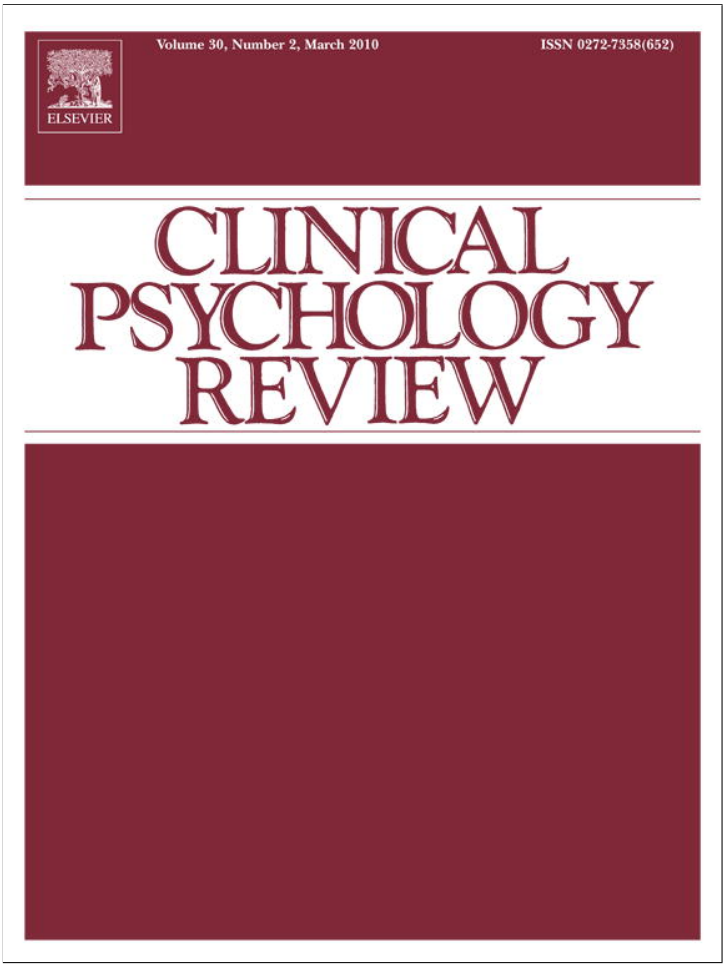

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# Emotion-regulation strategies across psychopathology: A meta-analytic review

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ABSTRACT

We examined the relationships between six emotion-regulation strategies (acceptance, avoidance, problem solving, reappraisal, rumination, and suppression) and symptoms of four psychopathologies (anxiety, depression, eating, and substance-related disorders). We combined 241 effect sizes from 114 studies that examined the relationships between dispositional emotion regulation and psychopathology. We focused on dispositional emotion regulation in order to assess patterns of responding to emotion over time. First, we examined the relationship between each regulatory strategy and psychopathology across the four disorders. We found a large effect size for rumination, medium to large for avoidance, problem solving, and suppression, and small to medium for reappraisal and acceptance. These results are surprising, given the prominence of reappraisal and acceptance in treatment models, such as cognitive-behavioral therapy and acceptance-based treatments, respectively. Second, we examined the relationship between each regulatory strategy and each of the four psychopathology groups. We found that internalizing disorders were more consistently associated with regulatory strategies than externalizing disorders. Lastly, many of our analyses showed that whether the sample came from a clinical or normative population significantly moderated the relationships. This finding underscores the importance of adopting a multi-sample approach to the study of psychopathology.



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Emotion regulation is increasingly being incorporated into models of psychopathology (Berenbaum, Raghavan, Le, Vernon, & Gomez, 2003; Greenberg, 2002; Kring & Bachorowski, 1999; Mennin & Farach, 2007). As "distress disorders" (Watson, 2005), depression and anxiety are widely viewed as the result of difficulties in regulating emotions (Campbell-Sills & Barlow, 2007; Gross & Munoz, 1995; Mennin, Holoway, Fresco, Moore, & Heimberg, 2007). Several theorists argue that individuals who cannot effectively manage their emotional responses to everyday events experience longer and more severe periods of distress that may evolve into diagnosable depression or anxiety (e.g., Mennin et al., 2007; Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008). In addition, models of eating disorders (Fairburn et al., 1995; McCarthy, 1990; Polivy & Herman, 1998, 2002) and alcohol abuse (Sher & Grekin, 2007; Tice, Bratslavsky, & Baumeister, 2001) suggest that individuals with poorly regulated emotions often turn to food or alcohol to escape from or down-regulate their emotions, creating risk for diagnosable problems in relation to food or alcohol.

