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CITATION
When Are Adaptive Strategies Most Predictive of Psychopathology?

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In recent work, we showed that putatively adaptive emotion regulation strategies, such as reappraisal and acceptance, have a weaker association with psychopathology than putatively maladaptive strategies, such as rumination, suppression, and avoidance (e.g., Aldao & Nolen-Hoeksema, 2010; Aldao, Nolen-Hoeksema, & Schweizer, 2010). In this investigation, we examined the interaction between adaptive and maladaptive emotion regulation strategies in the prediction of psychopathology symptoms (depression, anxiety, and alcohol problems) concurrently and prospectively. We assessed trait emotion regulation and psychopathology symptoms in a sample of community residents at Time 1 ($N = 1,317$) and then reassessed psychopathology at Time 2 ($N = 1,132$). Cross-sectionally, we found that the relationship between adaptive strategies and psychopathology symptoms was moderated by levels of maladaptive strategies: adaptive strategies had a negative association with psychopathology symptoms only at high levels of maladaptive strategies. In contrast, adaptive strategies showed no prospective relationship to psychopathology symptoms either alone or in interaction with maladaptive strategies. We discuss the implications of this investigation for future work on the contextual factors surrounding the deployment of emotion regulation strategies.

Keywords: emotion regulation, adaptive regulation strategies, maladaptive regulation strategies, flexibility, longitudinal

In the last decade, researchers have been increasingly interested in examining the process by which individuals regulate emotional states to respond to environmental demands (e.g., Gross, 1998; Kring & Sloan, 2010; Rottenberg & Gross, 2003). A substantial amount of theoretical and empirical work has focused on the processes by which emotion regulation strategies modify emotional experiences and their associated expressions, behavioral components, and physiological states, thereby increasing or decreasing the risk of psychopathology (e.g., Campbell-Sills, Barlow, Brown, & Hofmann, 2006; Gross, 1998; Hofmann, Heering, Sawyer, & Asnaani, 2009).

The work on emotion regulation strategies has led to a functional differentiation of strategies based on their ability to facilitate adaptive versus maladaptive responding. Specifically, strategies such as cognitive reappraisal, acceptance, and problem solving generally have been associated with adaptive outcomes, including reductions in the experience of negative affect (e.g., Goldin, McRae, Ramel, & Gross, 2007), increased pain tolerance (e.g., Hayes et al., 1999), effective interpersonal functioning (e.g., Richards & Gross, 2000), and diminished maladaptive cardiac reactivity (e.g., Campbell-Sills et al., 2006). In addition, these putatively adaptive strategies are important components of a variety of treatment modalities, ranging from traditional cognitive–behavioral therapy to newer, third-wave approaches (e.g., Beck, 1976; Hayes, 2008; Hofmann & Asmundson, 2008; Roemer, Orsillo, & Salter-Pedneault, 2008).

On the other hand, strategies like rumination, suppression, avoidance, and worry generally have been associated with maladaptive outcomes, including rebounds in negative affect following engagement with emotion-eliciting stimuli (e.g., Campbell-Sills et al., 2006), memory difficulties (e.g., Richards, Butler, & Gross, 2003), increases in sympathetic activation (e.g., Gross & Levenson, 1993; Wegner, Broome, & Blumberg, 1997), and diminished autonomic flexibility (e.g., Hofmann et al., 2005). Furthermore, the self-reported use of these strategies has been associated with the development and maintenance of a wide range of disorders, including depression (Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008), generalized anxiety disorder (Mennin, Holaway, Fresco, Moore, & Heimberg, 2007), alcohol problems (Nolen-Hoeksema & Harrell, 2002), and eating disorders (Nolen-Hoeksema, Stice, Wade, & Bohon, 2007).

Recently, we showed that putatively adaptive and maladaptive strategies differ in their strength of association with psychopathology. Specifically, we observed that maladaptive strategies are consistently more strongly associated with psychopathology than adaptive strategies (e.g., Aldao & Nolen-Hoeksema, 2010; Aldao, Nolen-Hoeksema, & Schweizer, 2010). It is interesting to note that we have seen this pattern of findings when examining both internalizing (e.g., depression, anxiety) and externalizing (e.g., alcohol, eating) pathology in community samples and samples including clinical and nonclinical groups, suggesting that this asymmetry in the strength of the relationship between adaptive and maladaptive strategies and psychopathology generalizes across the range of psychopathology (e.g., Harvey, Watkins, Mansell, & Shafran, 2004; Kring & Sloan, 2010). Understanding why adaptive strate-
gies are relatively weak predictors of psychopathology is important given the centrality of such strategies to treatment and prevention efforts (e.g., Beck, 1976; Hayes, 2008; Hofmann & Asmundson, 2008; Roemer et al., 2008). In particular, understanding when adaptive strategies are more or less useful would be critical to teaching clients how to use these strategies effectively.

