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Patterns of emotion regulation and psychopathology

Afsoon Eftekhari[*], **Lori A. Zoellner**, and **Shree A. Vigil**
Department of Psychology, University of Washington, Seattle, WA, USA

Abstract

Emotion regulatory strategies such as higher expressive suppression and lower cognitive reappraisal may be associated with increased psychopathology (Gross & John, 2003). Yet, it is unclear whether these strategies represent distinct cognitive styles associated with psychopathology, such that there are individuals who are predominantly "suppressors" or "reappraisers." Using cluster analysis, we examined whether women with and without exposure to potentially traumatic events evidence distinct patterns of emotion regulation frequency, capacity, suppression, and cognitive reappraisal. Four patterns emerged: high regulators; high reappraisers/low suppressors; moderate reappraisers/low suppressors; and low regulators. Individuals who reported infrequently and ineffectively regulating their emotions (low regulators) also reported higher depression, anxiety, and posttraumatic stress disorder (PTSD). In contrast, individuals who reported frequently and effectively using reappraisal and low levels of suppression (high reappraisers/low suppressors) reported the lowest levels of these symptoms, suggesting that this specific combination of emotion regulation may be most adaptive. Our findings highlight that the capacity to regulate emotions and the ability to flexibly apply different strategies based on the context and timing may be associated with reduced psychopathology and more adaptive functioning.

Keywords

emotion regulation; suppression; reappraisal; PTSD; psychopathology

Traumatic events often result in significant anxiety and emotional distress that persist long past the trauma. The prevalence of trauma exposure in the USA is surprisingly high (Solomon & Davidson, 1997), with a lifetime prevalence of posttraumatic stress disorder (PTSD) estimated at 24% among trauma survivors and at 9% in just the general population (Breslau, Davis, Andreski, & Peterson, 1991). Interestingly, although trauma exposure is high, not everyone develops chronic psychopathology (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995), highlighting the need to better understand factors that are predictive of developing psychopathology after exposure to traumatic events. Emotion regulation may be one such factor.

Emotion regulation is the unconscious or conscious (automatic or controlled) process used to increase, maintain, or decrease one or more components of an emotional response (Gross, 1999). These components include feelings, behaviors, and physiological responses that make up an emotion. Accordingly, strategies to regulate emotions can be viewed as adaptive or maladaptive responses depending on their function, context, and timing (e.g., Bonanno, 2004; Bonanno, Papa, Lalande, Westphal, & Coifman, 2004; Gross, 2001). When used in a maladaptive manner, emotion regulatory processes can be both emotionally and cognitively

[*]Corresponding author. aeftekha@u.washington.edu.

demanding, potentially resulting in or exacerbating negative mental health outcomes, such as depression, anxiety, and other symptoms of psychopathology (e.g., Eysenck, 2000; Gross & John, 2003; Gross & Muñoz, 1995; Price, Monson, Callahan, & Rodriguez, 2006; Turk, Heimberg, Luterek, Mennin, & Fresco, 2005). Therefore, it is important to better understand how people differ in the emotion regulation processes they utilize and how these individual differences are related to psychopathology.

Theories regarding emotion regulation have evolved from the psychoanalytic and stress and coping traditions and span across many subdisciplines of psychology, including developmental, personality, and social psychology (e.g., Campos, Campos, & Barrett, 1989; Derakshan & Eysneck, 1997; Ekman, 1984; Eysenck, 1992, 1997, 2000; Frijda, 1993; Gross, 1998; Lazarus, 1966, 1984, 1991; Lazarus & Folkman, 1984). Many of these theories highlight the interchange among cognitive processes and emotions in regard to how one copes with his or her environment (e.g., Eysenck, 1992, 1997, 2000; Frijda, 1993; Lazarus, 1966, 1984, 1991; Lazarus & Folkman, 1984; Lewis, 1996). For example, similar to classic models of appraisal and coping presented by Lazarus (1966, 1984, 1991), Frijda (1993) presents a model that views appraisal as both an antecedent and consequence of everyday emotional experience that can potentially serve an adaptive function in regulating affect. Building on this, Lewis (1996) proposes a model where there is feedback between cognitive and emotional elements, yielding appraisals that neither precede nor follow emotions. Rather, these cognitive and emotional elements arise together with emotion in the process of self-organization (Lewis, 1996). Both of these models examine the interchange between cognitive processes (i.e., appraisal) and emotions in the regulation of affect, which can in turn potentially impact psychological well-being. More specific to emotion regulation processes in psychopathology, Eysenck (1992, 1997, 2000) proposes a cognitive model of trait anxiety and its regulation, suggesting that individuals with a repressive coping style tend to regulate negative emotion by interpreting environmental stimuli, physiologic responses, behavior, and cognitions in a manner that minimizes threat. In doing so, they are able to cope with stressful experiences. While repression can be adaptive in regulating the experience of anxiety, it can also bear negative consequences because this coping style is also associated with high levels of defensiveness and cognitive biases (see Eysenck, 2000 for review).