Several individual emotion-regulation strategies have been hypothesized to be risk factors for or protective factors against psychopathology. In the meta-analysis presented here, we first review models of emotion regulation that have linked specific regulatory strategies to psychopathology. Then, we evaluate the empirical support for the relationships between symptoms of four psychopathologies and six emotion-regulation strategies. We ask which emotion-regulation strategies are most strongly associated with psychopathology. We also ask if there is specificity in the relationship between certain emotion-regulation strategies and different psychopathologies.

## 1. Models of emotion regulation

Emotion regulation has been conceptualized as processes through which individuals modulate their emotions consciously and nonconsciously (Bargh & Williams, 2007; Rottenberg & Gross, 2003) to appropriately respond to environmental demands (Campbell-Sills & Barlow, 2007; Cole, Martin, & Dennis, 2004; Gratz & Roemer, 2004; Gross, 1998; Gross & Munoz, 1995; Thompson, 1994). Individuals deploy regulatory strategies to modify the magnitude and/or type of their emotional experience or the emotion-eliciting event (Diamond & Aspinwall, 2003; Gross, 1998). The process of emotion regulation has been conceptualized as distinct from the emotion generation process (Cole et al., 2004; Gross & Thompson, 2007; Rottenberg & Gross, 2003) although the distinction between these two processes still remains the source of debate (see Campos, Frankel, & Camras, 2004).

Theoretical models associate successful emotion regulation with good health outcomes, and improved relationships and academic and work performance (Brackett & Salovey, 2004; John & Gross, 2004). Conversely, difficulties with emotion regulation are associated with mental disorders (Berenbaum et al., 2003; Greenberg, 2002; Kring & Bachorowski, 1999; Mennin & Farach, 2007) and incorporated into several models of specific psychopathologies, including borderline personality disorder (BPD; Linehan, 1993; Lynch, Trost, Salsman, & Linehan, 2007), major depressive disorder (Nolen-Hoeksema et al., 2008; Rottenberg, Gross, & Gotlib, 2005), bipolar disorder (Johnson, 2005), generalized anxiety disorder (GAD; Mennin et al., 2007), social anxiety disorder (SAD; Kashdan & Breen, 2008), eating disorders (Bydlowski et al., 2005; Clyne & Blampied, 2004; Fairburn et al., 1995; McCarthy, 1990; Polivy & Herman, 2002), alcohol-related disorders (Sher & Grekin, 2007; Tice et al., 2001) and substance-related disorders (Fox, Axelrod, Paliwal, Sleeper, & Sinha, 2007; Linehan et al., 2002; Sher & Grekin, 2007; Tice et al., 2001). Consequently, various therapeutic approaches incorporate some form of emotion-regulation training, including dialectical behavioral therapy (Linehan, 1993), emotion focused therapy (Greenberg, 2002), acceptance- and mindfulness-based therapy (Hayes, Strosahl, & Wilson, 1999; Roemer, Orsillo, & Salters-Pedneault, 2008; Segal, Williams, & Teasdale, 2002), and emotion-regulation therapy (Mennin & Fresco, 2009).

### 1.1. Adaptive and maladaptive emotion regulatory strategies

Over the years, different theoretical models have highlighted different specific strategies as adaptive or maladaptive. Dating back to 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, 1992; D'Zurilla, 1988; Marlatt, Baer, Donovan, & Kivlahan, 1988), two strategies thought to be adaptive across a variety of contexts are reappraisal and problem solving. Reappraisal involves generating benign or positive interpretations or perspectives on a stressful situation as a way of reducing distress (Gross, 1998). Maladaptive appraisal processes are thought to be at the core of depression and anxiety according to several models (Beck, 1976; Clark, 1988; Salkovskis, 1998), and thus cognitive-behavioral therapies for depression and anxiety focus on teaching reappraisal skills (Beck, Rush, Shaw, & Emery, 1979). More recently, Gross' (1998) influential model of emotion regulation highlights reappraisal as a strategy that results in positive emotional and physical responses to emotion-eliciting stimuli.

