Specificity of cognitive emotion regulation strategies: A transdiagnostic examination

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Despite growing interest in the role of regulatory processes in clinical disorders, it is not clear whether certain cognitive emotion regulation strategies play a more central role in psychopathology than others. Similarly, little is known about whether these strategies have effects transdiagnostically. We examined the relationship between four cognitive emotion regulation strategies (rumination, thought suppression, reappraisal, and problem-solving) and symptoms of three psychopathologies (depression, anxiety, and eating disorders) in an undergraduate sample (N = 252). Maladaptive strategies (rumination, suppression), compared to adaptive strategies (reappraisal, problem-solving), were more strongly associated with psychopathology and loaded more highly on a latent factor of cognitive emotion regulation. In addition, this latent factor of cognitive emotion regulation was significantly associated with symptoms of all three disorders. Overall, these results suggest that the use of maladaptive strategies might play a more central role in psychopathology than the non-use of adaptive strategies and provide support of a transdiagnostic view of cognitive emotion regulation.

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Introduction

Cognitive emotion regulation strategies are cognitive responses to emotion-eliciting events that consciously or unconsciously attempt to modify the magnitude and/or type of individuals' emotional experience or the event itself (Campbell-Sills & Barlow, 2007; Harvey, Watkins, Mansell, & Shafren, 2004; Rottenberg & Gross, 2007; Thompson, 1994; Williams & Bargh, 2007). In recent years, a substantial amount of work has been devoted to delineating the relationships between dispositions to use certain strategies and a variety of disorders, including depression (Garnefski & Kraij, 2006; Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008), mania (Feldman, Joormann, & Johnson, 2008), generalized anxiety disorder (Mennin, Holoway, Fresco, Moore, & Heimberg, 2007), post-traumatic stress disorder (Tull & Roemer, 2003), social anxiety disorder (Kashdan & Breen, 2008), and eating disorders (Nolen-Hoeksema, Stice, Wade, & Bohon, 2007; Piran & Cormier, 2005).

Overall, several cognitive emotion regulation strategies have been argued to have negative associations with psychopathology (i.e., adaptive) whereas others have been associated with the etiology and maintenance of clinical disorders (i.e., be maladaptive). Stress and coping theories (Billings & Moos, 1981; Carver, Scheier, & Weintraub, 1989; Folkman & Lazarus, 1986) and early cognitive-behavioral approaches to psychopathology (Beck, 1976; Cooper, Russell, Skinner, Frone, & Mudar, 1982; D'Zurilla, 1988; Marlatt, Baer, Donovan, & Kivlahan, 1988) suggested that reappraisal and problem-solving should be adaptive across a variety of contexts. Reappraisal involves generating benign or positive interpretations of a stressful situation as a way of reducing distress (Gross, 1998). Cognitive theories put maladaptive appraisal processes at the core of depression and anxiety (Beck, 1976; Clark, 1988; Salkovskis, 1998). More recently, Gross's (1998) influential model of emotion regulation highlights reappraisal as a strategy that results in positive emotional and physical responses to emotion-eliciting stimuli. Cognitive-behavioral therapies for depression and anxiety focus on teaching reappraisal skills (Beck, Rush, Shaw, & Emery, 1979; Clark & Wells, 1995).

Problem-solving responses are conscious attempts to change a stressful situation or contain its consequences (Billings & Moos, 1981). Problem-solving measures can include cognitions directed at solving a problem (e.g., brainstorming solutions, planning a course of action) or an orientation toward problem-solving as a way of coping with stressful circumstances. Problem-solving coping can have beneficial effects on emotions by modifying or eliminating stressors. Low problem-solving orientation or poor problem-solving skills have been associated with depression (Billings & Moos, 1981; D'Zurilla, Chang, Nottingham, & Faccinno, 1988), anxiety (Chang, Downey, & Salata, 2004; Kant, D'Zurilla, &
In the past, we examined the relationship between self-reports of these strategies and symptoms of four cognitive emotion regulation strategies and symptoms of four types of psychopathology: depression, anxiety, eating disorders, and substance use. 2 When we collapsed all the symptom types together, we found mixed evidence for specificity in the relationship between emotion regulation strategies and psychopathology: although we found that all four strategies were significantly associated with psychopathology, the maladaptive strategies of rumination and suppression were more strongly associated with symptoms than the adaptive strategies of reappraisal and problem-solving. Delineating which strategies have stronger associations with psychopathology can help us identify the ones that play a more central role in the development, maintenance, and remission of various disorders. This has implications for prevention programs that focus on the development of cognitive emotion regulation skills (e.g., Brackett & Katulak, 2006) as well as treatments that focus on teaching regulatory skills (Beck, 1976; Fairburn et al., 1998; Fairholme, Bosseus, Ellard, Ehrenreich, & Barlow, 2010; Hayes, Strosahl, & Wilson, 1999; Roemer, Orsillo, & Selarts-Pedneault, 2008). Specifically, if we can identify what strategies are more protective against, or stronger risk factors for, psychopathology, we can ensure that these strategies are targeted in prevention and intervention programs.

