purging was the outcome used in the two RCTs of adolescent BN, change on the EDE was not included in the definition of recovery in those studies.\(^24,25\) Our results suggest that including measures of change on eating related psychopathology as assessed by the EDE is likely important at the EOT in predicting longer-term recovery in this age group.

For BED, the best predictors of recovery were global changes in eating related psychopathology. The fact that binge eating rates themselves were not highly predictive of longer term recovery from BED is somewhat surprising as many would anticipate that behavior change in this domain is evidence of effective treatment over the longer term.\(^47–49\) On the other hand, according to our analyses the importance of changes in eating related psychopathology at EOT suggests that cognitive change in this domain may be an important driver for maintenance of behavioral change over time. It is noteworthy that the EDE global EDE score that predicts recovery at follow-up in BED is exactly 1 SD above the community mean supporting the usefulness of the EDE as a measure of eating related psychopathology in BED. These results also suggest it is likely worthwhile to consider changes in eating related cognitions in considering recovery in BED.

Our results provide little support for the use of a uniform transdiagnostic definition of recovery for eating disorders, encompassing three criteria: weight, psychological, and compensatory behaviors. Only one entity, adolescent bulimia nervosa, had two predictive criteria: psychological and compensatory behaviors. This suggests that the best outcomes predicting future success are likely to be different in different disorders. At the same time, the results of this study provide support for the use of the EDE as a useful EOT measure for most types of EDs in predicting longer term outcome, though specific thresholds vary among the diagnostic groups.

There are significant limitations to this study. It is an exploratory study utilizing existing databases of participants and conducting a secondary analysis. Further, sample sizes are generally small and limit both our power to detect effects as well as limiting the generalizability of our findings. As noted, there are no agreed upon definitions for recovery for eating disorders and the transdiagnostic approach utilized here is only one approach, and other definitions of recovery could have been utilized. In addition, we modified this transdiagnostic approach for adults with AN, because so few participants in the studies recovered using it. We utilized data only in participants where we had sufficient follow-up data so not all participant data
ing analyses using shared definitions of recovery and to promote report-
ance from scientific and clinical organizations to definitions of recovery will require collaboration
the present study. Such research to test threshold
rather than using an arbitrary length of time as in
interval to establish recovery can be determined,
studies with an array of measures so that the time
come are necessary. We need to speak a common
language and share common goals to make signifi-
cant progress in understanding and treating eating
disorders. However, current data suggest in the field
of eating disorders we still need to consider the
stage of illness and specific diagnoses of patients
when considering the definition of recovery.

From a clinical perspective, understanding that EOT outcomes may vary by diagnosis and age
group is important because when we conclude
treatment, it is important to inform our patients of
the real likelihood that the effects of our treatment
will be enduring and lead to recovery. Future
research would benefit from longer-term treatment
studies with an array of measures so that the time
interval to establish recovery can be determined,
rather than using an arbitrary length of time as in
the present study. Such research to test threshold
definitions of recovery will require collaboration
among researchers as well as leadership and guid-
ance from scientific and clinical organizations to
develop and recommend working definitions of
recovery in eating disorders and to promote report-
ing analyses using shared definitions of recovery outcomes in RCTs.

References

3160.

2. Frank E, Prien RF, Jarrett RB, Keller MB, Kuper DJ, Lavori PW, et al. Conceptu-
alization and rationale for consensus definitions of terms in major depressive disorder: Remission, recovery, relapse, and recurrence. Arch Gen


5. Couturier J, Lock J. What constitutes remission in adolescent anorexia nervo-
sa: A review of various conceptualizations and a quantitative analysis. Int J Eat

6. Kuper DJ. Achieving adequate outcomes in geriatric depression: Standard-

7. Kraemer HC, Frank E, Kuper DJ. How to assess the clinical impact of treat-
ments on patients, rather than the statistical impact of treatments on meas-

son, recovery, relapse, and recurrence in eating disorders: Conceptualiza-
tion and illustration of a validation strategy. J Clin Consult Psychol 2002;58:
833–856.

9. Pike K. Long-term course of anorexia nervosa: Response, relapse, remission,

10. Bjork T, Clinton D, Norring C. The impact of using different outcome measures
on remission rates in a three year follow-up of eating disorders. Eur Eat


12. Herzog DB, Dorer DJ, Keel PK, Selwyn ME, Ekeladl ER, Flores AT, et al. Recov-
ery and relapse in anorexia and bulimia nervosa: A 7.5-year follow-up

13. Strober M, Freeman A, Morrell W. The long-term course of severe anorexia nervo-
sa in adolescents: Survival analysis or recovery, relaps, and outcome predictors


comparison of cognitive-behavioral therapy and interpersonal psychother-

16. Mitchell J, Davis L, Goff G. The process of relapse in bulimia nervosa. IJED

17. Fairburn CG, Peveler RC, Jones R, Hope RA, Doll HA. Predictors of 12-month
outcome in bulimia nervosa and the influence of attitudes to shape and

dictors of patients with bulimia nervosa who achieved abstinence through

care and cognitive-behavioural therapy for bulimia nervosa: Randomised

20. Pyle RL, Mitchell JE, Eckert ED. Maintenance treatment and 6-month out-
come for bulimic patients who respond to initial treatment. Am J Psychiatry

21. Olmsted M, Kaplan A, Rockert W. Rate and prediction of relapse in bulimia

22. Cogley CK, PK. Requiring remission of undue influence of weight and
shape on self-evaluation in the definition of recovery from bulimia nervosa.

23. Olmsted M, Kaplan A, Rockert W. Defining remission and relapse in bulimia

comparison of family-based treatment and supportive psychotherapy for

controlled trial of family therapy and cognitive behavior therapy guided self-care for adolescents with bulimia nervosa and related conditions. Am J

26. Hay P, Bacaletich J, Stefano S, Kashyap P. Psychological treatments for buli-
mia nervosa and bingeing. Cochrane Database Syst Rev. In: The Cochrane

ized clinical comparison of group cognitive behavioral therapy and group


49. Safer DL, Robinson AH, Jo B. Outcome from a randomized controlled trial of group therapy for binge eating disorder: Comparing dialectical behavior therapy adapted for binge eating to an active comparison group therapy. Behav Ther 2010;41:106–120.