5-2023

Understanding the role of emotion regulation tendencies in the momentary associations between negative affect and eating disorder behaviors.

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Understanding the Role of Emotion Regulation Tendencies in the Momentary Associations Between Negative Affect and Eating Disorder Behaviors

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Submitted in partial fulfillment of requirements for Graduation *summa cum laude*

And

For Graduation with Honors from the Department of Anatomical Sciences and Neurobiology

University of Louisville

May 2023

Acknowledgements

The writing of this thesis has been an intensely rewarding process, and its completion would not have been possible without the support of several people.

First, I would like to thank my mentors, Dr. Cheri Levinson and Dr. Christina Ralph-Nearman, for allowing me to work in their lab and always providing a positive and constructive environment for me to learn. I would also like to thank my lab supervisors, Rowan Hunt and Taylor Penwell, who have worked with me closely throughout this process and have provided invaluable guidance and encouragement. From the bottom of my heart, thank you all for your constant, above-and-beyond support.

Personally, I would also like to thank my roommates and close friends for their patience in my absence from hangouts over the past several months. At least I have contributed a cause for celebration. And last, but certainly not least, I am indebted to my family—my parents, brothers, sisters-in-law, and nephews/niece. While I know you each know your importance in my life, I would be in no position to be writing this kind of statement, in my thesis, at the end of my undergraduate career, were it not for your love and constant relief throughout the years. I love you all, and as genuinely as one can mean this statement: thank you.

Abstract

Background: Eating disorders (EDs) are becoming increasingly commonplace and are associated with a broad array of dangerous medical complications. Further, EDs frequently present alongside comorbid psychiatric disorders (e.g., depressive and anxiety-related disorders), which are known to both predict ED severity and reduce treatment efficacy. While diagnostic categorization persists, Fairburn et al.'s (1993) transdiagnostic model suggests EDs may be more closely related and maintained through core, shared symptoms. To assess ED maintenance, momentary triggers such as negative affect (NA) are becoming increasingly centralized. The present study examines the roles of depressive symptoms and worry in predicting and moderating the relationships among NA (i.e., guilt/anxiety) and ED behaviors (i.e., binge eating, dietary restriction, and driven exercise) to provide a clearer description of ED psychopathology. **Method:** Participants (N = 130) completed diagnostic screenings and baseline questionnaires at onset, and ecological momentary assessment (EMA) surveys sent to their mobile phones four times per day for 25 days. The relationships among NA and ED behaviors were tested using mixed effects models in R. **Results:** State and trait NA were differentially implicated in the associations among ED behaviors and depression/worry. Depressive symptoms and worry were each moderators of the relationship between driven exercise and state NA. **Discussion:** The present study broadens current understanding of NA's contribution to ED psychopathology and emphasizes the significance of psychiatric comorbidities in maintaining ED behaviors. Future research should assess the treatment of depressive cognitions in reducing binge eating behaviors and the efficacy of emotional regulation strategies in reducing dietary restriction and driven exercise.

Lay Summary

Eating disorders (EDs) are becoming increasingly commonplace and are closely associated with a variety of dangerous health consequences, both mental and physical. Having another psychiatric disorder (e.g., comorbid depression or anxiety) alongside an ED diagnosis is known to increase the severity of each disorder's symptoms, as well as reduce the effectiveness of their treatment. In the field of ED research, there is currently a growing interest on assessing moment-to-moment changes in various triggers which may serve to maintain ED behaviors. One such momentary trigger—negative affect (NA), generally characterized by unpleasant emotions—has been especially linked to ED maintenance, and its analysis is useful in more descriptively assessing the interactions among EDs and their comorbidities.

The present study aimed to clarify the contributions of worry and depression to the associations among NA (i.e., guilt/anxiety) and ED behaviors (i.e., binge eating, dietary restriction, and driven exercise). We found that depressive symptoms were generally more involved in the interactions among NA and both dietary restriction and binge eating behaviors. For the relationship between NA and driven exercise, worry and depressive symptoms played similar roles, and further served to predict the strength of the relationship between momentary NA and driven exercise. The authors suggest that, for individuals who experience frequent states of NA, the treatment of depressive cognitions be assessed in reducing binge eating behaviors, and that emotional regulation strategies be assessed for their efficacy in reducing dietary restriction and driven exercise. These findings stress the significance of comorbid psychiatric disorders in maintaining ED behaviors and provide a clearer description of the role NA plays in their interactions.

Understanding the Role of Emotion Regulation Tendencies in the Momentary Associations Between Negative Affect and Eating Disorder Behaviors

Based on estimates from the most recent Global Burden of Disease study in 2019, eating disorders (EDs) affect around 55.5 million people worldwide and further account annually for up to $400 billion in economic cost (Santomauro et al., 2021; Streatfeild et al., 2021). While for many, the daily routine of eating is regular, casual, and instinctive, for those with an ED, the process can often be complicated, troublesome, and altogether discouraging. EDs have become increasingly widespread today, and the prevalence of ED behaviors, with or without diagnosis, is continuing to rise (Demmler et al., 2020). Hospitalizations due to ED diagnoses have risen more than twofold since the beginning of 2018 (Asch et al., 2021). Additionally, EDs such as anorexia nervosa (AN) are associated with the second-highest mortality out of any mental disorder, up to six times that of the general population, and surpassed in lethality only by opioid-use disorder (Murray et al., 2018; Krausz et al., 2021). Despite this increase in prevalence and distinction in severity, the low rates with which ED treatment is sought—between 25% and 38%—are notably disproportionate (Romano et al., 2022). When treatment is sought, efficacy rates are remarkably low, with up to 60% of AN patients never reaching remission (Fichter et al., 2017). Across EDs, the presentation of fears which serve to maintain ED behaviors is highly heterogeneous, unique even within categorical diagnoses (Brown & Levinson, 2022). For such a dangerous set of disorders diverse in presentation, there is a necessity for more specific research which could increase the personalization of treatments and their efficacy.