Another important question regards the relationship between cognitive regulatory strategies and psychopathology involves whether these strategies are more strongly related to certain disorders than to others. Leading theorists have argued that difficulties using cognitive emotion regulation strategies, including in rumination, thought suppression, reappraisal, and problem-solving, may be critical transdiagnostic factors underlying several forms of psychopathology (Ehring & Watkins, 2008; Fairburn, Cooper, & Shafran, 2003; Gross & John, 2003; Harvey et al., 2004; King & Sloan, 2010; Mansell, Harvey, Watkins, & Shafran, 2009; Moses & Barlow, 2006; Purdon, 1999; Rassin, Merckelbach, & Muris, 2000). Unfortunately, most studies on cognitive emotion regulation have been disorder-specific, limiting tests of transdiagnostic models. Identifying cognitive emotion regulation strategies that have transdiagnostic effects can inform the development of interventions targeting these strategies, thereby having preventative and treatment effects across a range of disorders.

In our meta-analysis, we found that the relationships between emotion regulation strategies were stronger for depression and anxiety than for eating and substance use disorders. These results suggest that, not surprisingly, mood and anxiety disorders might be more closely related to certain problems in cognitive emotion regulation than disorders in which mood disturbances are not as central (see Garnefski, Kraaij, & van Etten, 2005) and that eating disorders are more consistently related to cognitive emotion regulation strategies than substance use.

Our meta-analysis provided important clues as to the relative strength of different cognitive emotion regulation strategies in predicting psychopathology, and which types of psychopathology these strategies were most related to. There are significant limitations of the meta-analytic approach, however. Given the nature of a meta-analytic review, we could only examine the relationships between individual strategies and disorders independently of one another, and thus could not simultaneously model the relationships among all strategies and disorders (see Rosenthal & DiMatteo, 2001). This is problematic for several reasons. First, we could not test whether the any lack of specificity found in the relationship between strategies and psychopathology could be attributed to shared variance among the strategies. In other words, we could not test whether each strategy would still show significant associations with psychopathology when examined in models that included other strategies. Additionally, we could not examine whether the strategies would intercorrelate and potentially load onto latent factors.
factor of cognitive emotion regulation characterized by frequent use of maladaptive strategies and infrequent use of adaptive strategies.

Second, the nature of univariate effect sizes did not allow us to examine whether the relationships between regulatory strategies and specific disorders were reflecting transdiagnostic processes or the shared variance resulting from symptom overlap among disorders. In particular, we could not test whether the relationship between eating disorders and regulatory strategies would be maintained when accounting for the roles of depression or anxiety. That is, eating disorders might be related to regulatory strategies because of their associations with distress (e.g., Macht, Hatup, & Ellgring, 2005; Polivy & Herman, 2002). Similarly, we could not address the high comorbidity seen in depression and anxiety (Watson, 2009). In this respect, a better understanding of the phenomenology of processes uniquely associated with each of these disorders requires the isolation of the shared variance between them. Although the interpretation of the residual variance that results when a covariate has been included in a multivariate model is not without problems (see Miller & Chapman, 2001), it is still a parsimonious and straightforward way to identify which variables are more strongly associated with anxiety and which with depression (e.g., Aldao, Menning, Linardatos, & Fresco, 2010; Joormann & Stoeber, 1999; Menning, Heimberg, Turk, & Fresco, 2005).

In the present investigation, we sought to address these limitations by examining the relationships among reappraisal, problem-solving, thought suppression, and rumination, and their relationships to symptoms of depression, anxiety, and eating disorders in one large sample of young adults. Consistent with our previous results (Aldao, Nolen-Hoeksema, et al., 2010), we tested the hypothesis that all the cognitive emotion regulation strategies would show high intercorrelations and therefore fall on a latent factor of cognitive emotion regulation. Additionally, in line with the results of our meta analysis, we predicted that the maladaptive strategies would show stronger associations with the latent factor as well as with psychopathology. Lastly, we examined whether each of the three symptom types would continue to be associated with cognitive emotion regulation when interrelationships among symptom types was taken into account.

Methods

Participants

Participants were 252 undergraduate students at a private university in the northeastern United States who completed a battery of self-report questionnaires administered online using Survey Monkey. All of them received a $5 compensation (in the form of an Amazon.com gift certificate) and they were also entered into a lottery to win an iPod Touch (awarded to 1 in 30 participants). They provided online consent according to the guidelines specified by our Human Subjects Committee. The mean age of the sample was 18.44 (SD = .66) and a little over half the sample (55.6%) identified as female. In terms of ethnic background, 5.6% identified as African American, 22.2% as Asian/Asian American, 55.2% as Caucasian, 7.9% as Hispanic, 8% as Native American, and 8.3% as mixed or other.

Materials

Participants completed the following measures:

Emotion regulation measures. The Problem-Solving subscale of the COPE (Carver et al., 1989) is a 16-item measure of the tendency to take a problem-solving approach to stressful situations. It has four subscales: Active Coping, Planning, Suppression of Competing Activities, and Restraint Coping. Items are scored on a 4-point scale with higher scores indicating greater use of problem-solving to cope. The four subscales have good internal consistency in the literature (Carver et al., 1989; α’s .62–.80) and in this study (α’s .75–.91). We initially conducted analyses using the four separate scales; the results across the scales were very similar to the results using the total scale comprised of the average score across the four scales. Thus, for parsimony, we report here the analyses using the total problem-solving score (n = 90).

