The impact of emotion (dys)regulation on eating disorder outcomes: A longitudinal examination in a residential eating disorder treatment facility

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Abstract

Eating disorders (ED's) are a complex set of disorders associated with a high rate of comorbidities and innumerable deleterious outcomes (e.g., medical complications, high rates of death by suicide). The complexities of ED's are further compounded by treatment dropout, poor treatment outcomes, and relapse. One way to better understand said complexities is to investigate broad, transdiagnostic risk factors that contribute to the etiology and maintenance of EDs. An established, transdiagnostic risk factor in the ED literature is emotion regulation or lack thereof. However, only a handful of studies have longitudinally investigated the role of emotion (dys)regulation and ED treatment outcomes, and even fewer have examined these factors in a residential treatment center. As such, we sought to longitudinally examine the relationship between emotion (dys)regulation and ED outcomes in a sample of 101 female ED patients in a southeastern U.S. residential ED treatment facility. Consistent with our hypothesis, there were statistically significant improvements in both emotion dysregulation and eating pathology from admission to discharge. Further, improvements in one's ability to engage in emotion regulation strategies was a statistically significant predictor of improvement in ED cognitive symptoms. Also consistent with hypothesis, improvement in emotion dysregulation was a statistically significant predictor of improvement in eating disorder risk. These findings are consistent with previous literature that substantiates the role of emotion dysregulation in eating disorders and provides further evidence for the impact of emotion dysregulation on eating pathology in residential eating disorder patients. Though additional work is needed to improve and validate treatment approaches in residential treatment centers, the current study provides promising evidence that the treatment in these centers is associated with improvement in emotion dysregulation and ED pathology/risk.

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The impact of emotion (dys)regulation on eating disorder outcomes: A longitudinal examination in a residential eating disorder treatment facility

Eating disorders (EDs) are characterized by disturbances in eating and food-related behaviors as well as disturbances in perceptions of weight and shape. These disorders cause significant distress and impairment in several domains of functioning including interpersonal relationships, occupational demands, and physical well-being. In line with the updated diagnostic criteria of the Diagnostic and Statistical Manual of Mental Disorders (5th ed; DSM-5; American Psychiatric Association, 2013), Udo and Grilo (2018) estimated the lifetime prevalence rates of EDs to be 0.80% for anorexia nervosa (AN), 0.28% for bulimia nervosa (BN), and 0.85% for binge eating disorder (BED) though these estimate can vary depending on numerous factors (e.g., study location, sample demographics, diagnostic approaches; Galmiche el al., 2019; Santomauro et al., 2021; Wu et al., 2020). Risk factors for EDs include sex, race and ethnicity, childhood eating and gastrointestinal issues, increased shape and weight concerns, negative selfevaluation, and sexual abuse (Jacobi et al., 2004). EDs are highly comorbid with other mental health disorders such as substance use disorders (Bahji et al., 2019), anxiety disorders (Swinbourne et al., 2012) and mood disorders (Godart et al., 2007) and can lead to poor physical health outcomes including cardiac and gastrointestinal problems, loss of muscle and bone density, and endocrine abnormalities (Cost et al., 2020; Rome & Ammerman, 2003). Additionally, those with EDs are at an increased risk for suicide (Smith et al., 2018; Steinhausen, 2009), treatment dropout (Dejong et al., 2012), poor treatment outcomes, and relapse (Grilo et al., 2012). In fact, a review of the past 50 years of research confirms that individuals with EDs have significantly elevated mortality rates (Arcelus et al., 2011; Ward et al., 2019). Therefore, it is crucial to understand factors that may contribute to treatment outcomes in this population.

Although there have been many advances in evidence-based psychosocial interventions (EBPIs) for EDs, issues with treatment efficacy and access remain. Notably, only one in ten individuals with EDs receive clinical treatment due to a variety of reasons including lack of identification of the disorder, financial barriers, and reluctance to seek treatment (Akey, 2013; Becker et al., 2005; King, 1989; Mond et al., 2007; National Eating Disorder Association Statistics, 2015; Peterson et al., 2008). Additionally, when individuals with EDs do receive care, more times than not, it is not an EBPI (Cooper & Bailey-Straebler, 2015; Fairburn & Wilson, 2013; Lilienfeld et al., 2013). Despite these barriers, EBPIs do exist for treating EDs and are utilized in a variety of treatment settings including residential treatment centers. Among a nationally representative sample of individuals with a lifetime diagnosis of AN (n=275), BN (n=91), and BED (n=256), it was found that outpatient counseling services were most utilized (AN: 28.1%; BN: 56.2%; BED: 36.0%), followed by self-help/support groups (AN: 10%; BN: 33.9%; BED: 29.1%), medication for AN (7.8%), and 12-steps groups (25.3%) for BN and BED (18%; Coffino et al., 2019). Recommended treatment approaches vary depending on type of ED and age group, but EBPIs for EDs typically include family-based therapy for adolescents and cognitive behavioral therapy approaches for adults (Hay et al., 2014; National Institute for Health and Care Excellence, 2017). Although ED treatment goals vary, they typically include weight restoration, reversal of starvation in AN, normalizing eating and excessive exercise behaviors, and treating comorbid psychological disorders (Guarda & Attia, 2018). Historically, specialty programs housed in academic centers emphasize weight restoration as well as cognitive behavioral and exposure-based behavioral approaches (Guardia & Attia, 2018). However, despite the widespread dissemination of cognitive behavior therapy (CBT) for eating disorders (Waller, 2016), many residential treatment centers (i.e., non-hospital treatment settings) have

maintained an eclectic therapeutic approach implementing a wide range of interventions. These centers often provide full-time housing and multi-disciplinary treatments that include meal support, a variety of recreational activities, and group and individual therapy (Friedman et al., 2016). Though this approach may be appealing to individuals who are apprehensive about gaining weight and adjusting their eating patterns, the efficacy of this approach is unknown (Attia et al., 2016)

EDs are a complex set of disorders with innumerable nuances. To obtain a better understanding of treatment outcomes, an examination of transdiagnostic risk factors that may lead to changes in ED outcomes is necessary. Specifically, one transdiagnostic factor worthy of examination in residential ED treatment settings is emotion regulation.