While every ED is personalized with individual experience, network analysis models suggest that the core symptoms across EDs are relatively conserved. That is, many of the symptoms (e.g., desire to be thin, guilt after overeating, perfectionist tendencies) experienced by

an individual diagnosed with binge eating disorder (BED), for example, may also be experienced by one diagnosed with AN (Schlegl et al., 2021). Further, ample research suggests that the longitudinal stability of ED diagnoses is poor (Stice et al., 2009; Forbush et al., 2017). For example, one study demonstrates that over a five-year timespan, 36% of participants with AN developed bulimia nervosa (BN) and 27% of participants with BN migrated to AN diagnoses (Tozzi et al., 2005). The frequently overlapping psychopathology of EDs is exemplified by Fairburn et al.'s (1993) transdiagnostic model, which stresses the place of core symptoms shared across diagnoses as central to ED maintenance. An alternative approach to ED conceptualization as compared with transdiagnostic theories is based upon diagnostic categorization, and using such criteria to provide a brief characterization of common EDs will allow for a more holistic understanding of ED nosology alongside the associations often observed across diagnoses.

AN—perhaps the most widely-known of EDs, despite evidence suggesting its lifetime incidence may be lower than many of its counterparts (ranging from 0.8% to 1.9% in females)—is characterized in the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) by three criteria: first, significant weight loss resultant from restriction of sufficient nutriment; second, a preoccupied fear around weight gain or the idea of becoming fat; and third, distress around the way in which one perceives their body, even at dangerously low body mass indices (BMIs; Dahlgren et al., 2017; American Psychiatric Association [APA], 2013).

Slightly more frequent with lifetime prevalence rates of 3.0% to 3.6% for females, BED is defined by the repeated presence of episodes wherein an individual eats much more than what would be typically considered normal and experiences a sense of loss of control over this eating; the disorder is further accompanied by feelings of distress around such binge eating episodes (Dahlgren et al., 2017; APA, 2013).

BN, experienced by up to 2.6% of females throughout their lifetime, is similarly characterized by recurrent binge eating, but is succeeded by behaviors (e.g., purging, inappropriate use of laxatives or diuretics, excessive exercise) meant expressly to compensate for this high caloric intake (Dahlgren et al., 2017; APA, 2013).

These three, typically considered as the most prevalent, are accompanied in prominence by perhaps the most commonly diagnosed ED: other specified feeding or eating disorder (OSFED), which has been suggested to occur in up to 11% of females throughout their lifetime, and is used to characterize EDs that do not readily and wholly meet criteria for any standard ED (Dahlgren et al., 2017; APA, 2013). While not alone in ED classification, these four are generally discussed as the most archetypal, and are telling markers of the vast disparities confined within one, single class of psychiatric disorders (Brandt & Crawford, 2019; reference Table 1 for an overview of ED diagnoses).

Although these disorders are categorically diverse, Fairburn et al.'s (1993) transdiagnostic model efficaciously argues that the ED behaviors which maintain EDs may be more often shared across diagnoses. In particular, binge eating, dietary restriction, and excessive exercise are commonly observed across diagnoses and are notably adverse (Bicaker & Racine, 2022). In an Iranian study of 637 college students with or without an ED diagnosis, 24.7% of males and 27.5% of females were shown to regularly engage in binge eating; 14.3% of males and 11.4% of females were shown to regularly restrict food intake; and 3.2% of males and 1.9% of females were shown to regularly over-exercise (Sahlan et al., 2020). Such behaviors, then, present in relative homogeneity across both sexes and are not confined to the Western world—which sees rates of prevalence for college-aged females at around 22.2% for dietary restriction, 11.4% for driven exercise, and 3.2% for binge eating (Fitzsimmons et al., 2016). ED behaviors

such as these have been suggested to predict higher BMI and psychological distress across both sexes (Kärkkäinen et al., 2018). Beyond these symptoms, more explicitly pernicious outcomes are commonplace. Specifically, EDs often present alongside or give later rise to medical complications such as improper hormonal balances, a compromised immune system, and—most notably, accounting for one-third of all deaths in ED patients—cardiovascular issues such as irregular blood pressure, abnormal heart rate, decreased cardiac output, and even reduced ventricular mass in children (Peebles & Sieke, 2019). The risks posed by the behaviors associated with EDs and disordered eating in general are thus an additional factor which must be thoughtfully considered in the clinical setting.

Attempts to effectively treat EDs are further complicated by the disorders' high potential for comorbidity alongside other psychiatric diagnoses, especially depressive and anxiety-related disorders. The DSM-5 (APA, 2013) characterizes depressive disorders by disturbances in mood alongside changes in physical and mental capabilities which result in significant functional impairments. Alternatively, anxiety disorders (e.g., generalized anxiety disorder) are defined by the presence of fear and anxiety, which manifest tangibly in the form of behavioral disturbances, such as restlessness or difficulty concentrating (APA, 2013). Although nearly everyone experiences episodic disturbances in mood and states of anxious distress, the clinical population of individuals diagnosed with an anxiety or depressive disorder suffers significant impairments in their ability to function and in their quality of life and are plagued consistently by the persistence and severity of their symptoms (Jenkins et al., 2021).

Indeed, those with an ED diagnosis alongside a comorbid depression or anxiety diagnosis tend to present with more severe psychopathology than those diagnosed with an ED alone, especially in cases involving major depressive disorder (MDD; Hughes et al., 2013). In

consideration of comorbidities within AN alone, rates of incidence for anxious and depressive disorders range from 65-68%, stressing their prevalence (Herzog et al., 1996). Further research concerning the general role of psychiatric comorbidities within EDs has suggested that individuals diagnosed with an anxiety disorder or MDD engage significantly more frequently in ED behaviors, and have higher rates of ED diagnosis, as compared to those without an MDD or anxiety disorder diagnosis (Garcia et al., 2020). For ED patients with MDD, this complicates outcomes by significantly decreasing the population's likelihood not only to seek out or initiate treatment, but also to engage with treatment in cases where it is sought (Burket & Hodgin, 1993). To further confound efficacies, the specific contributions of comorbidities to ED prognoses remain unclear, especially when applied to any derived benefit shared across treatments (Kim & Annunziato, 2020). There is therefore a clear need for meaningful research elucidating the roles of comorbid psychiatric disorders in maintaining ED symptoms, in hopes to inform improvements in both treatments and outcomes (Williams & Levinson, 2022).