The Emotion Regulation Questionnaire (ERQ; Gross & John, 2003) is a 10-item measure that assesses individual differences in the dispositional use of two emotion regulation strategies: cognitive reappraisal and expressive suppression. Items are scored on a 7-point scale with higher scores indicating more use of a strategy. The internal consistency for the Reappraisal subscale in this sample was good (α = .82). We chose not to use the expressive suppression subscale and instead to use a measure of thought suppression (see below) because Gross and John (2003) have argued that expressive suppression is a “form of response modulation that involves inhibiting ongoing emotion-expressive behavior...that primarily modifies the behavioral aspect of the emotion response tendencies...and will not be helpful in reducing the experience of negative emotion, which is not directly targeted by suppression...” (Gross & John, 2003, p. 349). Indeed, in a line of experimental work, Gross and colleagues have shown that expressive suppression does not modify the subjective experience of emotion (Gross, 1998, Gross & John, 2003; Gross & Levenson, 1993; Richards & Gross, 2000) and that its effects consist of cardiac rebounds (i.e., increased sympathetic activation; Gross, 1998; Gross & John, 2003; Gross & Levenson, 1993) and cognitive difficulties (i.e., memory problems; Richards & Gross, 2000).4

The Ruminative Response Scale (RRS; Treynor, Gonzalez, & Nolen-Hoeksema, 2003) is a 22-item measure that assesses an individual’s tendency to engage in repetitive, ruminative behavior in response to distress. Items are score on a 4-point scale, with higher scores indicating more use of rumination. Treynor et al. (2003) have removed those items with a strong content overlap with depression and factor analyzed the remaining items to produce two subscales.

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4 We calculated correlations between ERQ Suppression and the regulatory strategies. It did not correlate with ERQ Reappraisal (r = .01, p = .91), which is in line with the CFA conducted by Gross and John (2003) in their initial validation of the ERQ and with subsequent investigations examining both subscales (e.g., Fresco et al., 2007; Magar, Phillips, & Hosie, 2008). In addition, it did not correlate with Problem-Solving Total (r = .06, p = .32), RS Brooding (r = .09, p = .17), or RS Pondering (r = .06, p = .35). However, it did correlate mildly with WBSI (r = .13, p = .05). This pattern of correlations suggests that ERQ suppression does not correlate with strategies aimed at modifying internal experience, with the exception of thought suppression, which might share a common inhibitory process. In addition, ERQ Suppression did not correlate with MASQ Anxious Arousal (r = .06, p = .38) or EDE total (r = .01, p = .89) and it correlated positively with MASQ Anhedonic Depression (r = .21, p < .01), thus suggesting a limited role in psychopathology symptoms in this normative sample, consistent with the arguments of Gross and John (2003) that expressive suppression will not have a strong relationship with the subjective experience of emotion.
The Pondering subscale reflects attempts to analyze problems leading to depression (e.g., “analyze recent events to try to understand why you are depressed;” “write down what you are thinking about and analyze it”). The Brooding subscale reflects the moody rumination at the core of Nolen-Hoeksema’s (1991) rumination theory (e.g., think, “What am I doing to deserve this?” “think about a recent situation, wishing it had gone better.”). The authors reported adequate reliability (α = .77 and α = .72 for Brooding and Pondering, respectively). In our sample the reliability was good (α = .81 and α = .83 for Brooding and Pondering, respectively).

The White Bear Suppression Inventory (WBSI; Wegner & Zanakos, 1994) is a 15-item measure designed to assess whether individuals have a tendency to suppress thoughts and experience thought intrusion. Items are scored on a 5-point scale, with higher scores indicating more thought suppression. The internal reliability for this measure in previous studies has been good (α’s ranging from .87 to .89; Wegner & Zanakos, 1994). In the current sample, reliability was also good (α = .94). We utilized this measure (instead of the ERQ Suppression) because of its focus on the regulation of internal experience.

**Psychopathology symptom measures.** The Mood and Anxiety Symptom Questionnaire – Short Form (MASQ-SF; Watson & Clark, 1999) is a 62-item measure that assesses symptoms that commonly occur in anxiety and mood disorders. Items are rated on a 5-point scale, with higher scores indicating higher levels of anxiety and depression. The short form version of the questionnaire consists of four subscales: General Distress: Anxious Symptoms (GDA) subscale, which consists of 11 items that reflect non-specific symptoms of anxiety (e.g., “was unable to relax”), General Distress: Depressive Symptoms (GDD) subscale, which consists of 12 items that reflect non-specific depression symptoms (e.g., “felt sluggish or tired”), Anxious Arousal (AA) subscale, which consists of 17 items that assess anxiety-specific symptoms (e.g., “startled easily”), and Anhedonic Depression (AD) subscale, which consists of 22 items that assess symptoms specific to depression (e.g., “felt withdrawn from other people”). In the present investigation, we only utilized the AA and AD subscales, since they have lower overlap and are therefore likely to produce stronger empirical differentiation between the highly overlapping constructs of anxiety and depression (this sample, for AA and AD, r = .29, p < .01, whereas for GDA and GDD, r = .70, p < .01.). Watson et al. (1995) reported high levels of internal consistency in multiple samples. In our sample, the α’s for AA and AD were .91 and .93, respectively.