Emotion (dys)regulation

The idea that we can change and interact with our emotions instead of being a passive bystander to them dates back thousands of years. As such, many conceptualizations of emotion regulation (ER) have emerged in the literature. Gross's (1998) seminal paper integrated previous work on emotion regulation to form a conceptual framework of this broad construct. This review culminated in one of the most influential models of emotion regulation, the Process Model, which describes five domains of emotion regulation: choosing what situations one will be exposed to, changing one or more aspects of a situations, choosing what part of the situation to pay attention to, changing one's appraisal of a situation, and modifying one's emotion-related responses.

More clinically oriented perspectives of ER conceptualize the construct as the ability to *control* emotional expressions and experiences, especially negative emotions, and the reduction of the arousal caused by said emotions (Cortez & Bugental, 1994; Garner & Spears, 2000; Kopp,

1989; Zeman & Garber, 1996). Other researchers emphasize the *functionality* of emotions, proposing that ER does not necessarily involve attempts to immediately reduce negative affect (Cole et al., 1994; Thompson, 1994). The latter conceptualization suggests that the inability to experience and differentiate across a breadth of emotions may be just as maladaptive as the inability to mitigate and regulate strong negative emotions. Likewise, researchers have suggested that adaptive ER not only involves the modulation of emotions, but the ability to monitor and evaluate emotional experiences (Thompson & Calkins, 1996). This perspective highlights the significance of emotional awareness and understanding in the construct of ER.

Modern clinical interests have focused on the concept of emotion dysregulation. Emotion dysregulation is an extension of the construct of emotion regulation and is thought to be a central feature in many forms of psychopathology, including EDs (Mallorqui-Bague et al., 2017; Thompson, 2018). To address the previous lack of cohesiveness in the definition of ER as well as the lack of comprehensive emotion dysregulation assessment tools, Gratz and Roemer (2004), developed a scale to capture and measure these complexities in a clinically contextualized model (Hallion et al., 2018). They defined emotion dysregulation as a multidimensional construct that involves: a) lack of awareness, understanding, and acceptance of emotions; b) lack of access to adaptive strategies to modulate the intensity and/or duration of emotional responses; c) unwillingness to experience emotional distress in the pursuit of desired goals; and d) an inability to engage in goal-directed behaviors while experiencing distress. Due to the high internal consistency, good test-retest reliability, adequate construct and predictive validity, and wide dissemination of the Difficulties in Emotion Regulation Scale, (DERS; Gratz and Roemer, 2004), the current study used this conceptualization to examine whether changes in emotion

dysregulation are associated with changes in ED outcomes over the course of residential ED treatment.

Emotion Dysregulation and Eating Disorders

Emotion dysregulation is implicated in several forms of psychopathology including EDs, and the relationship between emotion (dys)regulation and eating pathology is well documented. In fact, emotion dysregulation is considered a central target in several ED treatment approaches (Safer et al., 2009; Wildes & Marcus, 2011; Wonderlich et al., 2015). A large body of research has substantiated the relationship between emotion regulation difficulties and EDs across the diagnostic spectrum (Brockmeyer et al., 2014; Prefit et al., 2019), lifespan (Prefit et al., 2019; Trompeter et al., 2021a; Trompeter et al., 2021b), and treatment settings (Brown et al., 2020; Meneguzzo et al., 2021). Meta-analytic results indicate a positive relationship between ED pathology and the lack of ability to be aware of, recognize, and regulate emotions (Prefit et al., 2019). Studies investigating the relationship between emotion (dys)regulation and ED pathology have taken both cross-sectional (Monell et al., 2022; Ruscitti et al., 2016; Segal & Golan, 2016) and longitudinal (BED: Bodell et al, 2019; Mallorqui-Bague et al., 2018; AN: Racine & Wildes, 2015) approaches. Empirical evidence suggests associations between emotional dysregulation and ED symptoms in AN symptom maintenance (Racine & Wildes, 2015) and outcomes in BN (MacDonald et al. 2017; Peterson et al., 2017). Further, specific emotion regulation difficulty trends among ED diagnostic classifications have been identified including avoidance of emotion and difficulties in emotional expression in AN (Haynos & Fruzzeti, 2011; Oldershaw et al., 2015) and negative urgency and affect instability in BN (Berner et al., 2017; Pearson et al., 2015). However, findings on specific aspects of emotion dysregulation and their associations with specific ED symptoms are mixed (Monell et al., 2018). Across the literature, those with the binge-purge subtype of AN (AN-BP), exhibit greater difficulties in impulse control than those with the restrictive subtype of AN (AN-R; Brockmeyer et al., 2014; Haynos et al., 2014; Mallorqui-Bague et al., 2018; Rowsell et al., 2016). ED Cognitive symptoms (i.e., restraint, body, shape, weight concerns) have been shown to be uniquely associated with limited emotional awareness in people with AN (Racine & Wildes, 2013). Additionally, an examination of a heterogeneous group of individuals with EDs (Pisetsky et al., 2017) found an association between cognitive ED symptoms and lack of access to emotion regulatory strategies across ED diagnoses, while Lavender et al. (2014) found no such associations in individuals with BN.

Though these study findings may suggest that the different aspects of difficulties in emotion regulation may be diagnosis-specific, they must be interpreted in light of several limitations. For example, the study conducted by Mallorqui-Bague et al. (2018) was the only one to examine the breadth of ED diagnoses. Further, methodologies and outcome variables used, size and composition of samples, and effect sizes varied widely among these studies. In consideration of these limitations, many researchers have determined that a transdiagnostic approach to emotion dysregulation provides the most utility in the examination of EDs (Brockmeyer et al., 2014; Monell et al., 2018; Prefit et al., 2019). In line with this, Brockmeyer et al., (2014) and Mallorqui-Bague et al. (2017) found that when compared to healthy controls, individuals in inpatient ED treatment report more difficulties in emotion regulation. Further, several researchers have found evidence for broader patterns in emotion dysregulation in eating disorders as evidenced by similar total DERS scores across ED diagnoses (Ruscutti et al., 2016) and the emergence of significant differences in emotion dysregulation between ED patients versus university student controls, but not among diagnostic classifications within patients diagnosed with EDs (Monell et al., 2018). Specifically, Monell et al., 2018 found that across ED

diagnoses, ED patients demonstrated poorer emotional awareness, greater non-acceptance of emotions, and limited access to emotion regulation difficulties compared to university students. Taken together, these findings suggest that emotion dysregulation is a transdiagnostic factor in EDs.