In line with this, Gross presents a process model of emotion regulation (Gross, 1998) that was developed from the work of several key emotion theorists such as Ekman (1972), James (1884, 1894), Levenson (1994), and Plutchik (1990), and focuses on the role of appraisal of both external and internal events that then result in emotions (Gross, 1998; Gross & Muñoz, 1995). This model builds on previous work by further differentiating various aspects of the emotion regulation process such as an individual's capacity to regulate emotions, that is, one's ability to tolerate and experience emotions, the frequency at which emotion regulation processes occur, and how the timing of emotion regulatory strategies impact the effectiveness of the strategy. Specifically, Gross proposes that emotions begin when emotion cues (e.g., situations, places) trigger a set of behavioral, experiential, and physiological response tendencies (e.g., Gross, 2001). Once these tendencies are triggered, they may be modulated at various points in the emotion-generative process. Emotion regulatory strategies used early in the process termed “antecedent-focused” emotion regulation (i.e., reframing the situation) are generally viewed as adaptive; whereas strategies used later in the process termed “response-focused” emotion regulation (i.e., concealing or suppressing feelings) are often viewed as maladaptive (Gross, 1998; Gross & Muñoz, 1995).

Among various emotion regulatory strategies, expressive suppression and cognitive reappraisal are of particular interest due to their physiological, cognitive, and social consequences (e.g., Gross, 1998, 2001) and their applicability to the extant trauma exposure

and PTSD literatures. Specifically, expressive suppression occurs during response modulation after an emotion has been elicited and is aimed at actively inhibiting emotional responses. Because expressive suppression occurs later in the emotion-generative process, it often generates negative consequences (e.g., Clohessy & Ehlers, 1999; Gross & John, 2003; Richards, 2004; Richards, Butler, & Gross, 2003). Gross suggests that expressive suppression taxes cognitive resources due to the continual self-monitoring and modulation of emotional response tendencies that are activated. Indeed, the use of expressive suppression results in poor recall of information (e.g., Bonanno et al., 2004; Richards & Gross, 1999), increased physiological arousal (Gross & Levenson, 1993, 1997), and increased levels of anxiety and negative affect (Gross & John, 2003). Furthermore, the use of similar expressive suppression strategies has been linked to increased frequency of intrusive memories of potentially traumatic or negative events (Roemer & Borkovec, 1994; Shipherd & Beck, 1999) and the suppression of past traumatic events has been associated with increased illness and distress (e.g., Pennebaker, Kiecolt-Glaser, & Glaser, 1988). Within the PTSD literature, suppression may prevent the emotional processing of the traumatic event, resulting in the maintenance of PTSD (Clohessy & Ehlers, 1999). Furthermore, two variants of suppression, dissociation and emotional numbing, are implicated in the prediction of chronic PTSD (Clohessy & Ehlers, 1999; Foa, Riggs, & Gershuny, 1995; Ozer, Best, Lipsey, & Weiss, 2003; Shalev, Tuvia, Canetti, & Schreiber, 1996). Indeed, the presence of emotional numbing symptoms in the aftermath of trauma exposure may be one of the better predictors of subsequent PTSD (Feeny, Zoellner, Fitzgibbons, & Foa, 2000; Foa et al., 1995; Roemer, Litz, Orsillo, & Wagner, 2001).

Cognitive reappraisal, on the other hand, is a form of cognitive change that is used to neutralize a potentially emotion-eliciting situation before negative emotions are triggered (i.e., thinking about the situation in a manner such that one does not respond emotionally). Because it occurs earlier in the emotion-generative process, cognitive reappraisal often leads to better outcomes. (Gross, 1998; Richards & Gross, 2000) speculates that, because cognitive reappraisal occurs early in the process, it does not require constant self-regulation during an emotional event. It therefore, does not tax cognitive reserves. Indeed, cognitive reappraisal results in improved memory for emotionally charged events (Gross, 1998; Richards & Gross, 2000) and decreased anxiety and depression (Gross & John, 2003). Furthermore, it does not increase physiological arousal during emotionally charged events (Gross & Levenson, 1993, 1997) and has been viewed as adaptive for processing memories and integrating new experiences (Ehlers & Clark, 2000; Gross, 2002). Accordingly, cognitive appraisal may result in better mental and physical health consequences. Within the PTSD literature, Ehlers and Clark (2000) propose that emotional reactions seen in chronic PTSD are primarily dependent on cognitive appraisals of both the traumatic event and subsequent symptoms. Indeed, the presence of negative appraisals of a traumatic event and symptoms, the inability to reappraise a traumatic event and its consequences, and perceived negative and permanent change in personality and life aspiration differentiate those with and without chronic PTSD (e.g., Ehlers, Maercker, & Boos, 2000; Ehlers, Mayou, & Bryant, 1998; Foa, Ehlers, Clark, Tolin, & Orsillo, 1999).