Additionally, a meta-analysis of 99 studies has suggested that rumination may serve as one explanation for the high rates of comorbidity among EDs and depression and anxiety (Rickerby et al., 2022). Rumination is characterized as a maladaptive coping strategy, wherein an individual attempts unsuccessfully to regulate negative emotional states by thinking of them repetitively, instead resulting only in their amplification and sustenance (Aldao & Nolen-Hoeksema, 2010; Watkins, 2008). Consequently, rumination has been suggested as a means by which negative emotions are often intensified and prolonged (Blanke et al., 2022). Further, transdiagnostic models have centralized rumination in the maintenance of a broad array of distinct psychopathologies (Watkins & Roberts, 2020). Rumination in depressive disorders has been shown to predict changes in negative affect (NA) momentarily, as well as likelihood for

continued depression prospectively (Hjartarson et al., 2022). In anxiety disorders, rumination often presents in the form of worry, and has been suggested as a means by which patients attempt to gain a sense of control over feared outcomes (Rector et al., 2008; Nolen-Hoeksema, 2000). Finally, rumination is often framed as an underlying factor in subjective experiences of guilt and anxiety, two negative emotional experiences which ED patients are significantly more prone than the general population to endorse and which are known predictors for ED severity (Mendia et al., 2021; Smith et al., 2018).

Alongside the presence of rumination, worry is often significantly elevated in those with an ED and similarly contributes to an individual's negative emotional experiences (Startup et al., 2013). Whereas rumination is based upon a preoccupation with detrimental cognitions and their consequences with no intent to seek resolution, worry is more closely related to the process of fear, and occurs generally as an attempt to pacify the foreboding of situations with unknown or potentially undesirable outcomes (González et al., 2017; Nolen-Hoeksema & Morrow, 1993; Borkovec et al., 1983). In individuals with AN and BN, worry has been correlated with every ED symptom as measured by DSM-5 (APA, 2013) criteria, emphasizing its centralization in ED maintenance (Sassaroli et al., 2005). While it is thus known that worry is often a principal determinant in the outcomes of various EDs, more research is necessary to discern the precise mechanisms by which worry, and its related cognitive patterns, perpetuate ED behaviors (Sala & Levinson, 2016).

In the field of ED research, there is currently a growing interest on momentary triggers (e.g., emotional states, behaviors) which may serve to maintain the diverse symptomatology of EDs (Mason et al., 2021). NA is one such momentary trigger, which has been broadly defined within literature, although generally characterized by the presence of emotional distress and the

experience of any subjectively unpleasant emotional state, such as sadness, guilt, anxiety, shame, or anger (Watson et al., 1988). With the implication of guilt and anxiety in cognitive patterns such as worry and rumination, coupled with the maintenance of EDs and depressive and anxiety disorders by such cognitive patterns, guilt and anxiety are effective variables for the current study's assessment of NA (Bodell et al., 2019). NA—defined by items (e.g., sad, angry at self, afraid) from the NA subscale on Watson et al.'s (1988) Positive and Negative Affect Schedule—has been specifically shown to be a predictor of various ED behaviors, and ED patients with higher levels of NA generally present with more severe psychopathology (Wonderlich et al., 2015). There are a variety of theories suggested to explain the temporal interactions across ED symptoms and similar definitions of NA, such as the affect regulation model, which posits that individuals with EDs may engage in ED behaviors—e.g., binge eating—in attempt to regulate their emotions—i.e., to reduce NA (Berg et al., 2017). Similarly, the escape theory suggests that individuals binge eat to avoid self-awareness (Heatherton & Baumeister, 1991). Alongside such various explanations, Fairburn et al.'s (1993) transdiagnostic model exemplifies the notion that, although mounting in understanding, the momentary mechanisms at work in these interactions remain unclear. Despite distinctions such as these, emotion regulation has been nevertheless replicated as a central factor implicated in maintaining the psychopathology of EDs (Durkin, 2018). In addition to comorbidities, then, momentary antecedents play an important role in ED maintenance, and further analysis of their contribution to ED behaviors is crucial in more holistically understanding ED pathology (Schaefer et al., 2020).

Present Study

In a sample of individuals diagnosed with depression, we have recently conducted a study analyzing the relationship between NA and binge eating behaviors (Banet et al., 2022). Findings

demonstrated a strong positive correlation between sadness and urges to binge eat, as well as the presence of a moderating effect of emotional avoidance on that relationship. However, as this study was performed with a sample diagnosed with depression, the generalizability of its results to the ED population is thereby limited. The current study, then, aimed to replicate these results, but with a sample of individuals diagnosed with an ED. Additionally, by performing more intensive analyses using a variety of different variables, we aimed to explicate a more comprehensive picture of ED pathology alongside its comorbidities.

Specifically, in addition to assessing for the presence of depression within our ED sample, we included the assessment of worry, a central symptom across anxiety disorders (Aim [A] 1; Kertz et al., 2012). Then, we were able to examine the roles which depression and worry play in moderating the relationship between NA (i.e., guilt/anxiety) and binge eating behaviors (A2a). Additionally, in hopes to attain a broader understanding of the effect of emotion on ED pathology as a whole, we further aimed to examine how worry and depression moderate the relationships among NA and two other common ED behaviors: dietary restriction and excessive exercise (A2b and A2c).