Using adaptive strategies may be more difficult for people who are also prone to using maladaptive strategies, because maladaptive strategies may interfere with the use of adaptive strategies. Indirect evidence for this interference hypothesis comes from work showing that maladaptive strategies may narrow attentional focus, therefore preventing individuals from noticing the context around them (e.g., Borkovec, Alcaine, & Behar, 2004; Richards et al., 2003; Richards & Gross, 2000; Wenzlaff & Wegner, 2000). For example, Joormann and colleagues (see Joormann & D'Avanzo, 2010) have found that rumination interferes with the ability to shift attention away from negative stimuli. Therefore, to the extent that maladaptive strategies narrow attentional focus, they might interfere with individuals’ ability to access and properly deploy adaptive regulation strategies.

On the other hand, a compensatory hypothesis suggests that adaptive strategies may be most important in preventing psychopathology in people who also tend to use maladaptive strategies. This hypothesis is consistent with models suggesting that people have a repertoire of emotion regulation strategies that they use flexibly depending on circumstances (e.g., Bonanno, Papa, O’Neill, Westphal, & Coifman, 2004; Cheng, 2003; Kashdan & Rottenberg, in press; Westphal, Seivert, & Bonanno, 2010). Among people whose repertoire includes maladaptive strategies, adaptive strategies may be particularly predictable of psychopathology outcomes.

In this investigation, we sought to further investigate the asymmetry in the strength of the relationship between adaptive and maladaptive strategies and psychopathology. Specifically, we tested whether trait maladaptive strategies moderated the relationship between trait adaptive strategies and symptoms of psychopathology. Based on previous work (e.g., Aldao & Nolen-Hoeksema, 2010; Aldao et al., 2010), we predicted that maladaptive strategies would be a stronger predictor of psychopathology than adaptive strategies. In addition, we predicted that adaptive and maladaptive strategies would interact with each other. Two competing possibilities were examined. First, an interference hypothesis would suggest that adaptive strategies would have a weaker negative relationship with psychopathology when levels of maladaptive strategies are high than when they are low. On the other hand, a compensatory hypothesis would suggest that adaptive strategies would have a stronger negative relationship with psychopathology when levels of maladaptive strategies are high than when they are low.

In addition to examining this relationship concurrently, we evaluated the interplay between adaptive and maladaptive strategies and changes in psychopathology symptoms over time. Previous work has shown that the use of maladaptive strategies prospectively predicts the development and maintenance of psychopathology after controlling for baseline levels of psychopathology (for a review, see Aldao et al., 2010). However, little is know about how adaptive and maladaptive strategies interact to predict changes in psychopathology over time. Therefore, in the present investigation we also examined how the interaction between adaptive and maladaptive strategies at baseline (i.e., Time 1) predicted psychopathology symptoms one year later (i.e., Time 2), while controlling for baseline levels of psychopathology.

Method

Participants and Procedures

Participants were recruited through random-digit dialing of residential telephone numbers in San Francisco, San Jose, and Oakland, California; 1,789 individuals were initially called. Of those, 1,317 participated in the first interview, and 1,132 of the original participants responded to a second interview 1 year later. Individuals who did not participate in the second interview scored higher in psychopathology than those who did, t(1316) = 3.05, p < .01.

At Time 1, the mean age of the sample was 47 years old (SD = 15.22; range = 24–82), and 52.7% of the sample identified as female. Seventy percent of the sample was Caucasian, 9.2% Hispanic, 8% African American, 6.3% Asian or Pacific Islander, and 6.5% identified as other or did not respond (for other demographic information, see Nolen-Hoeksema, 2000).

At both Time 1 and Time 2, all participants were interviewed in person. All measures were read aloud to participants and response options were presented on cards. Interviewers were extensively trained in standardized responses to participants’ requests for clarification of items and probes to clarify participants’ answers to questions when necessary.