In summary, across Gross' model and in the growing emotion regulation literature, there is a belief that emotion regulatory strategies may be associated with psychopathology. In general, it is hypothesized that use of expressive suppression is associated with worse psychopathology, whereas cognitive reappraisal is not. This may be particularly critical in the area of PTSD given that disruptions in emotional processes (e.g., numbing, detachment, dissociation) potentially impact symptom maintenance and treatment outcome. Gross' process model of emotion regulation may be particularly applicable to trauma exposed populations because it provides a detailed examination of these two emotion regulation processes. Furthermore, as mentioned, despite growing interest in emotion regulation, no

study till date has explored Gross' defined expressive suppression and cognitive reappraisal in a sample of individuals with and without trauma exposure and related psychopathology. Moreover, it remains unclear whether psychopathology is related to a general style of "expressive suppression" and non-psychopathology is related to a general style of "cognitive appraisal." That is, do individuals with PTSD-related symptoms predominantly use an expressive suppression emotion regulation style?

As such, the present study sought to further our understanding of emotional regulation styles as they relate to PTSD and related psychopathology. Specifically, we examined whether individuals evidence distinct patterns of emotion regulation across measures of emotion regulation frequency, emotion regulation capacity, suppression, and cognitive reappraisal, and whether these patterns were associated with psychopathology (i.e., PTSD, depression, anxiety). Specifically, to address this, we chose to utilize cluster analysis, allowing for classification of individuals into homogenous groups (Aldenderfer & Blashfield, 1984). In addition, we chose to focus on undergraduate women as a sample of convenience. That said, undergraduates tend to mirror the general population in terms of rates of psychopathology and trauma exposure (e.g., Bernat, Ronfeldt, Calhoun, & Arias, 1998). Furthermore, due to potential gender differences in emotion regulation and the experience of negative affect (e.g., Fujita, Diener, & Sandvik, 1991; Thomsen, Mehlsen, Viidik, Sommerlund, & Zachariae, 2005), and due to women being at an increased risk when compared to men to develop psychopathology (e.g., PTSD) following trauma exposure (e.g., Breslau, 2001; Breslau, Davis, Andreski, Peterson, & Schultz, 1997), we chose to limit our sample only to women. We hypothesized that some individuals would tend toward using expressive suppression and others would tend toward using cognitive reappraisal strategies as their predominant emotion regulation strategy (i.e., there would be "suppressors" and "reappraisers"). We further hypothesized that those individuals who predominantly used expressive suppression would exhibit higher psychopathology, and those who predominantly used cognitive reappraisal would exhibit lower psychopathology.

Method

Participants

Participants were 301 female undergraduate students at two large, metropolitan university campuses. They were recruited from a subject pool of students currently enrolled in psychology classes. To participate in the study, participants must be females between the ages of 18 and 65. Participants reported a mean age of 18.98 years (*SD* = 3.11). In terms of ethnicity, 64.5% classified themselves as Caucasian, 24.6% Asian Americans, and 11.9% other backgrounds. Based on the individuals completing (*N* = 295) the Posttraumatic Stress Diagnostic Scale (PDS, Foa, Cashman, Jaycox, & Perry, 1997), 157 (52.2%) women reported experiencing a potentially traumatic event. Of these, 62.4% (*n* = 98) reported experiencing more than one potentially traumatic event. If individuals reported more than one event, based on the PDS, they selected the event that bothered them the most to assess Criterion A status and subsequent PTSD. Within the sample, 40.3% (*n* = 119) reported a Criterion A event. Of those individuals, 37.8% reported a sexual or non-sexual assault, 27.2% reported an accident or natural disaster, and 34.5% reported another type of event (with 42.6% involving some kind of loss, 18% involving and accident or some form of violence, and 13.1% involving witnessing or experiencing severe physical illness).

Measures

The means, standard deviations, and ranges for all measures are shown in Table 1.