In line with prior research indicating a higher incidence of depression than anxiety in those with EDs (Herzog et al., 1996), we hypothesized that there would be significantly high frequencies of both depression and worry within the sample, but that the frequency of depressive symptoms would be slightly higher than that for worry (Hypothesis [H] 1). Regarding the roles of worry and depressive symptoms in moderating the relationship between NA (i.e., guilt/anxiety) and binge eating, we hypothesized that individuals with higher baseline levels of both depression and worry would engage in more binge eating behaviors during states of NA (H2a). For the roles of worry and depression in the relationship between NA and dietary

restriction, we similarly hypothesized that individuals with higher baseline levels of both depression and worry would engage in more restrictive behaviors during states of NA, and that depression would again serve as a slightly more significant predictor of this relationship—in line with the literature's quantification of comorbidities among those with EDs (H2b). Finally, in reference to worry and depression as moderators of the relationship between NA and excessive exercise, we predicted that those with high worry would engage in more excessive exercise while experiencing NA, but that those with more depression would not engage in significant excessive exercise, regardless of their emotional state (H2c).

1. Method

1.1. Participants

The current dataset was one collected from a previous study conducted by the Eating Anxiety Treatment (EAT) Lab at the University of Louisville. Participants ($N = 130$) were recruited for the remote study from across the United States. Recruitment criteria included past or present diagnosis of an ED, and exclusion criteria included present mania, psychosis, or suicidality. For a summary of participant demographics, see Table 2.

1.2. Procedures

Participants first completed a clinical screening (see Diagnostic Assessment below) to determine eligibility. Upon confirmation of eligibility, they completed a set of baseline questionnaires (see Psychiatric Symptom Assessment below for questionnaires included in present analyses). After completing these baseline measures, participants completed the Daily Habits Questionnaire, an ecological momentary assessment (EMA) sent to participants' mobile phones four times per day for 25 days (100 time points). Each EMA consisted of 63 questions

which assessed emotions, ED urges and behaviors, and repetitive thoughts during and after meals.

1.3. Measures

1.3.1. Diagnostic Assessment

Two clinical screening interviews were used for this study to determine eligibility and ED diagnoses.

1.3.1.1. Structured Clinical Interview for DSM-5 (SCID-5; First et al., 2015). The SCID-5 is a semi-structured interview designed for research studies to determine DSM-5 diagnoses. The SCID-5 contains three ED modules, which the present study used to screen for the presence of AN (n = 35), BN (n = 17), and BED (n = 4). Participants who did not meet criteria for AN, BN, or BED, but still endorsed clinically significant ED symptoms were considered to have OSFED (n = 52). The SCID-5 has demonstrated excellent reliability and high specificity in accurately assessing DSM-5 diagnoses (Osório et al., 2019).

1.3.1.2. Eating Disorder Diagnostic Inventory (EDDI; Stice et al., 2008). The EDDI is a semi-structured interview designed to quantify the frequency and intensity of ED symptoms, alongside weight and menstrual histories over the past year. The EDDI contains yes-or-no questions, open responses, and Likert scales ranging from 0-6, and was used to confirm diagnoses from the SCID-5 and provide a clearer description of participants' ED presentations. The EDDI has demonstrated strong test-retest reliability for ED diagnostic assessment as well as predictive validity for future onset of depression (Stice et al., 2008).

1.3.2. Psychiatric Symptom Assessment

Two validated scales were used to measure psychiatric symptoms.

1.3.2.1. Penn State Worry Questionnaire (PSWQ; Meyer et al., 1990). The PSWQ is a 16-item Likert scale used to assess the presence of worry, a symptom central in a variety of anxiety disorders. The scale ranges from 1 (*Not at all typical)* to 5 (*Very typical*), wherein participants rate the frequency of their experiences with statements such as "I do not tend to worry about things" and "Once I start worrying, I cannot stop." Scores range from 16-39 (low worry), 40-59 (moderate worry), and 60-80 (high worry). The PSWQ has demonstrated favorable test-retest reliability and strong discriminant validity in assessing worry apart from anxiety and depression (Meyer et al., 1990). The internal consistency of the PSWQ for the current study was acceptable (α = .743).

1.3.2.2. Beck Depression Inventory-II (BDI-II; Beck et al., 1996). The BDI-II is a self-report measure consisting of 21 questions used to assess the presence and severity of a variety of symptoms commonly experienced by those with depressive disorders. Participants use a Likert scale ranging from 0 to 3 to indicate the frequency/severity with which they have experienced symptoms such as "sadness," "loss of pleasure," and "changes in sleeping pattern" over the last two weeks. Scores range from 0-13 (minimal depression), 14-19 (mild depression), 20-28 (moderate depression), and 29-63 (severe depression). The BDI-II has shown strong test-retest reliability and good sensitivity and specificity in detecting depression (Wang & Gorenstein, 2013). The internal consistency of the BDI-II for the current study was excellent (α = .915).

1.3.3. Ecological Momentary Assessment

1.3.3.1. Daily Habits Questionnaire (DHQ; Levinson et al., 2018). The DHQ is a 63-item self-report measure designed to assess the presence of common symptoms and behaviors in EDs as well as symptoms in commonly diagnosed, comorbid psychiatric disorders. For the present study, three DHQ items were used as measures of excessive exercise, binge eating, and

dietary restriction, wherein participants were to "Please rate how much [you] have engaged in the following behaviors since [your] last meal or snack from 1 (not at all) to 6 (a lot)." DHQ items for guilt and anxiety were also used as a measure for NA, in which participants were asked, "How anxious [or guilty] are you feeling currently about your last meal or snack?" on a scale from one to six. The DHQ has demonstrated strong reliability in accurately assessing mealtime cognitions (Levinson et al., 2018). For a summary of the present study's use of the DHQ, see Table 3.