Measures

Depression was assessed with the 13-item Beck Depression Inventory–Short Form (BDI-SF; Beck & Beck, 1972). At Time 1, internal consistency was good (α = .83), and the test–retest correlation was .60 (p < .01). In addition, the module for major depression from the Structured Clinical Interview for the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (SCID; First, Spitzer, Gibbon, & Williams, 1997) was administered. Interrater reliability on this module was 100% (see Nolen-Hoeksema, 2000). We created a continuous measure of depression by summing the number of symptoms recorded for each respondent. Internal reliability at Time 1 was .68, and the test–retest reliability was .48 (p < .01).

Anxiety was assessed with the 21-item Beck Anxiety Inventory (BAI; Beck & Steer, 1990). Internal reliability at Time 1 was good (α = .88), and the test–retest correlation was .63 (p < .01). We did not administer the SCID modules on anxiety for this study because it was designed as a study of depression, but interviewers completed a one-item global rating of the anxiety levels of the respondents immediately after the interview on a scale ranging from 0 (none) to 4 (disabling), using information reported by the respondents on the BAI, as well as information provided by the respondents’ verbal and nonverbal behavior throughout the interview. Test–retest reliability was .32 (p < .01).

In the results section, we present analyses conducted with the full sample. Analyses conducted with the smaller set of individuals who participated in both assessments paralleled the results obtained with the full sample.
Alcohol-related problems were assessed with the relevant module of the SCID (First et al., 1997). We summed the number of problems recorded to create a continuous score from 0 to 8 (see Nolen-Hoeksema & Harrell, 2002). The internal consistency was good (α = .77). The test–retest reliability was .57 (p < .01).

Recent work shows that emotion regulation strategies are associated with all three types of psychopathology we assessed (e.g., Aldao & Nolen-Hoeksema, 2010; Kring & Sloan, 2010). Furthermore, measures of the three symptom types were intercorrelated (all ps < .01). Thus, we standardized scores on each of the measures (BDI, SCID depressive symptoms, BAI, anxiety rating, SCID alcohol use problems) and averaged them for an overall index of psychopathology. We note, however, that a similar pattern of results is found whether we use this aggregated index of psychopathology or separate indices of internalizing symptoms (depression and anxiety) or alcohol problems. For parsimony, we report only the analyses of the overall psychopathology index. The test–retest reliability for this index was .67 (p < .01).

The Ruminative Responses Scale (RRS; Treynor, Gonzalez, & Nolen-Hoeksema, 2003) is a 22-item measure that assesses the tendency to engage in ruminative behavior in response to stress. Treynor and colleagues (2003) have removed those items with a high content overlap with depressive symptoms. The resulting brooding subscale contains 5 items and reflects the moody rumination at the core of Nolen-Hoeksema’s (1991) rumination theory (e.g., “What am I doing to deserve this”). In this sample, internal reliability of the brooding subscale was good (α = .77).

The COPE Inventory—Short Version (Carver, 1997) is a shorter version of the original COPE inventory (Carver, Scheier, & Weintraub, 1989) that was adopted for this study because respondent burden was already high. We utilized the following scales: acceptance, positive reframing, behavioral disengagement, and denial. In the initial investigation (Carver, 1997), the internal consistency for these scales ranged from .57 to .64 and in the current investigation the range was from .53 to .70. In addition, because at the time this study was conducted (mid-1990s), there was no widely used measure of emotional, thought, and expressive suppression, we created a 4-item subscale assessing all aspects of suppression (i.e., expressive, thought, and emotional). These items had good internal consistency (α = .76).

To model the relationship between adaptive and maladaptive strategies, we created two composite scores reflecting the use of each type of strategy. The composite score of adaptive strategies consisted of the average between COPE acceptance and COPE positive reframing and the composite score of maladaptive strategies consisted of the average of COPE behavioral disengagement, COPE denial, suppression, and RRS brooding (they were all on the same scale). We submitted these subscales to a factor analysis and showed that the maladaptive scales loaded onto one factor (34% of the variance), and the adaptive scales loaded onto a separate factor (23% of the variance). The internal consistency was adequate for adaptive strategies (α = .68) and good for maladaptive strategies (α = .81). The scores were mildly correlated with each other (r = .13, p < .01), likely because individuals who experience elevated levels of distress tend to use many types of coping or emotion regulation in their attempts to regulate their negative affect (e.g., Nolen-Hoeksema et al., 2008).