Emotion regulation questionnaire (ERQ)—Emotion regulation questionnaire (ERQ, Gross & John, 2003). The ERQ is self-report measure developed to assess the use of two emotion regulation strategies: cognitive reappraisal and expressive suppression. The present study utilized the original 24-item version, yielding four subscales: reappraisal (seven items), expressive suppression (five items), capacity (one item), and frequency (one item). Both the reappraisal and expressive suppression subscale are identical to the later 10-item version. Specifically, the capacity item reads, "Overall, I have a great deal of control over my emotions" and the frequency item reads, "I tend to control my emotions most of the time." Items are rated on a seven-point Likert scale (1 = *strongly disagree to* 7 = *strongly agree*), with mean scores computed for all subscales in order to ensure similar scaling for comparison. The ERQ has been shown to have good test–retest reliability (.69) and high internal consistently (.79 reappraisal, .73 suppression). The suppression subscale has been shown to be positively related to a measure of inauthenticity (ß = .47) and the venting subscale of the COPE (Carver, Scheier, & Weintraub, 1989; ß = –.43). It has also been shown to be negatively related to the attention (ß = .41), clarity (ß = .30), and repair (ß = .26) scales of Trait-Meta Mood scale (Salovey, Mayer, Golman, Turvey, & Palfai, 1995); negative mood regulation (ß = –.22) as assessed by the Negative Mood Regulation scale (Cantanzaro & Mearns, 1990); rumination (ß = .19), and extraversion (ß = –.41). Conversely, the reappraisal subscale is related to the reinterpretation scale of the COPE (ß = .43), the repair scale of the Trait-Meta Mood (ß = .36), and negative mood regulation (ß = –.30). It was negatively related to rumination (ß = –.29) and neuroticism (ß = –.20).

Posttraumatic stress diagnostic scale (PDS)—The PDS is a 49-item self-report measure that assesses DSM-IV Criteria A status, PTSD symptom severity, and PTSD diagnosis (Foa et al., 1997). Criteria A status is assessed through 12 questions regarding potentially traumatic events, selection and brief description of the event that has been most disturbing over the past month, and four yes/no questions assessing objective and subjective reactions to the most distressing event. The presence of current PTSD symptoms is assessed using 17 items that correspond to DSM-IV symptoms for PTSD (five re-experiencing, seven avoidance, and five arousal), asking participants to rate the frequency of each symptom in the past month on four-point Likert scale (0 = *not at all or only one time* to 3 = *five or more times a week/almost always*) in respect to the traumatic event. Sample items include: "Having upsetting thoughts or images about the traumatic event that came into your head when you didn't want them to," "Trying no to think about, talk about, or have feelings about the traumatic event," and "Being overly alert (for example, checking to see who is around you, being uncomfortable with your back to a door, etc.)." Impairment is assessed through a series of yes/no questions regarding functioning across a variety of domains (e.g., work, social). Total symptom severity is obtained by summing the 17 items, with higher scores reflecting greater symptom severity. Subscale scores are obtained by summing reexperiencing, avoidance, and arousal items, respectively. The PDS has been shown to have high internal consistency (.92), good test–retest reliability (.83), and good diagnostic agreement with the SCID (82% diagnostic agreement; sensitivity .89, specificity .75) (Foa et al., 1997).

Beck depression inventory (BDI)—Beck depression inventory (BDI, Beck, Ward, Mendelsohn, Mock, & Erbaugh, 1961). The BDI is a 21-item self-report measure of depression. Items are rated on a four-point Likert scale, with total scores ranging from 0 to 63, with higher scores indicating more severe symptomatology (e.g., "I do not feel sad (0)" to "I am so sad or unhappy that I can't stand it (3)"). The BDI has been shown to have good internal consistency, ranging from .69–.90, and test–retest reliability (.60).

State-trait anxiety inventory (STAI)—State-trait anxiety inventory (STAI, Spielberger, 1983). The STAI is a 40-item self-report measure assessing both state anxiety and trait anxiety. There are 20 items assessing state anxiety on a four-point Likert scale (1 = *not at all* to 4 = *very much so*; e.g., "I feel tense," "I feel jittery"), and 20 items assessing trait anxiety on a four-point Likert scale (1 = *almost never* to 4 = *almost always*; e.g., "I feel inadequate," "I worry too much over something that doesn't really matter"), with both state and trait anxiety scorings ranging from 20 to 80. For state anxiety, test–retest reliability is .40; and for trait anxiety, test–retest reliability is .81 (Spielberger, 1983). The STAI also has good internal consistency (.83–.92; Spielberger, 1983).