Within the broader umbrella of emotion dysregulation, particular difficulties may be associated with specific ED behaviors and symptoms, regardless of an individual's ED diagnostic status. Indeed, Monell et al. (2018) found that limited access to emotion regulation strategies was uniquely associated with cognitive ED symptoms (e.g., restraint, eating, shape, and weight concerns) across all ED diagnoses. Further, MacDonald and colleagues (2017) found that rapid improvements in access to emotion regulation strategies significantly and uniquely predicted remission in ED cognitive symptoms among patients presenting with binge/purge behaviors. Additionally, among a community sample of adolescents, binge eating, driven exercise, and fasting, but not purging, were uniquely associated with emotion dysregulation (Trompeter et al., 2021). Evidence from these studies further supports the idea of taking a transdiagnostic approach and examining emotion dysregulation and its associations with ED symptoms and behaviors, rather than specific ED diagnoses.

Changes in Emotion Dysregulation and ED outcomes

Few studies have longitudinally examined the relationship between emotion dysregulation and ED outcomes across disorders, and those that exist have mixed results. Preyde and colleagues (2016) found that among residential ED patients, there was a significant improvement in affective problems and eating disorder risk as evidenced by EDI-3 Affective Problems Composite (APC; composed from the Interoceptive Deficits and Emotional Dysregulation subscales) and Eating Disorder Risk Composite (EDRC; composed from the

Drive for Thinness, Body Dissatisfaction, and Bulimia subscales) from admission to discharge. Moreover, while APC at admission did not significantly predict improvement on the EDRC during treatment, improvement in APC significantly predicted eating disorder risk at discharge. Further supporting the longitudinal role of emotion dysregulation in ED outcomes, Monell et al. (2022) found that increases in emotion dysregulation as evidenced by total DERS scores were strongly associated with ED symptomatology and increased risk of still having an ED at 1-year follow up among Swedish specialized ED treatment patients. Among those in a partial hospitalization program (PHP), changes in emotion dysregulation were moderately correlated with changes in ED symptoms over time (Brown et al., 2020). Specifically, patients with AN-BP/AN-R subtypes and BN displayed improvements in emotion dysregulation across the board from admission to discharge, but only those with BN still demonstrated these improvements upon follow-up examination (Brown et al., 2020). These longitudinal findings point towards the integral role of emotion dysregulation in the mitigation and maintenance of ED symptomology.

Although these studies begin to address the gap in the literature concerning changes in emotion dysregulation predicting ED outcomes, there are several notable differences in methodology across the studies that have been conducted to date. First, participants in Preyde et al. (2016) participated in treatment that implicated emotion dysregulation as the central cause in eating disorders. As mentioned above, most ED treatments are eclectic in nature and may not have emotion dysregulation as a central tenant. The focus on emotion dysregulation may have contributed to the significant improvement in emotion regulation difficulties in this study and may not be generalizable to programs with less emphasis on emotion dysregulation as the central cause for EDs. Additionally, Brown and colleagues (2020) assessed patients in PHP, which is typically recommended for patients who require a higher level of care, but do not require

hospitalization due to medical instability or residential treatment for constant observation of behaviors (Brown et al., 2018). Therefore, the more intensive monitoring provided by residential ED treatment centers may produce different ED outcomes. Further, although Brown et al. (2020) examined subscales of DERS, it was in relation to ED diagnoses and not ED symptoms and behaviors. Lastly, Monell et al. (2018) and Preyde et al. (2016) conducted studies in Sweden. Though beauty and weight/shape standard may be similar, the same research questions are worth examining in an American population. Due to the notable differences mentioned above (Brown et al., 2020; Monell et al., 2022; Preyde et al., 2016) and lack of empirical longitudinal studies in this area, an investigation of changes in emotion dysregulation predicting ED outcomes in a residential treatment is warranted.

The Current Study

EDs are consistently acknowledged as one of the most difficult mental health disorders to treat due to the high rate of comorbidities (Bahji et al., 2019; Godart et al., 2007; Swinbourne et al., 2012), poor physical health outcomes (Cost et al., 2020; Rome & Ammerman, 2003), increased risk for death by suicide (Smith et al., 2018; Steinhausen, 2009), high rates of treatment inefficacy (Dejong et al., 2012) as well as treatment dropout and relapse (Grilo et al., 2012). For these reasons, investigating broad, transdiagnostic risk factors such as emotion dysregulation may present as a solution to better understand the complexities of these disorders and their outcomes. Despite the significant amount of existing research corroborating the relationship between emotion dysregulation and eating pathology across the lifespan, ED diagnoses, and treatment settings, there is a gap in the literature in how changes in emotion dysregulation over the course of treatment predict ED outcomes, specifically in residential treatment centers. As such, the current study seeks to examine the relationship between changes

in emotion dysregulation and ED symptoms between admission and discharge in a sample of female ED patients in a residential treatment facility. Specifically, we hypothesized that there would be an overall improvement in emotion dysregulation and eating disorder pathology/risk as evidenced by decreases in DERS total, DERS Strategies subscale, Eating Disorder Inventory-3 Affective Problems Composite scores, Eating Disorder Examination-Questionnaire Global scores, and EDI-3 Eating Disorder Risk Composite scores from admission to discharge (H₁). Further, based on findings from Monell et al., (2018), we hypothesized that increased access to emotion regulation strategies, as measured by the DERS Strategies subscale, would be associated with improvement in ED cognitive symptoms, as measured by the EDE-Q Global Scale, from admission to discharge (H_{2a}). Additionally, similar to Preyde et al. (2016), we hypothesized that improvement in EDI-3 Affective Problems Composite Scores would be associated with improvement in EDI-3 Eating Disorder Risk Composite Scores (H_{2b}).

Methods

Participants

All study procedures were approved by the Institutional Review Board of Auburn University (IRB approval #: 11-114). Participants in the current study were selected from 103 adult women receiving ED treatment at an ED residential treatment facility in the Southeastern United States. Participants who completed at least one of the key study measures at either admission or discharge were included in our analyses, which resulted in a final sample size of N = 101. The sample was, on average, 26.92 years of age (SD = 7.86, range = 18=58). Participants were predominately non-Hispanic/Latine (97%) and 93.07% White. Duration of stay ranged from 18 to 147 days (M = 68.85; SD = 31.23). Sample characteristics can be found in Table 1. Although data from this sample have been used in previous studies, (Bodell et al., 2021; Dodd et

al., 2018; Forrest et al., 2016; Kinnear et al., 2021; Smith et al., 2016; Smith et al., 2017; Velkoff & Smith, 2018; Witte et al., 2016; Zuromski et al., 2015), our aims and analyses are unique.