The Eating Disorders Examination-Questionnaire (Fairburn & Beglin, 1995) is adapted from the Eating Disorder Examination (EDE; Fairburn & Cooper, 1993) and it assesses a broad range of symptoms of eating disorders. It contains probe items (i.e., yes/no format) and items on a 7-point scale assessing behavioral features of eating disorders, with higher scores indicating more psychopathology. The dimensional scores are utilized to calculate the EDE-Q subscales: Restrainment, Shape Concern, Weight Concern, and Eating Concern. These subscales have shown good internal reliability (α’s ranging for the subscales ranging from .78 to .93; Luce & Crowther, 1999). We utilized the total score of the EDE-Q (e.g., Roefs & Jansen, 2002) in order to capture overall eating pathology. This score had very good reliability in our sample (α = .95).

**Results**

**Univariate associations**

We first examined the univariate relationships among the measures of cognitive emotion regulation strategies and symptoms of psychopathology (see Table 1). The results suggest that: 1) not all strategies were correlated with one another; 2) adaptive strategies (i.e., reappraisal and problem-solving) showed the expected negative correlations with psychopathology, and maladaptive strategies (i.e., brooding, pondering, and suppression) showed the expected positive correlations; 3) consistent with our previous meta analysis, the adaptive strategies showed weaker relationships with psychopathology symptoms than the maladaptive strategies; 4) surprisingly, problem-solving showed no association with eating symptoms. In order to explore this pattern of results at a multivariate level, we analyzed the relationships between cognitive emotion regulation strategies and psychopathology using structural equation modeling (SEM; Arbuckle, 2007).

**Structural equation modeling**

We ran SEM models using Maximum Likelihood estimation. Although SEM parameters estimated with this method tend to be robust to deviations from multivariate normality, standard errors and fit indices might still be biased (Bollen, 1989; West, Finch, & Curran, 1995). Therefore, we ran our models utilizing bootstrapping, a procedure involving the drawing of multiple random samples by sampling from the original sample with replacement that results in more stable parameters, particularly with small samples and samples where multivariate normality may not old (Byrne, 2010; Nevitt & Hancock, 2001; Preacher & Hayes, 2008). We obtained 1000 random samples.

We evaluated model fit by examining several fit indices that are based on different information, thus producing a comprehensive evaluation of our models. We examined: 1) chi square, which should be non-significant (Hu & Bentler, 1998), but is overly sensitive to sample size, thus resulting in a tendency to reject the null hypothesis (Ulman, 2007); 2) chi square/df, which adjusts for sample size and should be lower than 3 or 2 (e.g., Mennin et al., 2007; Ulman, 2007); 3) Tucker Lewis Index (TLI; Tucker & Lewis, 1973), which should be close to .95 (Hu & Bentler, 1999); 4) Comparative Fix Index (CFI; Bentler, 1990), which should be close to .95 (Hu & Bentler, 1999); 5) Root Mean Square Error of Approximation (RMSEA), which should have values close to .06 (Hu & Bentler, 1999), with values between around .08 representing reasonable errors of approximation (Browne & Cudeck, 1993); and 6) Hoelter’s Critical N (Hoelter, 1983), which measures the adequacy of sample size, and should be higher than 200 (Hoelter, 1983).

**Measurement model**

We first tested a structural model with the cognitive emotion regulation strategies loading onto a single latent factor of cognitive emotion regulation. We did not correlate the variances between the two measures coming from a same measure (i.e., RSQ Brooding and Pondering; Brown, 2006) because this resulted in Heywood cases (i.e., estimates of negative variance that prevent the model from running properly; Brown, 2006; Byrne, 2010). Surprisingly, problem-solving did not load onto the latent factor of cognitive emotion regulation (p = .73) and the overall fit of the model was poor, as evidenced by a large, significant chi square (χ²(5, N = 252) = 40.14, p < .01) and poor fit indices (chi square/df = 8.03; TLI = .67; CFI = .84; RMSEA = .17; Hoelter’s Critical N = 95). Removal of problem-solving in a subsequent model resulted in a good fit, as evidenced by both a non-significant chi square (χ²(2, N = 252) = 5.91, p = .05) and appropriate values in the additional fit indices (chi square/df = 2.96: TLI = .94; CFI = .98; RMSEA = .09; Hoelter’s Critical N = 392). As Fig. 1 and Table 2 indicate, brooding, pondering, suppression and reappraisal loaded significantly on the latent factor of cognitive emotion regulation. Three strategies (i.e., brooding, pondering, and suppression) had
Table 1

Bivariate correlations between measures of emotion regulation and psychopathology.