Anxiety sensitivity index (ASI)—Anxiety sensitivity index (ASI, Reiss, Peterson, Gursky, & McNally, 1986). The ASI is a 16-item measure assessing subjective fears of anxiety, physical sensations, and beliefs about why these sensations are harmful. Items are rated on a four-point Likert scale (0 = *very little* to 4 = *very much*), with total scores ranging from 0 to 48. Sample items include, "It is important to me not to appear nervous," "It scares me when I feel faint," and "It is important to me to stay in control of my emotions." The ASI has good test–retest reliability (.75) and high internal consistency (Reiss et al., 1986).

Ruminative response subscale of the response styles questionnaire (RRS)—Ruminative response subscale of the response styles questionnaire (RRS, Nolen-Hoeksema & Morrow, 1991). The RRS is a 21-item self-report subscale that assesses whether participants' responses to depressed mood are focused on the self (e.g., "I think back to other times I have been depressed"), on the symptoms (e.g., "I think about my feelings of fatigue and achiness"), and on the possible consequences and causes of the mood symptoms (e.g., "I think about a recent situation, wishing it had gone better"). The 22-items of this subscale are rated on a four-point Likert scale (1 = *never* to 4 = *always*), with total scores ranging from 22 to 88. The internal consistency of the RRS is good (.89; Nolen-Hoeksema & Morrow, 1991).

Results

Data analysis

Participants were classified according to their pattern of four subscale scores on the ERQ (frequency, capacity, cognitive reappraisal, suppression) using cluster analysis. All four subscales underwent *z*-score transformation. To identify groups of cases with similar responses on a set of variables, hierarchical cluster analysis was utilized (SPSS, Version 13.0, 2005. Chicago, Il: SPSS Inc). Using this method, a proximity metric is calculated to represent the dissimilarity between each pair of cases. Next, the two cases with the smallest distance are joined to form a group, with subsequent cases or groups of cases being combined. Specifically, we used a squared Euclidian distance metric and Ward's agglomeration schedule. These procedures produce a range of cluster solutions from the trivial number of participants (*N*-cluster) to a one-cluster solution. The decision to select one clustering solution over another is often dependent on the goals of classification (Aldenderfer & Blashfield, 1984). Specifically, our goal was to maximize separation of the sample and also preserve adequate cluster size to allow for analysis of medium to large size effects. Thus, we planned to employ the solution with the largest number of clusters having no fewer than 35 participants in any cluster.

Cluster solutions

Participants with similar emotion regulatory patterns were identified by use of cluster analysis. Optimal number of clusters was determined by examining the agglomeration coefficients and resulting dendrogram, resulting in a four substantive cluster solution of

emotion regulatory patterns. The mean subscale scores for each profile are shown in Figure 1. Cluster 1 (n = 120) consisted of participants who reported high levels of emotion regulation capacity, frequency, cognitive reappraisal, and suppression, termed henceforth as *high regulators*. Cluster 2 (n = 78) consisted of participants who reported high levels of emotion regulation capacity, frequency, and cognitive reappraisal but low levels of suppression, termed *high reappraisers/low suppressors*. Cluster 3 (n = 50) consisted of participants who reported moderate levels of emotion regulation capacity, frequency, cognitive reappraisal, and low levels of suppression, termed *moderate reappraisers/low suppressors*. Finally, Cluster 4 (n = 42) consisted of participants who reported low levels of emotion regulation capacity, frequency, cognitive reappraisal, and moderate levels of suppression, termed *low regulators*.

Internal validity—To examine the internal validity of these patterns, we evaluated the degree to which hierarchical clustering differentiated between ERQ subscales, using separate between-group analyses of variance (ANOVA) for each of the ERQ subscales: capacity, frequency, cognitive reappraisal, and suppression. As shown in Table 2, high regulators scored higher than low regulators on all ERQ subscales. The high reappraisers/ low suppressors consistently reported higher levels of emotion regulation frequency, capacity, and reappraisal than the moderate reappraisers/low suppressors; but both clusters reported similar levels of suppression. Thus, there was evidence of four distinct regulatory patterns, suggesting good internal validity.

External validity—To examine the external validity of the emotion regulation clusters, we examined whether cluster membership was associated with psychopathology (i.e., depression, state anxiety, trait anxiety, PTSD) and other related measures (i.e., anxiety sensitivity, rumination). As shown in Table 3, individuals in the low emotion regulator cluster reported greater psychopathology across all general psychopathology measure (i.e., depression, state anxiety, trait anxiety) than individuals in the high regulator, high reappraiser/low suppressor, and moderate reappraiser/low suppressor clusters. Furthermore, it should be noted that individuals in the high reappraiser/low suppressor cluster consistently reported the least amount of general psychopathology compared to all other clusters. Although both anxiety sensitivity and ruminative response style displayed similar, though less robust, pattern of results, ruminative response style did not differ across clusters.