1.4. Data Analytic Procedures

Statistical analyses were computed in R programming language version 4.2.0 (R Core Team, 2022), using the *nlme* package for multilevel modeling (MLM; Pinheiro et al., 2021). Participants completed four EMA surveys per day for 25 days, allowing for a total of 100 within-person observations. Average item-level missingness was 21.28% (range = 0.00-49.5%). Missing items were imputed using the Kalman filter of the imputeTS package (Moritz & Bartz-Beielstein, 2017). Mixed effects models allowed us to examine the relationship between variables both within individuals (level one) and between individuals over time (level two). Level one data consisted of repeated measures collected at each survey, which were nested within level two units (i.e., participants). As suggested by prior research, time-varying predictors (i.e., predictor variables administered via EMA) were disaggregated into between-person (i.e., person mean-centered average; level two) and within-person (i.e., individual observation minus their person mean-centered average; level one).

To test our hypotheses examining whether state and trait NA (i.e., guilt/anxiety) during meals predict post-meal ED behaviors (i.e., binge eating, dietary restriction, driven exercise), we conducted three sets of linear mixed effects models: each set using one post-meal ED behavior as

a dependent variable. We also conducted models testing worry and depression as moderators of the relationship between momentary (i.e., state) NA and ED behaviors. Within these models, we included between- and within-person variables as fixed effects. Intercepts were allowed to vary across persons, and trait and state NA at the previous time point were also included as fixed effects.

We built our base models (i.e., models testing relationship between NA and the three ED behaviors) in the following steps: 1) intercept only models; 2) models with intercept and level one effects; 3) models with intercept, level one, and level two effects and 4) models with intercept, level one and two effects, and random slopes for level one effects. Models reported are those that best fit the data (as determined by AIC and BIC values Likelihood ratio tests at $p <$ .05). Significance for fixed effects was set at *alpha* < .05. Residuals were normally distributed for fixed and random effects. We report Cohen's *d* as a measure of effect for fixed effects using the *EMAtools* package (Kleiman, 2021).

2. Results

2.1. Demographic Characteristics

One hundred thirty participants with current or past ED diagnoses were included in present analyses. 82.31% of participants reported their race as White (n = 107) and 95.38% of participants reported their gender as cisgender woman (n = 124). The mean age was 29.33 (SD = 9.27; Range = 16 – 62). The majority of participants met criteria for a current ED (n = 108), and the remaining participants had been diagnosed with an ED in the past (n = 22). A full diagnostic overview and complete demographic information can be found in Table 2.

2.2. Prevalence of Worry and Depression

In assessing the prevalence and severity of worry and depressive symptoms, score ranges on the PSWQ and BDI-II were used. 2.31% of participants (n = 3) provided responses indicating low worry; moderate worry was endorsed by 26.15% of participants (n = 34); high worry was endorsed by 70.00% of participants (n = 91), and 1.54% of participants (n = 2) did not complete the PSWQ. Alternatively, 26.92% of participants (n = 35) provided responses indicating minimal depression; mild depression was endorsed by 15.38% of participants (n = 20); moderate depression was endorsed by 24.62% of participants (n = 32); severe depression was endorsed by 31.54% of participants (n = 41), and 1.54% of participants (n = 2) did not complete the BDI-II. For a prevalence overview of worry and depressive symptoms, see Table 4.

2.3. Negative Affect and Binge Eating Analysis

Our first model assessed whether mealtime NA (i.e., guilt/anxiety) predicted post-meal binge eating. Both state (b = 0.08, p < .001, d = 0.45) and trait (b = 0.06, p = .044, d = 0.36) NA were significant, such that those who experienced higher mealtime NA were more likely to engage in a binge episode after their meal (Table 5). However, when including trait-level worry as a moderator and predictor, neither trait (b = 0.05, p =.055, d = 0.35) nor state (b = 0.08, p =.323, d = 0.02) NA predicted binge eating. For the model including depressive symptoms as a moderator and predictor, both trait (b = 0.06, p =.036, d = 0.38) and state (b = 0.08, p < .001, d = 0.22) NA predicted binge eating. Neither worry nor depressive symptoms were significant as predictors (worry: b = 0.00, p = .984, d = 0.00; depressive symptoms: b = 0.00, p = .781, d = 0.00) or as moderators (worry: b = 0.00, p = .934, d = 0.00; depressive symptoms: b = 0.00, p = .308, d = 0.02) of binge eating.

2.4. Negative Affect and Dietary Restriction Analysis

We then analyzed the role of mealtime NA (i.e., guilt/anxiety) in predicting post-meal urges to restrict food intake. Both state (b = -0.04, p < .001, d = 0.14) and trait (b = 0.30, p < .001, d =1.41) NA were significant predictors of dietary restriction, such that those with higher mealtime levels of NA were more likely to restrict food intake after their meal (Table 6). When including trait-level worry as a predictor and moderator, only trait NA (b = 0.31, p < .001, d = 1.45) and not state NA (b = -0.02, p = .415, d = 0.01) significantly predicted dietary restriction. When including depressive symptoms as a predictor and moderator, both state (b = -0.03, p < .001, d = 0.05) and trait (b = 0.26, p < .001, d = 0.98) NA were significant predictors of dietary restriction. Neither worry nor depressive symptoms were significant as predictors (worry: b = 0.00, p = .747, d = 0.06; depressive symptoms: b = 0.01, p = .113, d = 0.29) or as moderators (worry: b = 0.00, p = .616, d = 0.01; depressive symptoms: b = -0.00, p = .387, d = 0.02) of dietary restriction.