<table>
<thead>
<tr>
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<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>Mean (SD)</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>ERQ Reappraisal</td>
<td>.27**</td>
<td>–.18**</td>
<td>.01</td>
<td>.05</td>
<td>-.32**</td>
<td>-.20**</td>
<td>-.15*</td>
<td>4.5 (1.05)</td>
<td>1–7</td>
</tr>
<tr>
<td>PS total</td>
<td>.02</td>
<td>.18**</td>
<td>.10</td>
<td>.25**</td>
<td>-.17**</td>
<td>0</td>
<td>2.62 (.53)</td>
<td>1–4</td>
<td></td>
</tr>
<tr>
<td>RSQ Brooding</td>
<td>.54**</td>
<td>.55**</td>
<td>.51**</td>
<td>.37**</td>
<td>.36**</td>
<td>2.14 (.73)</td>
<td>1–4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>RSQ Pondering</td>
<td>.39**</td>
<td>.24**</td>
<td>.16</td>
<td>.29**</td>
<td>.36**</td>
<td>2.21 (.79)</td>
<td>1–4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>WBSI</td>
<td>.35**</td>
<td>.31**</td>
<td>.25**</td>
<td>2.84 (.95)</td>
<td>1–5</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>MASQ Anhedonic Depression</td>
<td>.29**</td>
<td>.37**</td>
<td>.272 (.65)</td>
<td>1–3.47</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>MASQ Anxious Arousal</td>
<td>.27**</td>
<td>1.66 (.59)</td>
<td>1.10–4.67</td>
<td></td>
<td></td>
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<td></td>
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<tr>
<td>EDE total</td>
<td>1.34 (1.19)</td>
<td>0–5.48</td>
<td></td>
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</table>

Note: ERQ: Emotion Regulation Questionnaire; PS: problem-solving; RSQ: Response Styles Questionnaire; WBSI: White Bear Suppression Inventory; MASQ: Mood and Anxiety Symptoms Questionnaire; EDE: Eating Disorder Examination. *p < .05; **p < .01.

positive loadings, whereas reappraisal had a negative loading that was very low in magnitude, yet significant. Removal of this indicator or creation of a second latent factor with the indicators of reappraisal and problem-solving both resulted in Heywood cases and models that could not be run. Thus, we proceeded with the latent factor that included brooding, pondering, suppression, and reappraisal. We return to the statistical and conceptual implications of this issue in the discussion section.

Structural model

We examined a structural model, in which we evaluated the relationship between the latent factor of cognitive emotion regulation and symptom measures (i.e., MASQ AD, MASQ AA, and EDE-Q). Given the high overlap between symptoms of psychopathology (Watson, 2009), we allowed the MASQ AD, MASQ AA, and EDE-Q to correlate with each other.

The first version of the model produced a fair fit, with a significant chi square [X (11, N = 252) = 41.22, p < .01] and additional fit indices slightly outside of the ranges for good fit (chi square/df = 3.75, TLI = .85, CFI = .92, RMSEA = .11, Hoelter’s Critical N = 151). As a result, we examined the modification indices. Among the suggestions, one was the addition of a covariance path between the error variance or ERQ reappraisal and that of the latent factor of cognitive emotion regulation (M.I. = 10.02, parameter change = .08). Since this change would not result in the modification of the conceptual model, we decided to add this path in a second model.

The second structural model produced good fit. The chi square was still significant (10, N = 252) = 19.84, p < .05, but the rest of the indices were now within the ranges considered to be indicative of good fit (chi square/df = 1.98; TLI = .95; CFI = .97; RMSEA = .06, Hoelter’s Critical N = 294). In this model, as predicted, the symptom measures were significantly correlated with each other (r’s from .27 to .37; see Table 3, Fig. 2). In addition, all three symptom measures had significant regression weights onto the latent factor of cognitive emotion regulation, suggesting that even when the overlap between symptoms is taken into account, they are all correlated with cognitive emotion dysregulation (yet the coefficients were larger for depression symptoms, followed by anxiety symptoms and then by eating symptoms).

Discussion

A plethora of cognitive emotion regulation strategies has been conceptualized and operationalized in recent decades (e.g., Billings & Moos, 1981; Carver et al., 1989; Folkman & Lazarus, 1986; Garnefski & Kraaij, 2006; Gross, 1999; Nolen-Hoeksema et al., 2008; Wenzlaff & Wegner, 2000). These various strategies have generally been studied for their individual contributions to psychopathology; however, the studies that have compared several different strategies in the same sample have found that they tend to intercorrelate (e.g., Garnefski, Kraaij, & Spinbohein, 2001; Hong, 2007; Wenzlaff & Luxton, 2003), thus suggesting they might have low specificity.

In this study, we examined the relationships among four commonly studied cognitive emotion regulation strategies (i.e., reappraisal, problem-solving, rumination, and suppression) and found that three of them (reappraisal, suppression, and rumination, with its subcomponents of brooding and pondering) loaded on a single latent factor of cognitive emotion regulation. These results overall support a model of low specificity of regulatory strategies, with one exception: adaptive strategies appear to play a smaller role in cognitive emotion regulation than maladaptive strategies.