Diagnoses

As described in Witte et al., (2016), as part of the admission process to the residential treatment facility, ED diagnoses were determined using a semi-structured diagnostic assessment based on Diagnostic and Statistical Manual (4th ed., text revision; DSM-IV; American Psychiatric Association, 2000) criteria. Because these diagnoses were determined prior to the fifth iteration of the DSM (American Psychiatric Association, 2013), they had to be reassessed using participants' interview and self-report information bearing in mind the updated DSM-5 criteria. Diagnoses were made by doctoral-level researchers using the clinical interview information collected at admission and the Eating Disorder Examination—Questionnaire (EDE-Q; Fairburn and Beglin, 1994). Participants with interview/self-report data consistent with cognitive and behavioral symptoms of AN outlined by the DSM-5 and a BMI below 18.5 kg/m2 were given a diagnosis of AN (29.7%; n = 30). Participants with a BMI of 18.5 kg/m2 or above were evaluated for cognitive and behavioral symptoms of BN (29.7%; n = 30). Due to insufficient information to diagnose BED per DSM-5 criteria, those who did not meet criteria for AN or BN were assigned an eating disorder- not otherwise specified (EDNOS; 37.6%; n = 38) per DSM-IV criteria. Three participants (3.0%) did not have diagnostic data.

Procedure

As described in Witte et al., (2016), upon their arrival at the residential facility, an intake assessment was conducted with a trained staff member that included a detailed description of the research study. Those who chose to participate signed an informed consent document. Precise data on rate of participation is not available; however, it is estimated that upwards of 85% of

patients agreed to participate. Participation included completing a battery of computer-based measures within the first four days of admission and at discharge. Further permission was granted to researchers to review de-identified clinical files. No compensation was given for participation.

Residential Facility Treatment Protocol

To summarize, the treatment provided at the residential treatment facility would best be described as eclectic. Patients participated in weekly individual and group therapies and activities including CBT and Dialectical Behavior Therapy (DBT; Linehan 1993) groups, expressive arts, mindfulness, identity, strengths and values group, women's issues, relationships, relapse prevention, emotion regulation, goal-planning, etc. A copy of the treatment facility's weekly schedule at the time of data collection is provided in Figure 1.

Measures

All measures were administered at admission and discharge.

Difficulties in Emotion Regulation Scale (DERS; Gratz & Roemer, 2004)

The DERS comprises 36 items, and each item is scored on a scale of 1-5. The DERS provides a total score reflecting difficulties in general emotion dysregulation and six subscale scores with higher scores indicating greater difficulties. Reverse coding was necessary for some items. The six subscales are: Non-acceptance (non-accepting stance towards emotional distress), Goals (difficulties in maintaining goal-directed behavior when upset), Impulse (difficulties remaining in control of one's behavior when upset), Awareness (inattention to emotional responses and lack of emotional awareness), Strategies (limited access to effective and functional emotion regulation strategies when upset), and Clarity (lack of emotional understanding and clarity). The present study utilized the total and Strategies subscale scores. The DERS total score

and Strategies subscale scores have previously demonstrated good to excellent internal consistency and test-retest reliability (Gratz & Roemer, 2004). The DERS total (admission– α = .95; discharge– α = .96) and DERS strategies subscale (admission– α = .88; discharge– α = .92) demonstrated good to excellent internal consistency in the present sample.

Eating Disorder Examination Questionnaire – version 6 (EDE-Q; Fairburn and Belgin, 1994, 2008)

The EDE-Q is composed of 28 items assessing the frequency of eating disorder symptoms over the past 28 days on a scale of $0 = no \ days$; $1 = 1-5 \ days$; $2 = 6-12 \ days$; $3 = 13-15 \ days$; $4 = 16-22 \ days$; $5 = 23-27 \ days$; $6 = every \ day$ with higher scores indicating more symptom severity. EDE-Q items can be used to compute four subscales (i.e., Restraint, Weight Concern, Shape Concern, and Eating Concern). This measure has been found to have strong psychometric properties for both global and subscale scores in terms of internal consistency and test-retest reliability in both clinical and general populations (Gideon et al., 2016). The EDE-Q also demonstrates strong convergent validity with the Eating Disorder Examination (EDE), the gold-standard interview, from which the EDE-Q was derived (Gideon et al., 2016). In line with Monell et al., (2018), the four subscales of the EDE-Q (i.e., Restraint, Eating, Shape, Weight concerns; 22 items in total) are combined into the EDE-Q global score to assess cognitive ED symptoms. The EDE-Q global score (i.e., cognitive symptoms) demonstrated excellent internal consistency at admission (22 items; $\alpha = .95$) and discharge (22 items; $\alpha = .95$) in the present sample.

Eating Disorder Inventory-3 (EDI-3; Garner, 2004)

The EDI-3 includes 91 questions each answered on 6-point scale (Garner, 2004) and has 12 scales and 6 composites that are combinations of these scales. The 12 scales are: Drive for

Thinness, Bulimia, Body Dissatisfaction, Personal Alienation, Interpersonal Insecurity,
Interpersonal Alienation, Interoceptive Deficits, Emotion Dysregulation, Perfectionism,
Asceticism, Maturity Fears, and Self-Esteem. The Interoceptive Deficits and Emotional
Dysregulation scales together make the Affective Problems Composite (APC). The Drive for
Thinness, Bulimia, and Body Dissatisfaction scales together make the Eating Disorder Risk
Composite (EDRC). The EDRC is conceptualized as the level of cognitive and behavioral eating
concerns that could constitute risk for an ED.

Reverse coding was necessary for some of the items. The 1-6 rating scale was recalibrated to use 0–4-point scoring system. A score of 4 assigned to the responses farthest in the symptomatic direction (*always* or *never* depending on whether the item is keyed in the positive or negative direction), a score of 3 for the immediately adjacent response, a score of 2 for the next adjacent response, a score of 1 for the next adjacent response, and a score of 0 assigned to the two responses farthest in the "asymptomatic" direction (Garner, 2004). This was done in order to improve the reliability of some scales and to yield a wider range of scores (Garner, 2004). The EDI-3 scores are summed, and higher scores indicate greater dysfunction. The EDI-3 APC and EDRC composite scores were utilized in the present study. The scales comprising EDI-3 total score and APC/EDRC composites have previously demonstrated acceptable to excellent internal consistency and test-retest reliability (Clausen et al., 2010). The EDI-3 APC (admission- α = .89; discharge- α = .91), and EDI-3 EDRC (admission- α = .91; discharge- α = .94), demonstrated good to excellent internal consistency at admission and discharge in the present sample.