2.5. Negative Affect and Driven Exercise Analysis

Our final models first involved defining the role of mealtime NA (i.e., guilt/anxiety) in predicting post-meal engagement in driven (i.e., excessive) exercise. While the interaction between trait NA and driven exercise was not significant (b = -0.0003, p = .916, d = 0.00), state NA was a significant predictor (b = 0.11, p < .001, d = 0.66), such that those who experienced higher mealtime state NA were more likely to engage in driven exercise following their meal (Table 7). When trait-level worry was included as a moderator and predictor, both trait (b = 0.10, p < .001, d = 0.60) and state (b = -0.07, p < .001, d = 0.07) NA were significant predictors of driven exercise. Similarly, when depressive symptoms were included as a moderator and predictor, both trait (b = 0.08, p = .025, d = 0.41) and state (b = -0.02, p = .004, d = 0.05) NA remained significant predictors of driven exercise. While worry and depressive symptoms were

both non-significant as predictors (worry: $b = .002$, $p = .684$, $d = 0.07$; depressive symptoms: $b = 0.01$, $p = .307$, $d = 0.18$), worry and depressive symptoms were each significant moderators (worry: $b = .001$, $p < .001$, $d = 0.07$; depressive symptoms: $b = 0.00$, $p = .002$, $d = 0.06$) of the relationship between driven exercise and state NA.

3. Discussion

The present study investigated the presence of three ED behaviors—binge eating, dietary restriction, and driven exercise—within an ED sample, and further quantified the role of symptoms (i.e., depression and worry) experienced in commonly comorbid psychiatric disorders, in order to further elucidate the momentary mechanisms which maintain ED psychopathology. Our prevalence analyses for worry and depressive symptoms substantiated their implication in the ED population. Our MLM analyses first revealed that the relationship between NA and binge eating may be better explained by depressive symptoms than by worry. In assessing the relationship between NA and dietary restriction, depressive symptoms were similarly implicated, while worry emerged as one potential explanation for the association between NA and dietary restriction, when NA was considered characteristically rather than momentarily (i.e., trait versus state). Although NA remained implicated to differing extents during the consideration of depressive symptoms and worry within both models, worry and depressive symptoms themselves were nonsignificant as indicators of the relationship between NA and binge eating/dietary restriction. In our last set of analyses, however—in addition to NA's continued association with driven exercise in the consideration of both worry and depressive symptoms—we found that the strength of the relationship between NA and driven exercise was significantly moderated by both worry and depressive symptoms.

Our prevalence analyses for worry and depressive symptoms substantiates previous literature's quantification of both worry and depression as common risk factors for the onset and progression of ED symptoms (Herzog et al., 1996; Swinbourne et al., 2012; Garcia et al., 2020). However, the present analysis also suggests that, while depression may be endorsed in a more uniform distribution across severities, individuals with an ED may be more likely to endorse clinically severe levels of worry. While the current study suggests that depression may play a slightly more central role than worry in the momentary maintenance of EDs, our significant implication of severe worry in those with past or present ED diagnoses warrants more specific analyses to further quantify the mechanisms by which worry interacts with ED symptomatology.

The present implication of both state and trait NA in post-meal binge eating behaviors aligns with previous literature's quantification of high NA predicting likelihood for engagement in binge episodes (Stice et al., 2002; Wonderlich et al., 2015; Berg et al., 2017). This association is well-supported, so the finding that NA was no longer significantly related to binge eating in the consideration of worry—contrary to our predictions in H2a—was slightly unexpected. Previous literature suggests that both guilt and anxiety are implicated as significant factors in maintaining binge eating behaviors (McManus & Waller, 1995). It may be, then, that our assessment of NA here—by momentary measures for guilt and anxiety in the presence of worry—captures an aspect of binge eating with presently sparse quantification, suggesting rather that worry may be less related to subjective experiences of NA as a motivation to binge eat than previously posited (Stickney et al., 1999). Nevertheless, the consistency of the relationship between NA and binge eating in the consideration of depressive symptoms exemplifies the direct associations between rumination and depression, and differentially supports our predictions in H2a (Hjartarson et al., 2022). Together, alongside the finding that worry and depression are not

themselves adequate accounts for the association between NA and binge eating, these findings point to the notion that treatment of depressive cognitions should be prioritized in individuals who present with binge eating psychopathologies and endorse significant levels of NA. Indeed, such a suggestion has already found support within previous analyses (Linardon et al., 2017). In so doing, individuals with binge eating tendencies could see improvements not only in their engagement with unhealthy eating behaviors, but also in their mood and quality of life.

The implication of NA in dietary restriction was similarly supported by both present and previous analyses (Pila et al., 2019). While considering the role of depressive symptoms in the relationship between NA and dietary restriction, both trait and state NA maintained their associations, which substantiates the notion that depression and the ruminative cognitions implicated therein may be centralized in the maintenance of restrictive ED behaviors (Harrell & Jackson, 2008). In further support of the role rumination plays in regulating dietary restriction, we found here that trait, and not state NA, was a significant predictor of dietary restriction in the consideration of trait-level worry. Similarly, this qualification of worry's mechanisms in ED maintenance substantiates the notion that rumination and its preservation of negative emotional states beyond the present moment may explain continued engagement with restricting food intake. While such relationships have yet to be thoroughly characterized, present literature emphasizes differences in momentary intentions to restrict food intake versus more enduring and bona fide engagement with dietary restriction—a distinction often mediated by rumination (Reichenberger et al., 2019; Dondzilo et al., 2016). As supported by mounting literature, we believe that these results suggest that therapy which stresses emotional regulation techniques may be an effective means of reducing both the perceived adversity of subjectively negative

emotions, as well as engagement with significantly harmful, restrictive ED behaviors (Haynos & Fruzzetti, 2011).

