



The unique effects of angry and depressive rumination on eating-disorder psychopathology and the mediating role of impulsivity

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ABSTRACT

Negative affect and maladaptive emotion regulation strategies are associated with eating-disorder (ED) psychopathology. Depressive rumination is a maladaptive cognitive style associated with the onset, maintenance, and severity of ED psychopathology among both clinical and nonclinical samples. However, although anger is also strongly associated with ED behaviors, the associations between angry rumination and ED psychopathology, as well as mechanisms of the relationships between rumination and ED psychopathology, remain largely unknown. The current study sought to examine the unique influences of trait depressive and angry rumination on ED psychopathology and whether trait negative urgency (i.e., responding rashly to negative affect) mediated these relationships. Study 1 sampled undergraduate students ($N = 119$) cross-sectionally and longitudinally (five months), and Study 2 sampled patients with eating disorders ($N = 85$). All participants completed questionnaires assessing angry rumination, depressive rumination, ED psychopathology, and negative urgency. Angry rumination had consistent indirect effects on ED psychopathology via negative urgency among both clinical and nonclinical samples. However, there was mixed support for the influence of depressive rumination: whereas depressive rumination showed total and indirect effects on ED psychopathology in Study 1 cross-sectional analyses, no total or indirect effects emerged in Study 1 longitudinal analyses or in Study 2. Associations between depressive rumination and ED psychopathology may reflect the strong overlap between angry and depressive rumination. Interventions targeting angry rumination and negative urgency may enhance prevention and treatment of disordered eating across eating disorder diagnosis and severity.

1. Introduction

Negative affect is a fundamental component of etiological and maintenance models of eating disorders. Affect regulation models of eating disorders hypothesize that disordered eating behaviors serve an emotion regulation function for individuals experiencing negative affect who do not have more adaptive coping skills (Haynos & Fruzzetti, 2011; Polivy & Herman, 1993). Supporting these theories, heightened anger and sadness often precedes disordered eating (Lavender et al., 2016), and associations between emotion dysregulation and eating-disorder (ED) psychopathology in clinical (Lavender et al., 2015) and nonclinical (Haynos, Wang, & Fruzzetti, 2018) populations are well-established. However, coping with negative emotion is a broad construct that may confound the roles of more specific forms of coping. A better understanding of specific maladaptive strategies used for handling negative affect in ED psychopathology has the potential to inform treatments. Rumination, or a cognitive style involving the tendency to perseverate on negative feelings and problems, has received recent

attention as a maladaptive response to negative moods (Watkins, 2008). Researchers define different types of rumination based on the predominant negative content of the repetitive thoughts (Ciesla, Dickson, Anderson, & Neal, 2011), with depressive and angry rumination being distinct constructs that are differentially associated with various psychopathologies (du Pont, Rhee, Corley, Hewitt, & Friedman, 2017; Peled & Moretti, 2007, 2010). The current study explores the unique roles of both depressive and angry rumination, and a potential mediating mechanism, in predicting ED psychopathology in nonclinical and clinical samples.

Depressive rumination is consistently implicated in the development and maintenance of ED psychopathology. Depressive rumination prospectively predicts the onset of binge-eating and purging behaviors among undergraduates (Gordon, Holm-Denoma, Troop-Gordon, & Sand, 2012) and adolescents (Nolen-Hoeksema, Stice, Wade, & Bohon, 2007). Individuals with anorexia nervosa (AN) and bulimia nervosa (BN) also report significantly more depressive rumination than healthy individuals (Cowdrey & Park, 2012). Moreover, depressive rumination

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is associated with severity of ED psychopathology among nonclinical samples (Wang & Borders, 2017), individuals with AN (Rawal, Park, & Williams, 2010; Startup et al., 2013), BN (Naumann, Tuschen-Caffier, Voderholzer, Caffier, & Svaldi, 2015), and binge-eating disorder (BED) and obesity (Wang, Lydecker, & Grilo, 2017).