Data Analytic Plan

All analyses were conducted using SPSS 27 for Mac. For each variable, the amount of missing data ranged from 2% to 53%. Based on recommendations from Bodner (2008), we

generated 114 datasets with imputed values. SPSS computes pooled results across imputed data sets for paired samples t-test and linear regressions. However, SPSS does not report pooled standard deviations for any of the analyses we conducted; therefore, effect sizes were calculated from the original, raw data rather than from the pooled results (Cohen, 1988; Cohen, 1992). According to Cohen's 1992 guidelines, $d \ge 0.20$, $d \ge 0.50$, and $d \ge 0.80$ are considered small, medium, and large effect sizes, respectively. Further, according to Cohen's 1988 guidelines, $f^2 \ge 0.02$, $f^2 \ge 0.15$, and $f^2 \ge 0.35$ represent small, medium, and large effect sizes, respectively. Descriptive statistics and frequencies were conducted to assess for normality of the data and outliers.

To test our first hypothesis that participants would exhibit improvements in emotion dysregulation and eating pathology over the course of treatment, we conducted a set of paired t-tests to assess changes in overall emotion dysregulation (DERS total), access to emotion regulation strategies (DERS Strategies subscale), affective problems (EDI-3 APC), ED cognitive symptoms (EDE-Q global), and eating disorder risk (EDI-3 EDRC) scores from admission to discharge.

To evaluate whether change in emotion dysregulation predicts change in eating disorder symptoms (H_{2a} & H_{2b}), we first estimated change scores for access to emotion regulation strategies, affective problems, ED cognitive symptoms, and eating disorder risk from admission to discharge by subtracting the discharge scores from the admission scores (Campbell & Stanley, 1963). Though past researchers have criticized the use of change scores (Bohrnstedt, 1969; Campbell & Stanley, 1963; Cronbach & Furby, 1970; Thorndike, 1924) due to change scores' interrelationship with time 1 scores in their calculation and potential unreliability of their measurement, the present study utilized change scores as they allow for a more straightforward

interpretation of the results because the change scores are in their original metric and yield roughly equivalent results to residualized scores (Campbell & Stanley, 1963). However, to account for concerns about interrelationships between change scores and Time 1 scores, we controlled for admission emotion dysregulation and eating pathology scores in our models. Our model approach was confirmed to be appropriate based on a small simulation study run by a statistical consultant (I. Cero, personal communication, February 20, 2023).

To test H_{2a} , we estimated a regression model with change in access to emotion regulation strategies (i.e., $\Delta DERS$ Strategies) as our predictor variable and change in ED cognitive symptoms (i.e., ΔEDE -Q global) as our criterion variable, while controlling for access to emotion regulation strategies and ED cognitive symptoms at admission. To test H_{2b} , we estimated a regression model with change in affective problems (i.e., ΔEDI -3 APC) as our predictor variable and change in eating disorder risk (i.e., ΔEDI -3 EDRC) as our criterion variable, while controlling for affective problems and eating disorder risk at admission. For both regression models, we expected that there would be a positive relationship between change in emotion dysregulation and change in eating pathology such that as emotion dysregulation improves, so would eating pathology.

We conducted a post-hoc power analysis in G*power (Erdfelder et al., 1996). For hypothesis 1, we had 99.9% power to detect a medium effect size (d = .50; Cohen, 1992) for a paired samples t-test with N = 101. For hypothesis 2, we had 95% power to detect a medium effect size using a linear multiple regression with three predictor variables ($f^2 = .15$; Cohen, 1992).

Results

 $\underline{\mathbf{H_1}}$: Improvement in emotion dysregulation and eating pathology from admission to discharge

As hypothesized, a set of paired-sample t-tests (Table 3) showed that there was a statistically significant decrease from admission to discharge in emotion dysregulation (i.e., DERS total, DERS Strategies, EDI-3 APC) and eating pathology (i.e., EDE-Q Global, EDI-3 EDRC), with medium to large effect sizes (Cohen 1992).

H_{2a} & H_{2b}: Improvements in emotion dysregulation predicting improvements in eating disorder pathology/risk

We conducted two separate regression analyses to test whether improvements in emotion dysregulation predicted improvements in eating pathology from admission to discharge (see Tables 4 and 5). Consistent with hypothesis, improvements in one's ability to engage in emotion regulation strategies was a statistically significant predictor of improvement in ED cognitive symptoms; B = 0.28, SE = .01, p = .02 (H_{2a}). Also consistent with hypothesis, improvement in affective problems was a statistically significant predictor of improvement in eating disorder risk; B = 1.11, SE = .17, p < .001 (H_{2b}). Our effect sizes were medium to large (Cohen, 1992).

Discussion

The current study utilized a longitudinal approach to examine whether improvements in emotion dysregulation were associated with improvements in eating pathology/risk among a sample of residential eating disorder patients. Overall, there was statistically significant improvement in both emotion dysregulation and eating pathology/risk over the course of treatment. Additionally, we found that improvements in ability to engage in emotion regulation strategies and affective problems were positively associated with improvement in ED cognitive symptoms and eating disorder risk respectively. These findings confirm previous findings that

emotion dysregulation is an important transdiagnostic risk factor for ED's (Brockmeyer et al., 2014; Mallorqui-Bague et al., 2018; Monell et al., 2018) and underscore the utility of addressing emotion dysregulation in the treatment of eating disorders.

Previous literature has found that improvements in both emotion dysregulation and eating pathology occur over the course of treatment in intensive ED centers (i.e., partially hospitalized program, Brown et al., 2020; residential, Preyde et al., 2016). Consistent with previous findings, we found significant improvements in both emotion dysregulation and eating pathology across three measures of emotion dysregulation (i.e., DERS total, DERS Strategies, EDI-3 APC) and two measures of ED pathology/risk (i.e., EDE-Q, EDI-3 EDRC). Although these findings highlight the positive short-term outcomes associated with residential ED treatment, the long-term effects of the eclectic treatment approach adopted by residential treatment centers is still widely unknown (Peckmezian & Paxton, 2020). Moreover, as in previous research (Brown et al., 2020; Preyde et al., 2016) the current study did not include a control group. Therefore, it is unknown whether the improvements seen are attributable directly to the residential treatment received by our study participants. As a result, future studies should implement the use of a control group.