While the relationships between both binge eating and dietary restriction with NA were not explicable by worry and depression themselves, worry and depressive symptoms emerged as significant moderators of the relationship between driven exercise and state NA. That is, while individuals were experiencing higher levels of momentary NA, engagement in driven exercise was significantly more likely when feelings of depression or worry were also high. Here, NA remained significantly associated with driven exercise in the presence of both worry and depressive symptoms. While slightly antithetical to our predictions in H2c—that worry alone, and not depression, would be associated with driven exercise—these results both support previous literature and supplement it with the finding that those with an ED may be more at risk for engagement in excessive exercise during their subjective experiences with negative emotion, and especially while also feeling worried or depressed (Renz et al., 2019; Schlegl et al., 2018). One difference which emerged in this analysis was that driven exercise was itself associated with state, but not trait, NA. While distinct from findings for our binge eating and dietary restriction analyses, previous literature on the role of exercise in ED pathology is disproportionately sparse given its frequent implication within ED maintenance (Meyer et al., 2008). It may be, then—as these findings suggest—that there exist discrepancies between intent to excessively exercise and actual engagement with driven exercise, in a similar trend as that observed above for dietary restriction. As engagement with driven exercise may be made more likely by momentary and potentially fleeting negative emotional states while feeling worried or depressed, a similar focus on treating emotional dysregulation may prove effective in reducing impairment from excessive exercise and its related cognitions in both EDs and their comorbidities.

Altogether, these results stress the significance of the reciprocity shared between EDs and symptoms in comorbid depression and anxiety. Further, they provide additional evidence to suggest that, compared to more unidimensional explanations, Fairburn et al.'s (1993) transdiagnostic model may be an effective, more exhaustive characterization of EDs alongside separate, but frequently comorbid, psychopathologies. Finally, in generalizing these results clinically, we were able to suggest more targeted focuses for treatment—e.g., on emotional regulation—for specific factors which maintain EDs and could lead to potential improvements in their outcomes.

3.1. Strengths and Limitations

The present study possesses several strengths. We intended to characterize ED pathology alongside its comorbidities, and by using a sample with a diversity of ED diagnoses alongside a variety of measures for a broad array of psychiatric symptoms—considered both momentarily and characteristically—we were able to further elucidate the mechanisms by which depressive symptoms and worry maintain EDs. In line with this and previous discrepancies in explanations for the momentary interactions across ED symptoms and behaviors, our characterization of NA alongside worry and depressive symptoms supports a transdiagnostic approach to ED treatment and consideration, substantiating the theories of Fairburn et al. (1993). Additionally, such characterization provides clinical suggestions for more targeted treatment regimens, in regard to depressive cognitions and emotional dysregulation, which could enhance their efficacy. Finally, analyses of NA in EDs remain broad in their definition of NA, and our present assessment of guilt and anxiety not only substantiates the implication of NA within ED psychopathology, but further broadens its application within a diversity of subjectively negative emotional experiences (Gross, 2007; Haedt-Matt & Keel, 2011).

While strong in several respects, the present study is also limited by a variety of factors. The generalization of our results was limited demographically, as our sample was predominantly White, cisgender women (*n* = 106; 79.70%). Additionally, as a large portion of our data was obtained through self-report measures, the presence of self-report biases must be considered, as such measures assume the participants' honest and accurate provision of responses (Donaldson & Grant-Vallone, 2002). Finally, while our present definition of NA by momentary measures for guilt and anxiety captures an accurate, partial representation of negative emotional states, it neglects other variables often implicated in NA (i.e., sadness or shame), and assessing NA more broadly may lead future analyses to a more comprehensive characterization of the interactions shared across NA and ED symptomatology.

3.2. Directions for Future Research and Clinical Considerations

This study serves as a constructive advancement of the characterization of ED psychopathology as maintained by psychiatric comorbidities. As such, additional research is necessary to progress this understanding and more comprehensively quantify the contributions of psychiatric symptoms to EDs. First, the suggestions made for treatment herein could be advanced by studying the efficacy with which the promotion of emotional regulation strategies may improve ED severity alongside that of comorbid psychiatric disorders such as MDD or generalized anxiety disorder. Further, the distinct relationships between binge eating and depressive symptoms—as opposed to worry—should emphasize the importance in screening for, and potentially treating, depressive cognitions in the clinical consideration of binge eating behaviors. As the variation in observed results across ED behaviors may have been mediated by differences between intent versus actual engagement with these behaviors, future research should aim to quantify the precipitants which may encourage the shift from intention into engagement.

Including additional ED symptoms and cognitions—e.g., purging, fear of weight gain, calorie counting—as well as psychiatric symptoms and cognitions—e.g., obsessions, anhedonia, panic—in future analyses, too, would further elucidate a broader view of ED psychopathology within a more extensive consideration of variables. The present study further suggests that this may be more effectively facilitated by an increased focus on worry. Finally, resolving many of the above limitations by including a more diverse sample could improve the generalizability of any results, as well as their potential for clinical benefit.

3.3. Conclusion

As a whole, the present study serves as a meaningful expansion of the contributions which commonly comorbid psychiatric symptoms such as depression and worry make in maintaining ED psychopathology. While trait and state NA differentially predicted each of our three ED variables of interest—i.e., binge eating, dietary restriction, and driven exercise—NA remained generally implicated in each of these associations. Further, the inclusion of worry and depression suggests in each of these models that depression may be more implicated than worry in the relationship between NA and the respective ED variable of interest. The relationships between NA and driven exercise, as well as trait NA and dietary restriction, were still significant in the consideration of trait-level worry. Meanwhile, the relationships among all three ED behaviors with both state and trait NA remained significant in the consideration of depressive symptoms. Additionally, in the relationship between NA and driven exercise, individuals experiencing high NA were more likely to engage in excessive exercise while also feeling depressed or worried. These results corroborate a transdiagnostic approach to ED treatment—i.e., a consideration of ED psychopathology which centralizes the context of any comorbid psychiatric symptoms. Further, to materialize improvements in the outcomes of such

transdiagnostic treatment methods and the quality of life for individuals living with ED comorbidities, the consideration of depression and the efficacy of emotional regulation strategies in reducing ED and psychiatric symptom severities should be thoroughly assessed.

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Table 1. Overview of common eating disorder diagnoses.