Problem-solving responses are conscious attempts to change a stressful situation or contain its consequences. Problem solving is often assessed as an orientation or specific actions directed at solving a problem (e.g., brainstorming solutions, planning a course of action). Although problem-solving responses are not direct attempts to regulate emotions, they can have beneficial effects on emotions by modifying or eliminating stressors. Low problem-solving orientation or poor problem-solving skills have been theorized to lead to depression (Billings & Moos, 1981; D'Zurilla, Chang, Nottingham, & Faccinni, 1998), anxiety (Chang, Downey, & Salata, 2004; Kant, D'Zurilla & Maydeu-Olivares, 1997), substance use (Cooper et al., 1992), and eating disorders (VanBoven & Espelage, 2006). Training in problem-solving skills is a component of cognitive-behavioral therapies for all these disorders (Beck et al., 1979; Fairburn et al., 1995; Marlatt et al., 1988).

More recently, there has been an increased interest in the role of mindfulness in adaptive emotion regulation (Gratz & Roemer, 2004). Although the precise delineation of the components of mindfulness is still the source of much debate (Baer, Smith, & Allen, 2004; Bishop et al., 2004) one component that most researchers tend to agree upon is that of non-judgmental acceptance of emotions. Along these lines, mindfulness has been conceptualized as non-elaborative, non-

judgmental, present-centered awareness in which thoughts, feelings, and sensations are accepted as they are (Kabat-Zinn, 1990; Segal et al., 2002; Shapiro & Schwartz, 1999). Mindfulness-based therapies that emphasize taking a non-judgmental stance have been developed to treat a variety of disorders, including depression (Segal et al., 2002), anxiety (Roemer et al., 2008), eating disorders (Kristeller, Baer, & Quillian-Wolever, 2006), substance abuse (e.g., Breslin, Zack, & McMain, 2002; Marlatt et al., 2004), and borderline personality disorder (Linehan, 1993; Lynch et al., 2007). Research on acceptance as a regulatory strategy has suggested that using this strategy promotes good outcomes (Hayes et al., 1999; Heffner, Eifert, Parker, Hernandez, & Sperry, 2003) whereas low levels of acceptance have been shown in many disorders, including generalized anxiety disorder (McLaughlin, Mennin, & Farach, 2007; Roemer et al., 2008), panic disorder (Tull & Roemer, 2007), heroin use (Tull, Schulzinger, Schmidt, Zvolensky, & Lejeuz, 2007), and borderline personality disorder (Gratz, Rosenthal, Tull, & Lejuez, 2006).

In contrast, suppression and avoidance have long been seen as maladaptive responses to a variety of stressors, and risk factors for both distress (i.e., depression and anxiety) and maladaptive behaviors (particularly substance abuse; Carver et al., 1989; Folkman & Lazarus, 1980). Various forms of suppression and avoidance have been implicated in psychopathology. Gross' model (1998) focuses primarily on suppression of emotional *expression*, and argues that although expressive suppression may reduce the outward expression of emotion and possibly the subjective experience of emotion in the short term, it will be less effective in reducing emotion and physiological arousal over the long term (Gross, 1998; Gross & Thompson, 2007; John & Gross, 2004).

Other theorists have focused on the suppression of unwanted thoughts. Wenzlaff and Wegner (2000) have produced a large body of research showing that attempts to voluntarily suppress thoughts result in an increased accessibility of the suppressed thought (Wegner & Erber, 1992; Wegner, Schneider, Carter, & White, 1987) and increased emotional arousal, especially in the physiological domain, as evidenced by increased electrodermal responses to emotional stimuli (e.g., Wegner, Broome, & Blumberg, 1997). They have also suggested that chronic suppression might prevent habituation to emotional stimuli, and as such result in hypersensitivity to depression and anxiety-related thoughts and symptoms (Wegner & Zanakos, 1994; Wenzlaff & Wegner, 2000).

Hayes et al. (1999) have been concerned with what they call *experiential avoidance:* the suppression or avoidance of an array of psychological experiences, including thoughts, emotions, sensations, memories, and urges. They have argued that experiential avoidance can lead to a variety of negative outcomes, ranging from problems with mood to problems with substance use, because it paradoxically increases negative thoughts (i.e., Wenzlaff & Wegner, 2000) and prevents individuals from taking necessary action (Hayes et al., 2004). Similarly, emotion-regulation models of eating disorders suggest that avoidance of psychological experiences leads to binge eating and then maladaptive compensatory behaviors (e.g., Heatherton & Baumeister, 1991; McCarthy, 1990; Polivy & Herman, 2002). Hayes et al. propose that acceptance is an adaptive alternative to avoidance and they have developed a corresponding treatment: acceptance and commitment therapy (ACT; Hayes et al., 1999).