While both improved, there were relatively larger improvements in ED pathology/risk compared to improvements in emotion dysregulation. This may be reflective of the residential program's more explicit focus on pathology versus emotion dysregulation. Another possible explanation for this may be that the present study utilized self-reported assessment of emotion dysregulation. Self-report measures require insight that may be impaired in individual with ED's (Lavender et al., 2015). It may be the case that lack of insight into emotion regulation difficulties is more prominent in individuals with eating disorders compared to their insight into their ED

pathology/risk. This could potentially explain the smaller, yet still statistically significant, changes in emotion dysregulation indicated by the DERS self-report measures. Although assessing self-reported deficits in emotion dysregulation from individuals with ED represents a possible limitation, few alternatives (i.e., Emotion Regulation Interview; Werner et al., 2011) exist. Although future research should seek to use a multimethod assessment approach, our findings demonstrate that patients enrolled in residential ED treatment exhibit reductions in both emotion dysregulation and ED pathology/risk.

In line with Monell et al., (2018), we conceptualized ED cognitive symptoms as the EDE-Q items that captured the frequency of thought content, attitudes, efforts and negative emotions regarding shape, weight and eating. As hypothesized, and similar to Monell et al.'s (2018) findings, increased access to emotion regulation strategies predicted improvement in ED cognitive symptoms. When an individual gains increased access to emotion regulation strategies, they may be better able to manage distorted or exaggerated cognitions surrounding their eating habits, weight, and shape as well as the associated negative emotionality that is associated with these cognitions. Indeed, MacDonald and colleagues (2017) found that rapid increases in access to emotion regulation strategies during the first four weeks of intensive ED treatment predicted improvements in cognitive ED psychopathology at posttreatment. Additionally, although our approach differed from Brown et al. (2020) in that our outcome variable was ED cognitive symptoms rather than specific ED diagnoses, the overall pattern of results was similar in that some of the individuals in their study (i.e., persons with BN) exhibited significant improvements in their ability to access emotion regulation strategies from admission to discharge. Findings from Brown et al. (2020), Monell et al., (2018), MacDonald et al., (2017), and the present study support the idea that increased confidence in one's ability to effectively cope with one's

emotions is predictive of positive ED treatment outcomes. Additionally, our finding that improvements in an individual's ability to engage in adaptive emotion regulation strategies directly contributes to improvement in ED cognitive symptoms adds to existing literature that demonstrates that a wide range of emotion regulation deficits are related to cognitive ED symptoms (Lavender et al., 2014; Pisetsky et al., 2017; Racine et al., 2013).

Additionally, as hypothesized, and in line with Preyde and colleagues' (2016) findings, we found that improvement in affective problems predicted improvement in eating disorder risk. This finding demonstrates that improvements in one's ability to be aware of, recognize, and modulate their emotions as well as their ability to sense and be aware of their internal states is predictive of improvements in an individual's overall risk of engaging in cognitive and behavioral ED symptoms. Additionally, participants in Preyde et al., (2016) were involved in an ED treatment protocol that implicated emotion dysregulation as the central cause of eating disorders. Despite the current sample participating in a treatment that did not adopt the same theoretical approach as Preyde and colleagues (2016) sample, similar findings emerged. This may suggest that, when emotion dysregulation is addressed in ED treatment in any capacity, improvements in emotion dysregulation are associated with improvements in ED risk; alternatively, it may be the case that engaging in any ED treatment at all, even treatments without a specific emphasis on emotion dysregulation, might result in improved emotion dysregulation. Importantly, Preyde and colleagues (2016) examined their hypotheses in an inpatient setting in Canada, and nevertheless, similar findings emerged in the current sample of participants engaging in residential treatment in the United States. This consistency of results highlights the positive outcomes that can occur despite varying levels of monitoring and medical severity. Lastly, our study contributes to the limited body of research that has longitudinally investigated

emotion dysregulation and ED outcomes in treatment settings (Brown et al., 2020; Monell et al., 2018; Preyde et al., 2016).

To our knowledge, the current study is the first to utilize multiple measures of emotion dysregulation and eating pathology in a single investigation. Our findings were similar across all of the measures (i.e., improvements in emotion dysregulation and ED pathology/risk from admission to discharge; improvements in emotion dysregulation predict improvements in ED pathology/risk). This is notable as the DERS Strategies subscale and EDI-3 affective problem composite measure slightly different constructs. Specifically, the DERS Strategies subscale measures an individual's belief that once negative emotionality has escalated, there is not much they can do to modulate their emotions (Gratz & Roemer, 2004). Alternatively, the EDI-3 affective problems composite broadly captures two different but related constructs (i.e., emotion dysregulation and interoceptive deficits). Interoceptive deficits refer to a deficit in one's ability to identify hunger signals, satiety, and the tendency to confuse one's emotions with these bodily sensations (Garner et al., 1984). Further, although both the EDE-Q and EDI-3 EDRC capture the extent to which an individual is experiencing symptoms associated ED pathology and risk, the EDE-Q global score is comprised of items that mainly capture ED cognitive symptom, while the EDI-3 EDRC captures both cognitive and behavioral traits associated with EDs. Overall, the consistency of findings across operationalizations of emotion dysregulation and ED pathology/risk suggests that there is a broad and robust relationship that is not dependent on the specific measures or definitions used.

Although there are several strengths in the current study, the results should be considered in light of the study's limitations. One limitation was that there was a considerable amount of missing data; however, we used multiple imputation to mitigate its impact on our results.