Reappraisal loaded on the cognitive emotion regulation latent factor, yet its loading was negative and extremely low. Its removal from the model substantially deteriorated it, generating Heywood cases and preventing it from running. Similarly, alternative models in which reappraisal and problem-solving loaded onto a different latent factor of adaptive cognitive emotion regulation also resulted in Heywood cases and extremely poor fit. This suggests that reappraisal might belong in this latent factor of cognitive emotion regulation, yet its role in the process might be weaker. For example, most individuals who tend to use maladaptive strategies such as rumination and thought suppression may try to use reappraisal to improve their emotional responses to stress, but their maladaptive tendencies may be stronger than their ability to use reappraisal successfully.

Although problem-solving was correlated with reappraisal and the pondering subcomponent of rumination, it did not load onto the latent cognitive emotion regulation factor in the structural model. This suggests that problem-solving might be fundamentally different from other regulatory strategies, as it is an attempt to change a situation rather than an emotion. The adaptiveness of problem-solving may also depend even more on the context than...
for other strategies. For example, problem-solving may not be an adaptive strategy when facing an uncontrollable situation in which there is no problem to solve (e.g., Cheng, Hui, & Lam, 1999; Folkman & Lazarus, 1986).

Moreover, the bivariate correlations showed that problem-solving was significantly correlated with depression and anxiety symptoms but not with eating symptoms (see Table 1). This finding was at odds with the results from our meta-analysis, which suggested a small-to-medium effect size between problem-solving and eating symptoms. A few explanations come to mind. First, the meta-analysis results were calculated using only two effect sizes (one from a clinical population and one from a normative population) so this might have resulted in a spurious finding. Second, problem-solving (and perhaps adaptive strategies at large) might have a different relationship with psychopathology symptoms.

The notion that adaptive strategies might play a smaller role in the cognitive emotion regulation process is consistent with the results from our meta-analysis, in which we found that suppression, rumination, and avoidance were more strongly associated with psychopathology than problem-solving, reappraisal, and acceptance (Aldao, Nolen-Hoeksema, et al., 2010). Adaptive strategies might have lower associations with psychopathology symptoms at the dispositional level because their adaptiveness might be more context-dependent than the maladaptiveness of maladaptive strategies. For example, reappraisal might only be adaptive when the situation can actually be reframed, whereas rumination is maladaptive most of the time (Nolen-Hoeksema et al., 2008). Indeed, recent work suggests that when reappraisal is deployed later in the emotion regulation process, it results in maladaptive outcomes, such as increased sympathetic activation (Sheppes, Catran, & Meiran, 2009) and diminished self-control (Sheppes & Meiran, 2008). In this respect, the temporal dynamics of the deployment of emotion regulation strategies might be critical to the adaptiveness of strategies (Davidson, 1998; Thompson, 1994). In addition, recent work suggests that the relationship between adaptive strategies and symptoms of anxiety and distress might be moderated by gender (Zlomke & Hahn, 2010). Future work on the contextual adaptiveness of strategies would benefit from the use of experience sampling designs (e.g., Ebner-Priemer et al., 2007; Feldman-Barrett & Baret, 2001), the manipulation of several contextual factors within one experimental session, and the use of functional analyses as a framework (e.g., Ferster, 1973; Kohlenberg & Tsai, 1991; Linehan, 1993).

Another possibility is that, once individuals start using maladaptive strategies frequently, these quickly become their default way of managing affective states. As a result, they start using the adaptive strategies more haphazardly, which would result in more noise and therefore weaker correlations with psychopathology symptoms. This notion is consistent with models of emotion regulation that stress flexibility in the use of strategies as a sign of adaptive regulation (e.g., Bonanno, Papa, O’Neill, Westphal, & Colifm, 2004; Colffman & Bonanno, 2010; Cole, Martin, & Dennis, 2004; Gratz & Roemer, 2004). Future work on this respect should focus on quantifying the ability to flexibly switch strategies as the contingencies in the environment change.

The latent cognitive emotion regulation factor did include both the brooding and pondering subcomponents of rumination. Many studies have confirmed that the brooding subcomponent is associated with a variety of psychopathological symptoms (see reviews by Aldao, Nolen-Hoeksema, et al., 2010; Nolen-Hoeksema et al., 2008). The relationships between the pondering subcomponent and psychopathology have been more mixed (see review by Nolen-Hoeksema et al., 2008). The items on this subcomponent seem to reflect attempts to understand and solve one’s problems (e.g., “analyze recent events to try to understand why you are depressed,” “write down what you are thinking about and analyze it”). In this study, the pondering subcomponent was positively correlated with problem-solving, but it was also positively correlated with brooding, suppression, and all the symptom types (for similar patterns in other studies, see review by Nolen-Hoeksema et al., 2008). Thus, although people may engage in pondering to try to understand and solve their problems, pondering is associated with a pattern of maladaptive emotion regulation strategies and psychopathology symptoms. Some individuals may try to engage in pondering about their