Diagnosis	Diagnostic Criteria	Prevalence (%)
Anorexia Nervosa	Significant weight loss from lack of sufficient nutriment Preoccupied fear around weight gain or the idea of becoming fat Distress around the way in which one perceives their body	0.8 – 1.9
Binge Eating Disorder	Repeated engagement in episodes of eating much more than what would be typically considered normal, accompanied by a sense of loss of control Feelings of distress around these episodes	3.0 – 3.6
Bulimia Nervosa	Similar engagement in binge eating episodes as described above, although succeeded by compensatory behaviors (e.g., purging, misuse of laxatives/diuretics, excessive exercise)	Up to 2.6
Other Specified Feeding or Eating Disorder	Used to classify eating disorders that do not readily and wholly meet criteria for any standard eating disorder	Up to 11.0

Note. Diagnostic criteria summarized from the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (American Psychiatric Association, 2013). Prevalence statistics gathered for females throughout their lifetimes (Dahlgren et al., 2017).

Table 2. Participant demographic and descriptive statistics.

	M (SD)	**Range**
Age	29.33 (9.27)	16 – 62
	***n* (%)**	
Race		
Asian or Pacific Islander	7 (5.38)	
Black	3 (2.31)	
Hispanic	8 (6.15)	
Multi- or Bi-racial	4 (3.08)	
Native American	1 (0.77)	
White, non-Hispanic	107 (82.31)	
Gender		
Woman	124 (95.38)	
Man	4 (3.08)	
Not listed	1 (0.77)	
No response	1 (0.77)	
Eating Disorder (ED) Diagnosis		
Anorexia Nervosa	35 (26.92)	
Binge Eating Disorder	4 (3.08)	
Bulimia Nervosa	17 (13.07)	
OSFED	52 (40.00)	
Past ED Diagnosis	22 (16.92)	

Note. $N = 130$. OSFED = other specified feeding or eating disorder.

Table 3. Overview of Daily Habits Questionnaire (DHQ) items used.

DHQ Item Prompt	Construct Measured
"How anxious are you feeling currently about your last meal or snack?"	Negative affect
"How guilty are you feeling currently about your last meal or snack?"	Negative affect
"Please rate how much you have engaged in the following behaviors since your last meal or snack from 1 (not at all) to 6 (a lot):"	
"Binge eating"	Engagement in binge eating
"Restriction"	Engagement in dietary restriction
"Excessive exercise"	Engagement in excessive exercise

Table 4. Prevalence data for depression and worry.

Severity (BDI-II Score Range)	***n* (%)**	**Severity (PSWQ Score Range)**	***n* (%)**
Minimal depression (0-13)	35 (26.92%)	Low worry (16-39)	3 (2.31%)
Mild depression (14-19)	20 (15.38%)	Moderate worry (40-59)	34 (26.15%)
Moderate depression (20-28)	32 (24.62%)	High worry (60-80)	91 (70.00%)
Severe depression (29-63)	41 (31.54%)	No response	2 (1.54%)
No response	2 (1.54%)		

Note. N = 130. BDI-II = Beck Depression Inventory-II (Beck et al., 1996). PSWQ = Penn State Worry Questionnaire (Meyer et al., 1990).

Table 5. Negative affect (NA) and binge eating analysis.

Fixed Effects	***b***	***SE***	***t*-value**	***p***	***d***
(Intercept)	1.37	0.06	22.43	< .001	
NA_{trait}	0.06	0.03	2.04	.044	0.36
NA_{state}	0.08	0.00	25.66	< .001	0.45
(Intercept)	1.37	0.37	3.67	< .001	
NA_{trait}	0.05	0.03	1.94	.055	0.35
NA_{state}	0.08	0.08	0.99	.323	0.02
Worry	0.00	0.01	0.02	.984	0.00
Worry*NA_{state}	0.00	0.00	0.08	.934	0.00
(Intercept)	1.37	0.12	11.08	< .001	
NA_{trait}	0.06	0.03	2.11	.036	0.38
NA_{state}	0.08	0.01	12.50	< .001	0.22
Depression	0.00	0.00	0.28	.781	0.05
Depression*NA_{state}	0.00	0.00	1.02	.308	0.02

Table 6. Negative affect (NA) and dietary restriction analysis.

Fixed Effects	***b***	***SE***	***t*-value**	***p***	***d***
(Intercept)	2.28	0.08	28.83	< .001	
NA_{trait}	0.30	0.04	8.05	< .001	1.41
NA_{state}	-0.04	0.00	8.25	< .001	0.14
(Intercept)	2.43	0.48	5.09	< .001	
NA_{trait}	0.31	0.04	8.11	< .001	1.45
NA_{state}	-0.02	0.03	0.82	.415	0.01
Worry	0.00	0.01	0.32	.747	0.06
Worry*NA_{state}	0.00	0.00	0.50	.616	0.01
(Intercept)	2.00	0.19	10.34	< .001	
NA_{trait}	0.26	0.04	5.48	< .001	0.98
NA_{state}	-0.03	0.01	2.92	< .001	0.05
Depression	0.01	0.01	1.60	.113	0.29
Depression*NA_{state}	-0.00	0.00	-0.87	.387	0.02

Table 7. Negative affect (NA) and driven exercise analysis.

Fixed Effects	***b***	***SE***	***t*-value**	***p***	***d***
(Intercept)	1.47	0.06	23.24	< .001	
NA_{trait}	-0.0003	0.00	0.11	.916	0.00
NA_{state}	0.11	0.03	3.73	< .001	0.66
(Intercept)	1.28	0.38	3.39	< .001	
NA_{trait}	0.10	0.03	3.34	< .001	0.60
NA_{state}	-0.07	0.02	3.97	< .001	0.07
Worry	.002	0.01	0.41	.684	0.07
Worry*NA_{state}	.001	0.00	4.04	< .001	0.07
(Intercept)	1.32	0.15	8.48	< .001	
NA_{trait}	0.08	0.04	2.27	.025	0.41
NA_{state}	-0.02	0.01	2.85	.004	0.05
Depression	0.01	0.01	1.03	.307	0.18
Depression*NA_{state}	0.00	0.00	3.17	.002	0.06