By contrast, almost no literature has examined associations between angry rumination and ED psychopathology. Defined as a repetitive focus on anger and its causes and consequences (Sukhodolsky, Golub, & Cromwell, 2001), angry rumination exacerbates existing angry affect and is associated with emotion dysregulation, impulsivity, and risky behaviors (Selby, Anestis, & Joiner, 2008). Despite well-established associations between difficulties coping with anger and ED psychopathology (Waller et al., 2003), only two studies have examined angry rumination and ED psychopathology, showing positive correlations between angry rumination and symptoms of bulimia in undergraduates (Selby et al., 2008) and individuals with borderline personality disorder (Selby, Anestis, Bender, & Joiner, 2009). However, whether angry rumination is more broadly associated with ED psychopathology, and whether angry and depressive rumination uniquely influence ED psychopathology, remains unknown. Although angry and depressive rumination both involve negative repetitive thought processes, depressive rumination appears to be more strongly associated with internalizing psychopathology, whereas angry rumination is more strongly associated with externalizing psychopathology (Ciesla et al., 2011; du Pont et al., 2017; Peled & Moretti, 2010). Although some studies have found EDs to load onto a latent internalizing factor (Mitchell, Wolf, Reardon, & Miller, 2014), EDs are also strongly associated with externalizing disorders such as substance use disorders (Hudson, Hiripi, Pope, & Kessler, 2007). Thus, examining the unique influence of depressive and angry rumination on ED psychopathology extends this previous research and may enhance our understanding of how various types of rumination are differentially associated with psychological functioning.

Moreover, little is known about mechanisms underlying relationships between rumination and ED psychopathology. Impulsivity, and specifically negative urgency (i.e., responding rashly to negative affect), is one such potential mechanism. Both angry (Ciesla et al., 2011) and depressive (Valderrama, Miranda, & Jeglic, 2016) rumination are strongly correlated with negative urgency, and general negative rumination prospectively predicts increased negative urgency (Selby et al., 2008). Experimental evidence has also shown rumination to decrease self-control capacity, which is a related construct to negative urgency (Denson, Pedersen, Friese, Hahm, & Roberts, 2011). Negative urgency, in turn, prospectively predicts disordered eating in adolescents (Pearson, Combs, Zapolski, & Smith, 2012) and college students (Fischer, Peterson, & McCarthy, 2013). Thus, negative urgency may mediate associations between angry and depressive rumination and ED psychopathology. Individuals who continuously dwell on anger- or sadness-inducing experiences may be more likely to respond rashly to these unpleasant emotions, and engage in disordered eating behaviors to escape these negative thoughts and feelings (Heatherton & Baumeister, 1991).

The current pair of studies examined unique associations between angry and depressive rumination and ED psychopathology in nonclinical and clinical samples. We also examined negative urgency as a mechanism of these associations. Study 1 assessed these relationships longitudinally among a nonclinical sample of undergraduate students, and Study 2 examined these associations cross-sectionally in a mixed diagnostic group of treatment-seeking individuals with EDs. In both samples, we hypothesized that angry and depressive rumination would uniquely predict increased ED psychopathology. As ruminating on anger and sadness is associated with greater negative urgency, which is further associated with ED psychopathology, we also hypothesized that negative urgency would mediate these associations.

2. Study 1 method

2.1. Participants and procedure

Participants were 126 undergraduates recruited from a Psychology subject pool at a Northeastern college ($M_{\text{age}} = 19.7$, $SD_{\text{age}} = 1.10$, 84% female) who completed the survey at baseline and five-month follow-up. Participants identified as White/Caucasian, (63.2%), Asian/Asian American (10.5%), Hispanic/Latino (7.9%), South Asian/Indian (5.8%), Black/African American (2.6%), Arab/Arab American (1.1%), American Indian/Native American (0.01%), multi-ethnic (4.2%), and “other” (0.2%). Seven participants indicated that they were diagnosed with an eating disorder at some point in their lives and were excluded from all analyses, resulting in a final sample of 119 participants.