<table>
<thead>
<tr>
<th>Path</th>
<th>Parameter estimate</th>
<th>Standard error</th>
<th>Standardized estimate</th>
<th>p-Value</th>
<th>Bootstrap mean estimate bias</th>
<th>Bootstrap SE estimate bias</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cognitive emot reg → ERQ Reappraisal</td>
<td>–1.19</td>
<td>.27</td>
<td>–.53</td>
<td>&lt;.01</td>
<td>–.002</td>
<td>.004</td>
</tr>
<tr>
<td>Cognitive emot reg → RSQ Brooding</td>
<td>1.43</td>
<td>.17</td>
<td>.90</td>
<td>&lt;.01</td>
<td>.001</td>
<td>.001</td>
</tr>
<tr>
<td>Cognitive emot reg → RSQ Pondering</td>
<td>1.00</td>
<td>–</td>
<td>.59</td>
<td>–</td>
<td>.001</td>
<td>.002</td>
</tr>
<tr>
<td>Cognitive emot reg → WBSI total</td>
<td>1.28</td>
<td>.17</td>
<td>.62</td>
<td>&lt;.01</td>
<td>.000</td>
<td>.002</td>
</tr>
<tr>
<td>EDE total → cognitive emot reg</td>
<td>.06</td>
<td>.02</td>
<td>.15</td>
<td>&lt;.01</td>
<td>.000</td>
<td>.002</td>
</tr>
<tr>
<td>MASQ Anx Arousal → cognitive Emot Reg</td>
<td>.19</td>
<td>.05</td>
<td>.24</td>
<td>&lt;.01</td>
<td>.000</td>
<td>.002</td>
</tr>
<tr>
<td>MASQ Anhedonic Dep → cognitive emot reg</td>
<td>.32</td>
<td>.05</td>
<td>.44</td>
<td>&lt;.01</td>
<td>–.002</td>
<td>.002</td>
</tr>
</tbody>
</table>

Note: ERQ: Emotion Regulation Questionnaire; RSQ: Response Styles Questionnaire; WBSI: White Bear Suppression Inventory. There is no p-value for RSQ Pondering, since its loading was fixed to 1.
suggests that depressive symptoms had the largest coefficients from this preliminary observation, the consistency between cognitive regulatory strategies of depression and anxiety. To follow up some explanations for the potential stronger relationships to depression and anxiety than to eating disorders is in line with the results of our meta analysis showing that the brooding subscale was as strongly related to psychopathology in future studies in order to: reduce shared method variance, compensate for the biases resulting from self-report measures, and provide a more precise delineation of problems but fall into rumination and/or suppression. Interventions for such people may need to help them recognize when they are moving from pondering into rumination or suppression and then prevent this, perhaps through concreteness training (i.e., having individuals focus on the concrete details of a situation rather than on abstract evaluations of the situation). Watkins (2008) has shown that concreteness training can allow individuals to think about important self-relevant situations without falling into rumination.

The latent factor of cognitive emotion regulation, composed of rumination (brooding and pondering), suppression, and reappraisal was significantly related to all three types of psychopathology: depressive symptoms, anxiety symptoms, and eating disorders symptoms. An initial examination of the standardized coefficients suggests that depressive symptoms had the largest coefficients, followed by anxiety symptoms and then by eating symptoms. This is in line with the results of our meta analysis showing that the individual cognitive emotion regulation strategies had stronger relationships to depression and anxiety than to eating disorders (Aldao, Nolen-Hoeksema, et al., 2010) and with previous findings suggesting that internalizing symptoms are more strongly associated with emotion regulation than non-internalizing symptoms (e.g., Garnefski et al., 2005). Although premature to draw conclusions from this preliminary observation, the consistency between our previous findings and the trends in this study lead us to speculate on some explanations for the potential stronger relationships between cognitive regulatory strategies of depression and anxiety.

The larger coefficients for depression and anxiety might be the result of more overlap in item content between these measures than with eating disorder measures. The rumination scale has been criticized for its item overlap with depression (e.g., “I think about my feelings of sadness”). However, we used the version of the rumination questionnaire (i.e., the brooding and pondering subscales) from which Treynor et al. (2003) removed items that most obviously overlapped with distress and found they were still strongly related to depressive and anxiety symptoms. Similarly, our meta analysis found that the brooding subscale was as strongly related to depression and anxiety as was the full rumination scale (Aldao, Nolen-Hoeksema, et al., 2010).

An alternate explanation for the larger coefficients between cognitive emotion regulation measures and depression and anxiety symptoms is that eating disorder symptoms might themselves regulatory mechanisms utilized when experiencing high levels of anxiety and depression (Polivy & Herman, 2002). Evidence supporting this notion comes from findings showing that a history of negative affect is predictor of future onset or exacerbation of anorexic and bulimic symptoms (Burton, Stice, Bearman, & Rohde, 2007; Stice, Burton, & Shaw, 2004; Stice, Presnell, & Spangler, 2002). Further, Stice et al. (2002) have found that disordered eating behaviors in response to negative emotions more strongly predicted the development of bulimia nervosa in adolescent girls than did disordered eating behaviors that were in response to shape and weight concerns. Additionally, Macht et al. (2005) have found that students reported engaging in eating behaviors to distract themselves during periods of elevated stress.