To address the reactions following exposure to traumatic events, examining only Criteria A exposed individuals (n = 119), a pattern similar to other measures of psychopathology can be shown in Table 3, with the low regulator cluster consistently reporting greater PTSD severity, across all PTSD subscales, than the high reappraiser/low suppressor cluster. A similar pattern was further replicated when examining PTSD diagnostic status. Forty percent of low regulator cluster (40.9%) met PDS diagnostic criteria for PTSD; whereas, only 15.4% of the individuals in the high regulator, 13.3% of the high reappraiser/low regulator, and 18.2% in the moderate reappraiser/low suppressor clusters met PTSD diagnostic criteria, χ^2 (3, N = 115) = 7.56, p = .06.

To further explore these associations, we then examined whether trauma exposure itself could account for differences among clusters. Inconsistent with the above psychopathology pattern, only the moderate reappraiser/low suppressor cluster (22%) reported lower exposure to Criteria A events than the other clusters (High regulator: 43.7%; High reappraiser/Low suppressor: 41.1%; Low regulator: 52.4%), χ^2 (3, N = 284) = 10.08, $p < .05$. Due to the possibility of multiple trauma exposure increasing the likelihood of PTSD and emotion regulation difficulties (e.g., Baynard, Williams, & Siegel, 2001; Breslau, Peterson, Poisson, Schultz, & Lucia, 2004; Classen, Palesh, & Aggarwal, 2005), we also examined cluster membership and the experience of multiple potentially traumatic events (no event, one

event, and more than one event). Here, there was no strong associations between cluster membership and multiple exposure, χ^2 (4, N = 290) = 9.31, *ns* (Multiple exposure: High regulator: 38.3%, High reappraiser/Low suppressor: 26.9%, Moderate reappraiser/Low suppressor: 20%; Low regulator 42.9%). In summary, consistent across a variety of psychopathology measures, low regulators displayed greater psychopathology in comparison to high reappraisers/low suppressors; however, these differences could not be accounted solely due to trauma exposure.

Discussion

Four distinct clusters of emotion regulation emerged: high regulators, high reappraisers/low suppressors, moderate reappraisers/low suppressors, and low regulators, suggesting clear groupings of how individuals regulate their emotions. Notably, individuals with high reappraisal and low suppression, termed *high reappraisers/low suppressors*, consistently reported lower levels of depression, anxiety, and PTSD symptoms than individuals who reported regulating their emotions less effectively and infrequently using reappraisal and suppression, termed *low emotion regulators*. Difference in cluster membership could not be accounted for by trauma exposure. Although partially consistent with Gross and John (2003), suggesting an adaptive role of high reappraisal and low suppression, our findings also suggest a more general maladaptive emotion regulatory deficit in individuals with higher levels of psychopathology, with these individuals reporting both a reduced capacity and frequency of usage of either emotion regulation strategy.

The most common *high regulator* cluster (n = 120) consisted of individuals who reported frequently and effectively using *both* reappraisal and suppression. Membership in this cluster was associated with neither the highest nor the lowest levels of depression, anxiety, and PTSD symptoms. Thus, if anything, the most prominent grouping of individuals do not appear to be either “suppressors” or “reappraisers” but instead use both to help regulate their emotions. Although it does not appear that individuals are either “suppressors” or “reappraisers,” the presence of a *high reappraiser/low suppressor* cluster is most consistent with the notion that some individuals predominantly use reappraisal as an emotion regulatory style; that is, some are indeed “reappraisers.” Notably, the lower psychopathology evidenced in this cluster may be more associated with increased capacity and frequency of reappraisal and not associated with lower suppression; as lower psychopathology was not observed in the *moderate reappraiser/low suppressor* cluster. However, *high regulators* overall also did not evidence lower psychopathology. Nevertheless, this focus on reappraisal is consistent with previous literature on reappraisal being an adaptive emotion regulation strategy (e.g., Ehlers & Clark, 2000; Frijda, 1993; Gross, 1998; Gross & John, 2003; Richards & Gross, 2000). Specifically, individuals who reappraise stressors adaptively report lower levels of psychopathology and general distress (Gross & John, 2003; Gross & Muñoz, 1995), anxiety (e.g., Calvo & Eysenck, 2000), and negative emotions (e.g., Folkman & Lazarus, 1988). Furthermore consistent with Gross and John (2003), when we correlated overall cognitive reappraisal, expressive suppression, PTSD severity, the pattern of findings was in this hypothesized direction. Higher PTSD severity was modestly associated with lower cognitive reappraisal (r = –.19, p < .05).