Participants completed all questionnaires at baseline (time 1) and were compensated with course credit. Five months later (time 2), participants were informed of a follow-up study. If they agreed, participants completed the same measure of ED psychopathology and received course credit or a \$5 gift card as compensation. All participants provided informed consent before completing the initial 30-minute online questionnaire. All measures were presented in a randomized order. The college's Institutional Review Board (IRB) approved all research and recruitment procedures for Study 1.

2.2. Measures

2.2.1. Anger rumination

The 19-item Anger Rumination Scale (Sukhodolsky et al., 2001) assessed unintentional and repetitive thoughts that occur during and after angering situations. Items are rated on a scale from 1 (almost never) to 4 (almost always). Higher averaged scores indicate more angry rumination. This scale has good internal consistency and test-retest reliability and correlates strongly with measures of anger experience and expression (Sukhodolsky et al., 2001). In the current sample, Cronbach's alpha was 0.95.

2.2.2. Depressive rumination

The 22-item Ruminative Responses Scale (Nolen-Hoeksema & Morrow, 1991) assessed repetitive thoughts about the causes and consequences of sad moods. Items are rated on a scale from 1 (almost never) to 4 (almost always). Higher averaged scores indicate more depressive rumination. Individuals with higher scores are more likely to become depressed (Nolen-Hoeksema & Morrow, 1991), and the scale correlates highly with measures of depressive symptoms (Roberts, Gilboa, & Gotlib, 1998). In the current sample, Cronbach's alpha was 0.95.

2.2.3. Negative urgency

The 12-item negative urgency subscale of the UPPS Impulsive Behavior Scale (Cyders et al., 2007) assessed emotion-based rash action. Items are rated on a scale of 0 (not at all) to 4 (very much). Higher averaged scores indicate higher impulsivity. UPPS subscales differentiate between individuals with borderline personality disorder, pathological gambling, alcohol abuse, and healthy controls (Whiteside, Lynam, Miller, & Reynolds, 2005). In the current sample, Cronbach's alpha was 0.87.

2.2.4. Eating-disorder psychopathology

The 28-item Eating Disorder Examination-Questionnaire (Fairburn & Beglin, 1994) assessed ED psychopathology. Items are rated on a scale from 0 (no days) to 6 (every day). Higher averaged scores indicate greater ED psychopathology. The EDE-Q has shown strong validity (Berg, Peterson, Frazier, & Crow, 2012) and test-retest reliability (Rose, Vaewsorn, Rosselli-Navarra, & Wilson, 2013). In the current sample, Cronbach's alpha was 0.83 (time 1) and 0.90 (time 2).

2.3. Data analysis

Our primary analyses tested path models using MPlus version 7.0 (Muthén & Muthén, 1998–2012), with the missing-at-random assumption (Little & Rubin, 1987) and maximum likelihood estimation. We first created total effects and mediational models with ED psychopathology measured concurrently with the other variables (T1). We then created the same models with ED psychopathology measured five months later (T2). In all models, variables were modeled as single observed variables, the two rumination variables were allowed to co-vary, and gender was entered as a covariate. All models were just identified.

We examined mediation by calculating indirect effects of the two independent variables (T1 angry and depressive rumination) via negative urgency on ED psychopathology. Indirect effects were examined using 5000 bootstrap samples (Preacher & Hayes, 2008). We calculated the bootstrap estimates and bias-corrected 95% confidence intervals for each indirect effect; a confidence interval that does not include zero indicates a significant effect of mediation.

3. Results

3.1. Descriptive statistics

Descriptive statistics and correlations are shown in Table 1. Women reported more ED psychopathology than men at Time 1 ($p < .05$); we thus controlled for gender in all analyses. In the full sample, greater angry and depressive rumination were correlated with more negative urgency and ED psychopathology. Negative urgency was correlated with more ED psychopathology.