Additionally, SPSS does not report pooled standard deviations for any of the analyses we conducted; therefore, effect sizes were calculated from the original, raw data rather than the pooled data. Future research should strive to minimize the amount of missing data, if possible. Another limitation was that diagnoses were originally determined using *DSM-IV* criteria and reassessed using *DSM-5* criteria; however, given that the current study utilized a transdiagnostic approach to examine the study questions, we do not believe that this limitation hampers our ability to draw conclusions from our data. Further, it is unknown which specific aspects of the residential treatment approach were particularly impactful in leading to improvements in emotion dysregulation and/or ED symptoms. To determine this, studies that seek to dismantle existing ED treatments to assess the efficacy of specific treatment components is necessary (Bell et al., 2013; Manassee et al., 2019)

Another limitation to consider was that the present study only included data from two time points (i.e., admission and discharge), and several limitations are associated with this methodological approach. First, researchers are unable to examine curvilinear changes or trends with just two time points which could result in misestimation of change (Ployhart & Mackenzie, 2015). Second, this approach carries an increased risk of misestimating effect sizes and reliability (Rogosa, 1995; Willett, 1989). Third, temporal separation of measurement alone is not sufficient to establish causation and may hide or distort the true nature of the relationship between variables (Ployhart & Mackenzie, 2015). The present study, utilizing a two timepoint longitudinal approach, showed that improvements in emotion dysregulation are associated with improvements in ED symptoms. However, it is unknown if improvements in emotion dysregulation *preceded* improvements in ED symptoms or if improvement in ED symptoms preceded improvements in emotion dysregulation. Although additional timepoints would

contribute to a clearer understanding of temporal precedence, it is still possible that alternate factors could account for the improvements in both emotion dysregulation and ED symptoms. As a result, it would be beneficial for future studies to utilize an experimental design that manipulates the presence of treatment that specifically targets emotion regulation to determine whether this leads to improvements in ED symptoms vs a no-treatment or traditional treatment control group. Lastly, the sample was predominantly composed of White, non-Hispanic/Latine women. Though this largely reflects the demographics of clients in ED treatment centers (Brown et al., 2020; Monell et al., 2018; Preyde et al., 2016), these findings may be less generalizable to the much larger and more diverse group of individuals who experience eating disorders.

Despite its limitations, the current study further corroborates the transdiagnostic role of emotion dysregulation in eating disorders and provides further evidence that emotion dysregulation should continue to be a central target in ED treatment approaches (Safer et al., 2009; Wildes & Marcus, 2011; Wonderlich et al., 2015). Specifically, these findings demonstrate that over the course of residential ED treatment, there was improvement in both emotion dysregulation and ED pathology/risk. Further, findings show that increased access to emotion regulation strategies was predictive of improvements in ED cognitive symptoms among individuals in residential ED treatment centers. Additionally, it was found that overall improvements in affective problems was predictive of improvements in ED symptomatology broadly. Clinically, these findings suggest that attending to emotion dysregulation during residential treatment for eating disorders may help to successfully mitigate ED symptomatology. Although the long-term efficacy of the eclectic treatment approaches adopted by residential ED treatment centers remains largely unknown (Peckmezian & Paxton, 2020), previous literature does suggest that residential treatment centers may provide an opportunity for substantive

improvement in eating pathology and associated symptoms (Delinsky et al., 2010), and the present study findings provide concrete evidence of the positive outcomes that can be associated with residential ED treatment.

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Appendix

Table 1Sample Characteristics (N = 101)

	Mean (SD)
Age	26.92 (7.86)
Duration of Stay (in Days)	68.85 (31.23)
Demographic	n (%)
Race	
White	94 (93.07%)
Black or African American	2 (1.98%)
Native Hawaiian or Other Pacific Islander	1 (0.99%)
American Indian/Alaskan Native	1 (0.99%)
None reported	3 (2.97%)
Ethnicity	
Hispanic/Latine	2 (2%)
Non-Hispanic/Latine	96 (95%)
None reported	3 (3%)

Note. Five participants did not have demographics data at admission.

Table 2 *Bivariate Correlations for Variables of Interest (N = 101)*

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
1. DERS Total (A)	-														
2. DERS Total (D)	.41*	-													
3. △DERS Total	63*	.45*	-												
4. DERS Strategies (A)	.89*	.40*	54*	-											
5. DERS Strategies (D)	.33*	.85*	.40*	.43*	-										
6. △DERS Strategies	57*	.38*	.88*	58*	.48*	-									
7. EDE-Q Global (A)	.50*	.29*	24*	.43*	.16	27*	-								
8. EDE-Q Global (D)	.37*	.47*	.04	.29*	.28*	03	.67*	-							
9. △EDE-Q Global	20	.21	37*	19	.15	.32*	48*	.34*	-						
10. EDI-3 APC (A)	.70*	.28*	45*	.57*	.14	43*	.43*	.29*	20	-					
11. EDI-3 APC (D)	.45*	.60*	.06	.37*	.39*	01	.29*	.47*	.19	.66*	-				
12. △EDI-3 APC	34*	.35*	.63*	29*	.28*	.53*	21	.20	.47*	48*	.34*	-			
13. EDI-3 EDRC (A)	.60*	.27*	35*	.57*	.18	39*	.69*	.46*	34*	.59*	.35*	34*	-		
14. EDI-3 EDRC (D)	.36*	.58*	.14	.31*	.40*	.05	.53*	.80*	.27*	.40*	.60*	.19	.57*	-	
15. △EDI-3 EDRC	21*	.37*	.52*	23*	.26*	.46	11	.42*	.65*	16	.31*	.56*	39*	.54*	-

Note. DERS total = Difficulties in Emotion Regulation Scale total score; DERS Strategies = Difficulties in Emotion Regulation Scale Strategies

Subscale score; EDE-Q Global = Eating Disorder Questionnaire Global score; EDI-3 APC = Eating Disorder Inventory-3 APC = Affective Problems

Composite, EDRC = Eating Disorder Risk Composite, (A) = admission score, (D) = discharge score, \triangle = change score; * p < .05.

Table 3 Paired sample t-tests for emotion dysregulation and eating pathology from admission to discharge (N = 101)

Variable	Mean(SEM) admission	Mean(SEM) discharge	t	df	р	d
DERS Total	113.39 (2.67)	98.25 (2.49)	-5.41	100	<.001	-0.57
DERS Strategies	24.73 (0.73)	21.24 (0.74)	-4.25	100	<.001	-0.55
EDE-Q Global	4.00 (0.16)	2.10 (0.14)	-15.13	100	<.001	-1.45
EDI-3 APC	22.58 (1.30)	17.10 (1.20)	-5.30	100	<.001	-0.50
EDI-3 EDRC	59.1 (1.90)	42.29 (2.08)	-9.02	100	<.001	-0.80

Note. SEM = Standard Error Mean. DERS total = Difficulties in Emotion Regulation Scale total score; DERS Strategies = Difficulties in Emotion Regulation Scale Strategies Subscale score; EDE-Q Global = Eating Disorder Examination Questionnaire Global score; EDI-3 APC = Eating Disorder Inventory-3 Affective Problems Composite score; EDI-3 EDRC = Eating Disorder Inventory-3 Eating Disorder Risk Composite score. Effect size was based on the means and standard deviation computed from the raw (i.e., non-imputed) data.