Potentially more interesting, individuals with a generally low emotion regulatory style and moderate levels of suppression consistently reported the highest levels of depression, anxiety, and PTSD. This low capacity and frequency of emotion regulation may reflect a general deficit in emotion regulation flexibility. Both expressive suppression and cognitive reappraisal can be viewed as either adaptive or maladaptive responses depending on their function, context, and timing (e.g., Bonanno, 2004; Gross, 2001). As Bonanno and colleagues (Bonanno, 2004; Bonanno et al., 2004) suggest, the ability to enhance or suppress

emotional expression flexibly in accordance with the situation may result in better adjustment than the use of any one regulatory process. Similarly, this pattern of a more generally low emotion regulatory style in some individuals may reflect the presence of ego depletion. According to Baumeister and colleagues' ego depletion model (Baumeister, Bratslavsky, Muraven, & Tice, 1998; Baumeister, Muraven, & Tice, 2000), when internal resources are depleted, the ability to control one's thoughts, behaviors, and emotions are compromised. As such, those with psychopathology have taxed their internal resources. Possibly, low regulators represent those individuals whose resources are most depleted, resulting in less energy and ability to regulate emotions. Without the ability to tolerate and modulate emotions, low regulators would be less likely to distinguish contexts in which specific strategies are adaptive. Indeed, this is consistent with Litz's model of emotional numbing in PTSD (Litz, 1992; Litz & Gray, 2002), suggesting a depletion of emotional resources after periods of hyperemotionality.

Nevertheless, lower emotion regulation may be a consequence and not a cause of psychopathology; individuals with the highest levels of distress may in fact have the greatest difficulty regulating their emotions (e.g., Lynch, Chapman, Rosenthal, Kuo, & Linehan, 2006). In particular, a history of multiple and chronic trauma exposure, specifically childhood sexual abuse (CSA), may developmentally impair an individual's ability to effectively regulate his or her emotions resulting in worse mental-health outcomes (e.g., Baynard et al., 2001; Classen et al., 2005; Cloitre, Koenen, Cohen, & Han, 2002). Yet, within our current sample, there was no strong association between trauma exposure and cluster membership nor was there a strong association for those who experienced multiple traumas and cluster membership. However, our sample was too small to further specifically examine CSA (n = 10) and therefore we cannot conclude a lack of association between CSA and low emotion regulation. Several limitations should be noted. First, our present findings may reflect a lack of specificity in the Emotional Regulation Questionnaire measure. Specifically, this measure does not inquire regarding the nature and intensity of emotions that individuals are regulating. For example, emotion regulation during a distressing trauma-related memory may not be the same as that of the emotion regulation of an upsetting conversation with a boss or after an argument with a significant other. The ERQ does not specify the context around which individuals are regulating their emotions. Thus, the context surrounding emotion regulation may be both qualitatively and quantitatively different. Consequently, low regulators with higher psychopathology, may be attempting to regulate vastly different types of emotions than high regulators. Second, the cross-sectional design of the study does not allow for examination of causal relationships. Thus, we cannot conclude whether lower emotion regulation is a cause or consequence of psychopathology, just that they are associated. Third, only a few forms of emotion regulation were examined. For example, the role of emotional venting, denial, seeking support and other such regulatory strategies were not explored. Finally, our participants were female undergraduates, only some of whom met Criteria A exposure (40.3%) and fewer of whom met PTSD diagnostic criteria (8.1%). Furthermore, no interview assessment of PTSD was made nor was the present study a clinical sample, with a potentially restricted range of psychopathology scores. Yet, it should be noted that the PDS has good convergent validity with diagnostic assessment (Foa et al., 1997); and the current sample did report a full range of clinical scores, though the pattern of scores is more consistent with the general population than a clinical sample. Finally, further work is needed across other emotion regulation measures to better understand if our findings represent normal within group variation. Nevertheless, the four clusters that emerged here showed solid internal and external validity.

Our findings highlight that the capacity to regulate emotions and the ability to apply different strategies based on the context and timing may be critical components associated with heightened psychopathology. Clearly, the replication of this more general low pattern

of emotion regulation and the possible role of both cognitive flexibility and ego depletion as explanatory factors underlying psychopathology needs further exploration. If individuals with higher psychopathology tend to use various emotion regulation strategies less and have less success when utilizing them, consistent with our findings from the high reappraiser/low suppressor cluster, clinicians ought to focus on providing patients with a wide range of emotion regulation skills to apply, encouraging the use of adaptive emotion regulation strategies such as reappraisal at a greater frequency, and facilitating their flexible application depending on the situational context. It may be that the ability to flexibly regulate emotions in accordance with the situational context actually frees up internal resources, resulting in better mental health outcomes.