3.2. Cross-sectional path analyses

We first fit a total effects model with paths from T1 angry and depressive rumination to T1 ED psychopathology (see Fig. 1a). Depressive ($\beta = 0.31, p = .002$) but not angry ($\beta = 0.15, p = .16$) rumination uniquely predicted ED psychopathology. The two independent variables were significantly intercorrelated ($\beta = 0.58, p < .001$). We next created a mediation model (see Fig. 1b) with paths from the independent variables (T1 angry and depressive rumination) to the mediator (T1 negative urgency) and dependent variable (T1 ED psychopathology). Depressive ($\beta = 0.27, p = .004$) and angry ($\beta = .38, p < .001$) rumination predicted negative urgency. In turn, negative urgency predicted

Table 1
Descriptive statistics and correlations among variables in Study 1 and Study 2.

	M (SD) range		Correlations			
	Study 1	Study 2	1.	2.	3.	4.
1. Depressive rumination (T1)	2.06 (0.60) 1.00–3.78	2.73 (0.69) 1.00–4.00	–	<i>0.54**</i>	<i>0.29*</i>	<i>0.47**</i>
2. Angry rumination (T1)	2.04 (0.65) 1.00–4.00	2.22 (0.78) 1.05–4.00	<i>0.59**</i>	–	<i>0.48**</i>	<i>0.35**</i>
3. Negative urgency (T1)	2.08 (0.62) 1.10–3.60	2.63 (0.70) 1.08–4.00	<i>0.56**</i>	<i>0.51**</i>	–	<i>0.43**</i>
4. ED psychopathology (T1)	2.80 (1.34) 1.00–5.91	5.11 (1.52) 1.00–7.00	<i>0.40**</i>	<i>0.42**</i>	<i>0.37**</i>	–
5. ED psychopathology (T2)	2.70 (1.45) 1.00–6.36	–	<i>0.32*</i>	<i>0.40**</i>	<i>0.34**</i>	<i>0.84**</i>

Note: T1 = Time 1; T2 = Time 2. Correlations in the bottom half of the table are from Study 1 (nonclinical sample; $N = 119$). Correlations in the top half (in italics) are from Study 2 (clinical sample, $N = 85$). ED psychopathology was only assessed at T1 for Study 2.

* $p < .01$.
** $p < .001$.

ED psychopathology ($\beta = 0.37, p < .001$). Again, the effect of depressive ($\beta = 0.21, p = .04$) but not angry ($\beta = 0.01, p = .92$) rumination on ED psychopathology was significant. Estimated indirect effects and their confidence intervals (see Table 2) indicated that negative urgency mediated the paths between both angry and depressive rumination and ED psychopathology.

3.3. Longitudinal path analyses

We fit a total effects model with paths from T1 angry and depressive rumination to T2 ED psychopathology, controlling for T1 ED psychopathology. T1 ED psychopathology predicted T2 ED psychopathology ($\beta = 0.78, p < .001$). However, neither angry ($\beta = -0.09, p = .23$) nor depressive ($\beta = 0.13, p = .07$) rumination uniquely predicted ED psychopathology. The two independent variables were significantly intercorrelated ($\beta = 0.52, p < .001$).

We next created a mediation model with paths from the independent variables (T1 angry and depressive rumination) to the mediator (T2 negative urgency) and dependent variable (T2 ED psychopathology), controlling for T1 ED psychopathology. Again, T1 ED psychopathology predicted T2 ED psychopathology ($\beta = 0.72, p < .001$). Angry ($\beta = 0.31, p = .001$) but not depressive ($\beta = 0.17, p = .08$) rumination predicted negative urgency, and negative urgency predicted ED psychopathology ($\beta = 0.18, p = .01$). Neither angry ($\beta = -0.14, p = .05$) nor depressive ($\beta = 0.10, p = .16$) rumination directly predicted ED psychopathology. Estimated indirect effects and their confidence intervals indicated that negative urgency mediated the path between angry, but not depressive, rumination on ED psychopathology (see Table 2).