Still, the results from this study indicate that eating disorder symptoms are significantly related to cognitive emotion regulation strategies, even after accounting for the relationships between eating disorder symptoms and depressive and anxiety symptoms. This suggests that cognitive emotion dysregulation has unique relationships to eating disorder symptoms, even if this relationship is smaller in magnitude than the one between cognitive emotion dysregulation and depressive and anxiety symptoms. At the same time, it does not completely invalidate the notion that eating disorders might be developed and/or maintain as strategies to regulate negative affect. Future studies using longitudinal designs should clarify the predictive relationships over time between cognitive emotion regulation strategies, eating disorder symptoms, and depressive and anxiety symptoms and directly compare the role of cognitive strategies across these disorders.

An important goal for future research is to identify the mechanisms by which the latent cognitive emotion regulation factor might lead to more symptoms of depression, anxiety, and eating disorders. Previous work on rumination, thought suppression, and reappraisal provide some clues as to the mechanisms by which each of these contributes to psychopathology (Gross & Thompson, 2007; Nolen-Hoeksema et al., 2008; Purdon, 1999; Watkins, 2008; Wenzlaff & Wegner, 2000). The confluence of tendencies to both ruminate and suppress thoughts, and difficulties in reappraisal, may have deleterious effects on the quality of thought and exacerbation of mood that go beyond the effects of any of these regulatory deficits individually, however. For example, people who ruminate and do not try to reappraise may experience particularly rapid escalation of catastrophizing thoughts and distressing emotions; if they are also prone to suppression, they may turn to food, substances, or self-destructive behavior to escape from their thoughts and moods. As a result, people with the confluence of regulatory problems indexed by our latent cognitive emotion regulation factor may be especially emotionally reactive to stressors and prone to escapist behaviors that may be self-destructive.

Limitations

A first limitation was the use of self-report measures for both psychopathology and emotion regulation, thus resulting in shared method variance. In addition, self-reports of psychopathology symptoms provide an incomplete picture of an individual's pathology and should therefore be administered as part of a multimethod assessment including diagnostic interviews (e.g., Achenbach, Krukowski, Dumenci, & Ivanova, 2005; Meyer et al., 2001). To this end, structured clinical interviews could be used to assess psychopathology in future studies in order to: reduce shared method variance, compensate for the biases resulting from self-report measures, and provide a more precise delineation of
individual disorders (in this paper, we focused on general symptoms of depression, anxiety, and eating disorders). Our meta-analysis found stronger relationships between several emotion regulation measures and psychopathology in clinically diagnosed samples than in general population samples (Aldao, Nolen-Hoeksema, et al., 2010), underscoring the importance of assessing psychopathology with a multi-method approach.

Additionally, assessing emotion regulation strategies by self-report is problematic for several reasons, including the blurry lines between emotion generation and emotion regulation (Campos, Frankel, & Camras, 2004; Cole et al., 2004) and reporting biases (Robinson & Clore, 2002). In this respect, a comprehensive study of regulatory strategies will require a multi-modal method of assessment capturing the subjective, physiological, and behavioral emotional domains utilizing multiple measures of each domain. Future work examining the relationship between cognitive emotion regulation strategies and psychopathology as assessed by structured diagnostic interviews is needed to confirm the results here.

A second limitation of the present investigation is that we limited our scope to four cognitive regulatory strategies. We chose these strategies because they are among the most commonly studied ones in the field and therefore the most representative of current conceptualizations of emotion regulation. However, these strategies have been largely studied within the context of mood and anxiety disorders and might not be as relevant to the main affective processes to be regulated in other disorders (e.g., positive emotions resulting from the consumption of foods and other substances; elevated positive affect in mania). Future trans-diagnostic work on regulatory strategies should take this issue into account and seek to identify process that might be germane to diagnostic work on regulatory strategies should take this issue into account and seek to identify process that might be germane to regulatory strategies. Clinically, this might help account for the differences in the relationship between strategies aimed at the modification of subjective versus expressive emotional processes.

A third limitation of this study is its cross-sectional design, which did not allow for modeling of relationships among variables over time. Lastly, a fourth limitation stems from the nature of the population we studied, as we utilized an undergraduate sample, therefore restricting the range of psychopathology and potentially also that of cognitive emotion regulation strategies. Indeed, the models presented in this manuscript had good fit, but it could have certainly been higher. It therefore becomes critical to replicate the structural findings in a wide range of samples varying in severity of the symptoms and pervasiveness of the emotion regulation deficits.

Conclusion

We found mixed evidence for specificity in the relationship between multiple cognitive emotion regulation strategies and psychopathologies. The adaptive strategy of reappraisal showed a weaker association with a latent factor of cognitive emotion regulation and with psychopathology symptoms than maladaptive strategies. Clinically, this might help account for the difficulties teaching clients to incorporate adaptive strategies to their repertoire; the maladaptive strategies of rumination and thought suppression may often overwhelm clients’ attempts to reappraise. This suggests that interventions that directly focus on reducing rumination and thought suppression, such as teaching mindfulness meditation practices, may be critical precursors to interventions to teach clients to use reappraisal more successfully (e.g., Segal, Williams, & Teasdale, 2002; Watkins et al., 2007).

A latent factor of cognitive emotion regulation was significantly associated with symptoms of all three disorders, suggesting it has transdiagnostic effects. These results encourage the development of prevention and intervention programs focused on cognitive emotion regulation strategies, because such programs may have positive effects across a range of disorders.

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References