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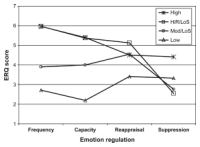

Figure 1.
ERQ scores on frequency, capacity, reappraisal, and suppression by cluster membership.

Table 1

Mean, standard deviation, and range on emotion regulation subscales and psychopathology measures.

Measure	*M*	*SD*	Range
Emotion regulation frequency (ERQ)	5.13	1.50	1–7
Emotion regulation capacity (ERQ)	4.70	1.57	1–7
Cognitive reappraisal (ERQ)	4.53	1.01	1–7
Suppression (ERQ)	3.50	1.18	1–7
Criterion A traumatic event (PDS)	40.3%		
Sexual or non-sexual assault	37.8%		
Accident or natural disaster	27.7%		
Other	34.5%		
PTSD Severity (PDS)[a]	8.03	9.84	0–14
PTSD Diagnosis (PDS)	8.1%	NA	NA
Depression (BDI)	7.91	7.03	0–41
Anxiety Sensitivity (ASI)	35.44	9.55	16–69
State Anxiety (STAI-State)	39.07	11.9	20–79
Trait Anxiety (STAI-Trait)	41.25	12.0	20–76
Rumination (RRS)	51.78	7.49	28–74

[a]Data presented above is for entire sample ($N = 295$), however, PTSD total symptoms is calculated for those reporting a Criterion A event ($n = 119$). PDS, Posttraumatic Stress Disorder Diagnostic Scale; BDI, Beck Depression Inventory; ASI, Anxiety Sensitivity Inventory; STAI, State-Trait Anxiety Inventory; RRS, Ruminative response subscale.

Table 2

Internal criterion validity: Emotion regulation subscales among clusters.

Variable	High regulator	High reappraisal/Low suppressor	Moderate reappraisal/Low suppressor	Low regulator	*F*	Cohen's d_s
Frequency	5.97 (.72)[a]	5.97 (.72)[a]	3.90 (.95)[b]	2.71 (.99)[c]	234.81	4.03
Capacity	5.40 (l.22)[a]	5.37 (.98)[a]	4.00 (.95)[b]	2.19 (l.02)[c]	108.17	2.94
Reappraisal	4.51 (.86)[b]	5.13 (.83)[a]	4.55 (l.00)[b]	3.41 (.82)[c]	35.27	1.97
Suppression	4.42 (.66)[a]	2.54 (.69)[c]	2.76 (.80)[c]	3.33 (1.44)[b]	93.28	2.21

Note: Means in the same row that do not share the same subscripts differ at $p < .05$. Cohen's d_S (Hedges's g) was used for calculating one-way, four group effect sizes.

Table 3

External criterion validity: Psychopathology among clusters.

Variable	High regulator	High reappraisal/Low suppressor	Moderate reappraisal/Low suppressor	Low regulator	*F*	d_s
Depression (BDI)	8.03 (6.16)[b]	4.53 (4.73)[a]	8.00 (6.78)[b]	13.02 (9.40)[c]	15.64	1.30
State Anxiety (STAI)	39.97 (10.40)[b]	32.84 (9.79)[a]	38.66 (10.78)[b]	48.31 (14.15)[c]	18.74	1.42
Trait Anxiety (STAI)	42.74 (10.80)[b]	33.78 (8.72)[a]	40.20 (10.87)[b]	51.85 (13.24)[c]	22.72	1.69
Anx. Sens. (ASI)	36.45 (8.52)[b]	32.59 (8.89)[a]	35.27 (9.85)	37.80 (11.12)[b]	3.86	.56
Rumination (RRS)	52.55 (7.35)	50.36 (7.25)	50.93 (7.72)	53.44 (7.35)	1.86	.37
PTSD total (PDS)[1]	7.58 (8.27)[a]	5.10 (7.04)[a]	5.27 (8.43)	14.23 (14.12)[b]	4.51	.97
Reexp[1]	2.42 (2.87)	1.73 (2.57)[a]	1.73 (2.45)	4.32 (4.52)[b]	3.27	.82
Avoid[1]	3.17 (3.85)	1.80 (3.08)[a]	2.27 (4.58)	5.50 (6.12)[b]	3.37	.87
Arousal[1]	1.98 (2.80)[a]	1.57 (2.64)[a]	1.27 (1.85)[a]	4.41 (4.45)[b]	4.6	.92

Note: Means in the same row that do not share the same subscripts differ at $p < .05$. Cohen's d_S (Hedges's g) was used for calculating one-way, four group effect sizes.

[1]PTSD was calculated only for individuals reporting a Criterion A event ($n = 119$).