4. Study 1 discussion

In a nonclinical sample, angry and depressive rumination had different unique effects on ED psychopathology. Cross-sectional analyses revealed that depressive rumination had a total effect on ED psychopathology, and an indirect effect via negative urgency. By contrast, angry rumination influenced ED psychopathology only indirectly through negative urgency.

Longitudinal analyses indicated that the total unique effects of depressive and angry rumination on ED psychopathology 5 months later were not robust when controlling for T1 ED psychopathology. However, the indirect effect of angry rumination via negative urgency remained significant, suggesting that rash responding to repetitive thoughts about angering events may predict future changes in ED psychopathology.

Study 1 provided novel information about how depressive and angry rumination are uniquely related to ED psychopathology – directly and indirectly via negative urgency – in non-clinical undergraduates. Study 2 sought to expand on these results by examining the same cross-sectional associations among treatment-seeking individuals with diagnosed EDs.

5. Study 2 method

5.1. Participants and procedure

Participants were 85 patients admitted to inpatient and partial hospitalization treatment programs at a Northeastern medical center ($M_{age} = 24.57$, range = 11–60, $SD_{age} = 9.95$, 87.1% female). Simulation studies (Fritz & MacKinnon, 2007) indicated that our sample size exceeded the minimum suggested sample size (recommended $n = 54–71$) achieve adequate (0.80) power for bias-corrected bootstrap mediation analyses. Participants identified as White/Caucasian, (87.1%), Asian/Asian American (3.7%), Hispanic/Latino (2.5%), Arab/Arab American (1.1%), multi-ethnic (1.2%), and “other” (1.2%). Participants had primary diagnoses of AN (79%), BN (9%), and other specified feeding and eating disorders (12%).

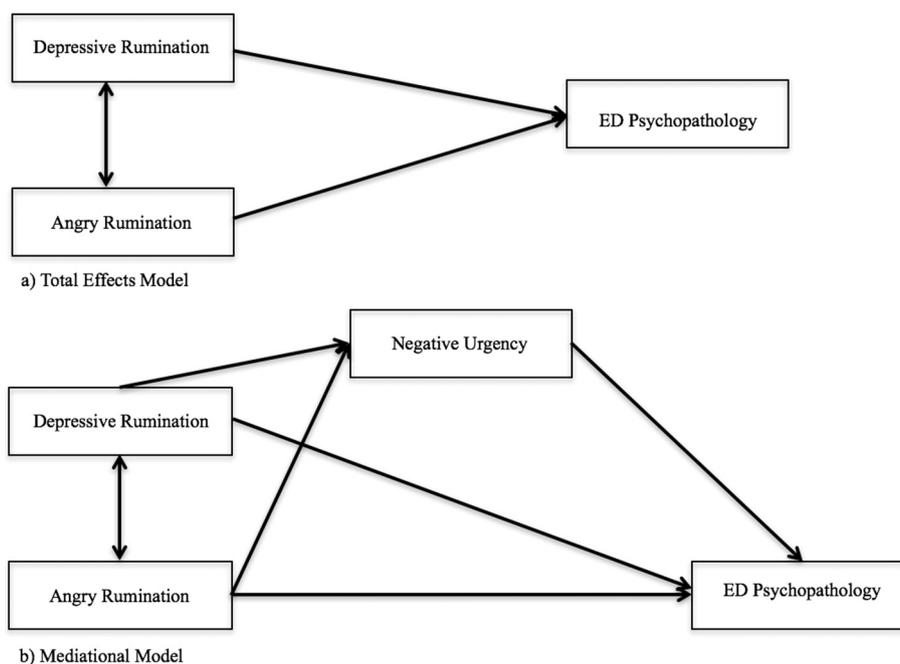


Fig. 1. a) Total effects model of depressive and angry rumination predicting eating-disorder (ED) psychopathology and b) mediational model with negative urgency mediating the associations between depressive and angry rumination and ED psychopathology.