### Results

#### Cross-Sectional Analyses

**Zero-order correlations.** We examined zero-order correlations at Time 1 for the full sample. Consistent with previous work, the psychopathology composite score was positively correlated with the maladaptive strategies composite score (r = .50, p < .01) and was not correlated with the adaptive strategies composite score (r = .03, ns).

**Regression analyses.** We predicted the psychopathology composite score using a stepwise regression. In the first step, we entered adaptive and maladaptive strategies scores (which were centered to reduce multicollinearity; Tabachnick & Fidell, 2007), ΔR² = .25, F(2, 1314) = 217.06, p < .01. Maladaptive strategies contributed significantly to the prediction of psychopathology, t(1314) = 20.81, p < .01, β = .50. Adaptive strategies were not a significant predictor, t(1314) = −1.58, ns, β = −.04. In a second step, we entered the interaction term between adaptive and maladaptive strategies, ΔR² = .004, F(1, 1313) = 7.34, p < .01. Maladaptive strategies remained a significant predictor, t(1313) = 20.88, p < .01, β = .50, and adaptive strategies became a significant predictor, t(1313) = −2.05, p < .05, β = −.056. More important, the interaction term between adaptive and maladaptive strategies was significant, t(1313) = −2.71, p < .01, β = −.07.

We followed this interaction with simple slopes analyses (Aiken & West, 1991). Both the interference hypothesis and the compensatory hypothesis entail predictions (in opposite directions) about the relationship between adaptive strategies to psychopathology in the presence of high versus low levels of maladaptive strategies. Thus, we evaluated the role of adaptive strategies as a predictor of psychopathology at high and low levels of maladaptive strategies (i.e., 1 ± SD above/below the mean). At low levels of maladaptive strategies, adaptive strategies were unrelated to psychopathology, t(1313) = .41, ns, β = .01. However, at high levels of maladaptive strategies, greater levels of adaptive strategies were related to lower levels of psychopathology, t(1313) = −3.08, p < .01, β = −.11.

#### Longitudinal Analyses

**Correlations.** We examined the relationship between strategies at Time 1 and psychopathology symptoms at Time 2. In parallel with the cross-sectional results, maladaptive strategies at Time 1 were correlated with the composite score of psychopathology at Time 2 (r = .41, p < .01), even after controlling for symptoms at Time 1 (r = .12, p < .01). In addition, mimicking the cross-sectional results, adaptive strategies at Time 1 were not correlated with the psychopathology at Time 2 (r = .03).

**Regression analyses.** We predicted psychopathology symptoms at Time 2 with adaptive and maladaptive strategies at Time 1. In Step 1, we entered psychopathology symptoms at Time 1 and, as expected, we found that it was a significant predictor, t(1124) = 29.87, p < .01, β = .67, ΔR² = .44. In Step 2, we entered adaptive and maladaptive strategies at Time 1 centered, ΔR² = .01, F(2, 1122) = 8.13, p < .01. Psychopathology symptoms at Time 1

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2 Available from the first author.
remained a significant predictor, $t(1122) = 24.11$, $p < .01$, $\beta = .62$, and the maladaptive strategies score was also a significant predictor, $t(1122) = 3.97$, $p < .01$, $\beta = .10$, but adaptive strategies was not a significant predictor, $t(1122) = .02$, $ns$, $\beta = .00$. In step 3, we added the interaction between adaptive and maladaptive strategies at Time 1, but, unlike in the cross-sectional models, it was not a significant predictor, $t(1121) = -.57$, $ns$, $\beta = -.01$.

Discussion

We examined the relationship between trait adaptive (positive reframing and acceptance) and maladaptive (rumination, suppression, behavioral disengagement, and denial) emotion regulation strategies in the prediction of psychopathology symptoms concurrently and prospectively. At the cross-sectional level, we found that maladaptive strategies moderated the relationship between adaptive strategies and psychopathology symptoms: adaptive strategies had a negative association with psychopathology symptoms only when levels of maladaptive strategies were elevated. This provides support for the compensatory hypothesis that adaptive strategies have significant relationships to psychopathology symptoms in the presence of maladaptive strategies. On the other hand, in prospective analyses, only maladaptive strategies predicted psychopathology symptoms at Time 2.