Table 4Regression results with change in EDE-Q Global symptoms as the criterion variable (N = 101)

	Variable	В	В	SEB p	R^2	$\triangle R^2$	$\triangle f^2$
			95% CI				
			[LL, UL]				
Block 1					.30	.30	.43
	DERS Strategies (A)	0.18	-0.18, 0.22	0.10 0.89			
	EDE-Q Global (A)	-2.20	-3.24, -1.18	0.53 <.001			
Block 2					.47	.17	.20
	DERS Strategies (A)	0.18	-0.55, 0.41	0.12 0.13			
	EDE-Q Global (A)	-2.17	-3.18, -1.15	0.52 < .001			
	ΔDERS Strategies	0.28	0.98, 1.03	.01 .02			

Note. CI = confidence interval; LL = lower limit; UL = upper limit; A = Admission; Δ = Change; DERS total = Difficulties in Emotion Regulation Scale total score; DERS Strategies = Difficulties in Emotion Regulation Scale Strategies Subscale score; EDE-Q Global = Eating Disorder Examination Questionnaire Global score. $\triangle R^2$ and R^2 values were calculated using the raw data (i.e., non-imputed). Effect size calculations utilized $\triangle R^2$ scores.

Table 5Regression results with change in EDI-3 EDRC as the criterion variable (N = 101)

	Variable	В	В	SE B	p	R^2	$\triangle R^2$	$\triangle f^2$
			95% CI					
			[LL, UL]					
Block 1						.21	.21	.27
	EDI-3 APC (A)	0.17	-0.16, .50	0.17	.316			
	EDI-3 EDRC (A)	-0.45	-0.67, -0.23	0.11	<.001			
Block 2						.52	.31	.45
	EDI APC (A)	0.55	0.25, 0.84	0.15	<.001			
	EDI-3 EDRC (A)	-0.40	-0.59, -0.21	.09	<.001			
	ΔEDI-3 APC	1.11	0.78, 1.42	0.17	<.001			

Note. CI = confidence interval; LL = lower limit; UL = upper limit; EDI-3 APC = Eating Disorder Inventory-3 Affective Problems Composite score; EDI-3 EDRC = Eating Disorder Inventory-3 Eating Disorder Risk Composite score; A = Admission; Δ = Change. $\triangle R^2$ and R^2 values were calculated using the raw data (i.e., non-imputed). Effect size calculations utilized $\triangle R^2$ scores.

Figure 1
Residential Treatment Facility's Weekly Schedule

7:00-8:00	A CONTRACTOR OF THE PARTY OF TH	onday	Tuesday	Wednesday	Thu	rsday					
7.00-0.00	Wake-	up, dress,	Wake-up, dress,		The second second		Friday		irday	Sund	lav
8:00-8:30	vital	s, meds	vitals, meds	Wake-up, dress, vitals, meds	Wake-up, dress, vitals, meds		Wake-up, dress, vitals, meds	Wake-up, dress, vitals, meds		Wake-up, dress vitals, meds	
8:30-9:30	Bre	akfast	Breakfast	Breakfast			The state of the s				
0.50-9.50	Morni	ng Walk	Morning Walk				Breakfast		kfast	Break	fast
9:30-10:15	-	PHP(off-		Self-	Mornin	g Walk	Yoga		ng Walk up day)	You	a
	Appts RTC	campus clients) Check-in LE	Spirituality Group* NS	Defense/Nutrition/ Ropes/Nutrition	Health & Wellness*		Gratitude Group*	Nutr Gro	rition up*	Optional Church	Fre
10:30-10:45	Sr	nack	Snack	Const (11)				E	P	Outing	Tim
10:45-12:15	Commun	nity Group		Snack (portable)	Sna	ick	Snack	Sna	ick	Snac	l-
	Process	Group NG	Process Group LH	12-step Group (11:15-12:15) CN	Process G	Process Group SM Process Gr		Process		Process (
12:15-12:30		leds	Meds	Meds		STATE OF STATE OF	Process Group CN	AP		LM & LH	
12:30-1:00	Lu	inch	Lunch	Lunch	Me		Meds	Meds		Meds	
1:30-2:50	СВТ	THE RES	Control of the last of the las	DBT	Lun	ch		Lur	nch	Lunc	
200	Group*	Tx Team (LH/SE)	Psychodrama* KH Tx Team (SM/EP)	Skills Group* (CN/SE)	Boundaries Group* CN	Tx Team (AP/EP)	Lunch Outing (12:30-2:30)	1		Expressive Arts Group	Visit ing Hour
3:00- 3:15		ack	Snack	Snack	Body Integration Group* Emotion Regulation		Therapeutic		Snack Outing		
3:30-4:30		fulness p* SM	Identity, Strengths & Values Group* AP	Expressive Arts			Emotion Regulation	Recreational Outing (with		Diagn Outling	
4:30-5:30		al Appts/ al Time	Individual Appts/ Personal Time	Group	Individual Personal		Individual Appts/Personal Time	portable snack)		Errand Outing	
5:30-5:45	Me	eds	Meds	Meds	Meds		Meds	Meds		Meds	
5:45-6:15	Din	nner	Dinner	Dinner	Dinner		Dinner	Dinner		Dinner (6-6:30)	
6:15 -6:45	Affirm: Appre	ation & ciation	Affirmation & Appreciation	Process Group (6:15-7:30) LM	Affirmat Appreci		Affirmation & Appreciation	Affirmation & Appreciation		Affirmation & Appreciation (6:30-7:00)	
7:00-8:30	Gro Ll	Н	Relationships Group AS	PHP RTC Home- Class (7:45-8:30) Time		Relapse Prevention Group LM Community C Assignments C LM		(7-9pm)		Goal Planning Group AG	
8:30-10:30	Free (Snack		Free Time (Snack 9:00)	Free Time (Snack 9:00)	Free Time (Snack 9:00)		Free Time	Movie Night Snack 9:00		Free Time (Snack 9:00)	
10:30**	Lights	s Out	Lights Out	Lights Out	Lights		Lights Out	Lights	Out	Lights O	
resident/eli	ject to change ent needs ours are from 's s may also be	7-9 Saturdays a	ogram and and 1:30-3:00 Sundays	Meals Groups	Outing	gs/Activities	Free Time Appts/Personal To		Physical .	Activity	d 3/1/11