Table 2
Unstandardized indirect effects and confidence intervals for negative urgency mediating the associations between rumination and eating-disorder psychopathology.

Independent variable	Bootstrap-estimated indirect effects	95% CI ^a
Study 1 (nonclinical sample; N = 119)		
Cross-sectional analyses		
Angry rumination	0.25	(0.07, 0.43)
Depressive rumination	0.20	(0.03, 0.36)
Longitudinal analyses ^b		
Angry rumination	0.11	(0.004, 0.22)
Depressive rumination	0.06	(-0.02, 0.15)
Study 2 (clinical sample; N = 85)		
Angry rumination	0.12	(0.03, 0.21)
Depressive rumination	0.00	(-0.22, 0.23)

^a Bias-corrected 95% confidence intervals (CI) that do not contain 0 indicate a significant indirect effect.

^b Models predicting eating-disorder (ED) psychopathology at Time 2 controlled for ED psychopathology at Time 1.

A research team member approached participants within a week of admission and informed of an opportunity to participate in a research study. Participants were included in the study if they were over 10 years old, spoke English, and provided informed consent/assent (participants younger than 18 years old provided assent and their parents/legal guardians provided informed consent). Participants were excluded if they had severe medical or psychiatric issues that limited their mobility or cognitive functioning. Participants or parents/legal guardians also provided informed consent for information to be collected from their medical records. After providing consent/assent, participants completed online questionnaires in a quiet room. All measures were presented in a randomized order. The hospital's IRB approved all research and recruitment procedures for Study 2.

5.2. Measures

All measures from Study 1 were included in Study 2. In this sample, Cronbach's alphas were 0.95, 0.93, 0.87, and 0.96 for the angry rumination, depressive rumination, negative urgency, and ED

psychopathology scales, respectively. Although the current sample included younger participants (n = 7 under 16 years old), the EDE-Q has been used in participants as young as nine years old (Binford, Le Grange, & Jellar, 2005), and the UPPS has been used in participants as young as ten (Yau, Potenza, Mayes, & Crowley, 2015).

6. Results

6.1. Descriptive statistics

Descriptive statistics and correlations are reported in Table 1. Participant age was not significantly correlated with any study variables. Women reported more depressive rumination than men (p = .04); thus, we included gender as a covariate in all analyses. In the entire sample, greater angry and depressive rumination were correlated with higher negative urgency and ED psychopathology. Negative urgency was correlated with more ED psychopathology.

6.2. Path analyses

We first fit a total effects model with direct paths from angry and depressive rumination to ED psychopathology (Fig. 1a). The rumination variables were allowed to covary, and gender was included as a covariate. The model was just identified. Neither angry (β = -0.08, p = .58) nor depressive (β = 0.15, p = .28) rumination uniquely predicted ED psychopathology. The two independent variables were significantly intercorrelated (β = 0.53, p < .001).

We next created a mediation model (Fig. 1b) with paths from the independent variables (angry rumination, depressive rumination) to the mediator (negative urgency) and dependent variable (ED psychopathology). As before, the independent variables were allowed to covary, and gender was included as a covariate. The model for the entire sample was just identified. Angry (β = 0.46, p < .001) but not depressive (β = -0.01, p = .99) rumination predicted negative urgency. In turn, negative urgency predicted ED psychopathology (β = 0.40, p < .001). Neither angry (β = -0.26, p = .06) nor depressive (β = 0.17, p = .21) rumination directly predicted ED psychopathology. Estimated indirect effects and their confidence intervals

indicated that negative urgency mediated the path between angry rumination and ED psychopathology (Table 2). Negative urgency did not mediate the path between depressive rumination and ED psychopathology.