In previous work (Aldao & Nolen-Hoeksema, 2010), we argued that adaptive strategies might show a weaker association with psychopathology because their usefulness might be context dependent (e.g., reappraisal might be mostly adaptive when the situation can actually be reframed, or acceptance might be most appropriate when there is little action an individual can take to change his or her circumstances). One type of context may be the affective and/or regulatory state of the individual. Sheppes, Cartan, and Meiran (2009) have shown that the effects of reappraisal depend on the point in time in the emotion process in which it is deployed. Similarly, in this investigation we found that adaptive strategies showed a negative association with concurrent levels of psychopathology only when levels of maladaptive strategies were elevated, therefore suggesting that their effectiveness might depend on the other regulation strategies present in an individual’s repertoire. Future empirical work should examine the process by which individuals select one strategy over another one (e.g., Egloff, Schmukle, Burns, & Schwerdtfeger, 2006) and can flexibly switch between using different strategies that might be differentially adaptive for varying contexts (e.g., Westphal et al., 2010). In addition, it will be important to examine other contextual factors, such as trait (e.g., emotion intensity, personality) and state (e.g., emotion reactivity, attention processes, physiology) components of affective processes.

Although this study provided an important examination of the moderating role of maladaptive strategies on adaptive strategies, it had limitations. The first was our use of self-report measures of psychopathology, which are subject to reporting bias (e.g., Robinson & Clore, 2002). We attempted to reduce the influence of such biases by administering these self-report measures in the context of an interview and combining them with indices of psychopathology that came from structured interviews and interviewer ratings. Still, replicating our findings with measures of psychopathology that are all based on structured clinical interviews will be important.

Second, our sample was a representative community sample, raising the question of whether our results would be replicated in comparisons of individuals with and without diagnosed disorders. In our previous meta analytic review (Aldao et al., 2010), we showed that maladaptive strategies were even more strongly related to psychopathology when clinical and nonclinical samples were compared than when community samples with a range of psychopathology were used; in contrast, adaptive strategies were weakly and inconsistently related to psychopathology in both community samples and comparisons of clinical and nonclinical samples. Thus, our meta analysis suggests that we would find similar results in comparisons of clinical and nonclinical samples. Furthermore, emotion regulation theories suggest that the relationship between specific strategies and emotional distress or unhealthy behaviors (such as substance misuse) can be characterized as continuous (for reviews, see Kring & Sloan, 2010); thus, the use of a community sample with a representative range of psychopathology provide an appropriate test of these theories. Still, the generalizability of our results to comparisons of clinical versus nonclinical samples remains to be tested.

Third, emotion regulation was measured through self-report questionnaires, although they were administered in the context of a structured interview. Future studies should utilize experimental designs to assess the multiple emotional domains (e.g., subjective, physiological, and behavioral; Bradley & Lang, 2000). Furthermore, we only examined 6 regulation strategies, so it will be important for future work to incorporate a wider range of strategies (e.g., problem solving, seeking social support).

Fourth, an alternative hypothesis for the stronger association between maladaptive strategies and psychopathology is that the scales used to measure maladaptive strategies have content overlap with psychopathology scales. In this investigation, we sought to minimize this potential confound by utilizing the brooding scale of the RRS (Treynor et al., 2003), from which the items that most obviously overlapped with distress were removed. In addition, the COPE scales we used had minimal overlap with symptoms of negative affect.

In this investigation, we examined the relationship between adaptive and maladaptive emotion regulation strategies in the prediction of psychopathology symptoms concurrently and prospectively. When predicting psychopathology symptoms cross-sectionally, we found support for a context-dependent role of

Figure 1. Cross-sectional interaction between adaptive and maladaptive strategies. High and low levels correspond to 1 SD above or below the mean, respectively. Adaptive strategies have a negative association with psychopathology only at high levels of maladaptive strategies.
adaptive strategies, so that their association with psychopathology symptoms was moderated by levels of maladaptive strategies. Specifically, adaptive strategies had a negative association with psychopathology symptoms only at high levels of maladaptive strategies, providing support for a compensatory hypothesis. In contrast, adaptive strategies did not show an association with psychopathology symptoms longitudinally, suggesting their predictive power might be limited.

References


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