7. Study 2 discussion

Study 2 examined unique effects of angry and depressive rumination on ED psychopathology in treatment-seeking individuals with diagnosed EDs. Similar to results in Study 1, angry rumination was only indirectly associated with ED psychopathology through negative urgency. However, unlike Study 1, Study 2 found no unique total or indirect effects of depressive rumination on ED psychopathology.

8. General discussion

The current pair of correlational studies examined angry rumination, depressive rumination, negative urgency, and ED psychopathology among nonclinical (Study 1) and clinical (Study 2) samples. Both studies found remarkably consistent results for the unique indirect effect of angry rumination on ED psychopathology through negative urgency. Study 1 demonstrated that these findings persisted even when examining longitudinal change in ED psychopathology, controlling for Time 1 ED psychopathology. Study 2 further replicated these results among treatment-seeking individuals with diagnosed EDs. Although we cannot infer causation, the current studies suggest that ruminating in response to anger may increase tendencies to behave rashly in response to negative emotion, which in turn influence ED psychopathology. This is the first study to examine the effect of angry rumination on general ED psychopathology, as well as the first to control for the effect of depressive rumination. The consistent indirect effect via negative urgency in both clinical and nonclinical samples suggests that angry rumination may uniquely influence ED psychopathology by depleting individuals' self-regulatory resources, and particularly their ability to control their behavior when experiencing strong negative emotions. These results provide important new information that add to a growing literature on the influence of negative repetitive thought on ED psychopathology by demonstrating the consistent and unique effects of angry rumination, an important construct that has received considerably less attention than depressive rumination.

Although the current studies showed consistent support for the unique role of angry rumination in ED psychopathology, they revealed somewhat discrepant findings for the unique role of depressive rumination. Whereas Study 1 found a total unique effect of depressive rumination cross-sectionally, this relationship was marginally significant ($p = .07$) longitudinally and was not significant in a clinical sample. These results contrast with previous research showing significant associations between depressive rumination and ED psychopathology among nonclinical and clinical samples (e.g., Nolen-Hoeksema et al., 2007; Startup et al., 2013). Interestingly, the moderate-sized and significant zero-order correlations between depressive rumination and ED psychopathology in the current studies were similar to or larger than those in previous research (e.g., Holm-Denoma & Hankin, 2010; Nolen-Hoeksema et al., 2007; Rawal et al., 2010). However, the current studies are the first to examine the unique influence of depressive and angry rumination on ED psychopathology within the same model. Previous research has also found significant zero-order correlations among angry rumination, depressive rumination, and clinical outcomes but differential and unique effects of angry and depressive rumination when considered in the same regression model (Peled & Moretti, 2007, 2010). We note that the correlations between depressive rumination and ED psychopathology (although significant) were less consistent across time and the two samples than were correlations between angry rumination and ED psychopathology. Future research might control for current depressive symptoms when examining the effect of trait depressive rumination. Researchers should also continue to examine

whether depressive rumination is uniquely associated with ED psychopathology when considering the influence of other forms of negative repetitive thought across time as well as in both clinical and non-clinical samples.

Although Study 1 found an indirect effect of depressive rumination via negative urgency cross-sectionally, this effect was not significant longitudinally or in Study 2. This likely reflects inconsistent associations between depressive rumination and negative urgency: whereas depressive rumination was strongly correlated with negative urgency in Study 1, the correlation in Study 2 was much weaker. By contrast, angry rumination was strongly correlated with negative urgency in both samples. Thus, perhaps individuals with clinically significant eating disorders who engage in depressive rumination may not experience heightened impulsivity to the same extent as do those engaging in angry rumination. The Response Styles Theory posits that negative thinking is a mechanism of depressive rumination, and research has consistently shown depressive rumination to enhance negative and self-critical thoughts (Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008). Thus, greater self-criticism or perfectionism may better explain the link between depressive rumination and ED psychopathology. A recent review on the etiology of EDs found consistent predictive associations of self-oriented perfectionism on eating-related concerns (Culbert, Racine, & Klump, 2015). Future research might examine these and other potential mechanisms of the influence of depressive rumination on ED psychopathology, particularly in clinical samples.

Findings for the current studies should be considered in context of their limitations. Although our use of longitudinal methodology in Study 1 is a noteworthy strength, Study 2 utilized a cross-sectional design. We are thus unable to determine temporal associations between our variables or make any assumptions about causality within this clinical sample. It is possible, for instance, that ED psychopathology increased rumination via negative emotions (Cowardley & Park, 2011). Similarly, tendencies for negative urgency may make people more likely to engage in rumination. Future research should use longitudinal or experimental methodology to determine the temporal associations between these variables. In addition, it is possible that the unique variance left over after controlling for one type of rumination simply represented tendencies for anger and depression, rather than pure indices of angry and depressive rumination. Future research will need to tease apart the unique roles of negative emotion and ruminative thinking on ED psychopathology. Studies with larger samples could also examine whether a latent trait of negative repetitive thought is a better predictor of ED psychopathology than different types of rumination (e.g., McEvoy & Brans, 2013).

Other limitations include measurement and sampling issues. Although the wide age range of our clinical sample increases the generalizability of our findings, some measures used in the study (e.g., depressive and angry rumination) have not been validated in younger age groups. Future research might use youth-specific measures to examine whether these results replicate in adolescent samples. Our clinical sample was also primarily comprised of individuals with AN, raising the question of whether our results would be similar in individuals with BN or BED. Given that diagnoses characterized by binge-eating and purging behaviors are associated with heightened impulsivity (Claes et al., 2015), we expect that angry rumination would predict ED psychopathology via negative urgency in patients with BN and BED. However, future research will need to examine whether the current findings remain consistent across individuals with various EDs. As rumination is also strongly associated with other diagnoses such as major depressive disorder and substance use disorders (Nolen-Hoeksema et al., 2007), future research might also control for the presence of these disorders when examining the influence of depressive and angry rumination on ED psychopathology.

Despite these limitations, these studies contribute important new information about the role of ruminative thought processes on ED psychopathology. Angry rumination was indirectly related to ED

psychopathology through negative urgency among nonclinical and clinical (primarily AN) samples. By contrast, no consistent relationships between depressive rumination and ED psychopathology emerged when including angry rumination in the models. These findings highlight the need for future research to consider both angry and depressive rumination when assessing relationships between rumination and psychological functioning. Our results suggest that interventions targeting angry rumination and negative urgency may assist in reducing ED psychopathology across eating disorder diagnosis and severity, particularly for individuals with high levels of angry rumination and negative urgency. Additionally, prevention efforts targeting rumination and negative urgency among individuals with subthreshold disordered eating may reduce the risk of developing more severe ED psychopathology. Mindfulness-based interventions that use meditation techniques to teach patients how to be less reactive to unpleasant emotions effectively reduce rumination (Shapiro, Oman, Thoresen, Plante, & Flinders, 2008). There is also an increasing interest and empirical support for incorporating mindfulness-based therapies in the treatment of subthreshold and clinically significant EDs (Juarascio et al., 2013; Katterman, Kleinman, Hood, Nackers, & Corsica, 2014). Similarly, rumination-focused cognitive behavioral therapy (CBT) reduces rumination by teaching patients more helpful coping strategies and encouraging more concrete thinking (Watkins et al., 2011). Although rumination-focused CBT has not been used with patients with EDs, CBT is a well-established treatment for EDs, particularly BN and BED (Brownley, Berkman, Sedway, Lohr, & Bulik, 2007; Shapiro et al., 2007). Therefore, future research might examine whether rumination-focused CBT reduces ED psychopathology, and whether decreased rumination and negative urgency are mechanisms of these various treatments' efficacy. These clinical implications should be considered cautiously because they were not directly tested. However, future research of this kind would provide important information on the utility of an enhanced focus on reducing angry rumination and negative urgency in prevention and intervention efforts for individuals who engage in disordered eating behaviors.

Conflicts of interest

None.

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