Avoidance has also been conceptualized in the behavioral domain. Mowrer's (1947) two-stage theory of fear proposes that: 1) fear is acquired through classical conditioning and, 2) as individuals avoid the feared stimulus, extinction cannot take place and thus the fear is maintained via operant conditioning. This model has been mostly applied to anxiety disorders, such as panic disorder (Barlow, Craske, Cerny, & Klosko, 1989; Lissek et al., 2009); posttraumatic stress disorder (Foa & Kozak, 1986); specific phobia (Merckelbach, de Jong, Muris, & van den Hout, 1996); and agoraphobia (Rachman, 1993). Interestingly, avoidance of other non-fear-based negative states, such as withdrawal symptoms after substance use, has also been linked to psychopathology. In this case, individuals going through withdrawal find this state unpleasant and might try to regulate it by avoiding it, that is, by using the substance they are abstaining from (Baker, Piper, McCarthy, Majeski, & Fiore, 2004).

Finally, instead of avoiding or suppressing negative thoughts and moods, some individuals repetitively focus on their experience of the emotion and its causes and consequences (Nolen-Hoeksema et al., 2008; Trapnell & Campbell, 1999; Watkins, 2008). People often say they engage in rumination because they want to understand and solve their problems (Papageorgiou & Wells, 2003), but rumination is negatively related to problem solving (Hong, 2007). Indeed, rumination in the context of distress appears to interfere with good problem solving, and may immobilize individuals in indecision (Ward, Lyubomirsky, Sousa, & Nolen-Hoeksema, 2003). Although rumination has been most frequently studied in relation to depression and anxiety, it has recently been studied in relation to substance use and eating disorders (e.g., Nolen-Hoeksema, Stice, Wade, & Bohon, 2007). Binge-drinking and binge eating may be attempts to escape from the aversive self-awareness maintained by rumination (Heatherton & Baumeister, 1991).

Thus, three emotion-regulation strategies that have been widely theorized to be protective against psychopathology are reappraisal, problem solving, and acceptance. Three strategies that have consistently been argued to be risk factors for psychopathology are suppression (including both expressive suppression and thought suppression), avoidance (including both experiential avoidance and behavioral avoidance), and rumination. However, a direct comparison of the degree to which they relate to psychopathology is needed.

In this meta-analysis, we address two main questions. First, we are interested in the relationship between each of the six regulatory strategies of interest and psychopathology. That is, we propose to investigate the sensitivity of each emotion-regulation strategy in capturing variance in different types of psychopathology. Identification of strategies that might have stronger relationships to specific psychopathologies can inform the improvement of existing treatments and provide avenues for new interventions (Berking, Wupperman, Orth, Meier, & Caspar, 2008). The second aim of the present investigation is to examine the specificity of each of these six regulatory strategies, that is, are emotion-regulation strategies related to some forms of psychopathology but not others. Emotion-regulation strategies have been mostly examined within the context of internalizing disorders, but as noted earlier, recently emotion-regulation models have also been applied to externalizing disorders (e.g., Nolen-Hoeksema et al., 2008). We limited the psychopathologies we surveyed to four types that several theorists have claimed are linked to emotion-regulation deficits: depression (Gross & Munoz, 1995; Nolen-Hoeksema, 1991), anxiety (Campbell-Sills & Barlow, 2007; Mennin et al., 2007), eating disorders (Bydlowski et al., 2005; Clyne & Blampied, 2004; Fairburn et al., 1995; McCarthy, 1990; Polivy & Herman, 2002), and substance-related disorders (Fox et al., 2007; Sher & Grekin, 2007; Tice et al., 2001).

## 2. Measuring emotion regulation

Along with the increased interest in individual differences in emotion regulation in recent years, there has been a proliferation of self-report measures of emotion-regulation strategies (see Table 1). These measures have advantages and disadvantages. On the one hand, because self-report measures are easy and quick to administer, there is a sufficient body of research from which to draw data for this meta-analysis. In addition, self-report scales typically measure dispositional tendencies toward certain emotion-regulation strategies, and thus supposedly assess what participants do across time and different contexts. This is in line with Campbell-Sills and Barlow's (2007) argument that in conceptualizing emotion dysregulation and its relationship to psychopathology we need to take a "long view," assessing patterns of responding to episodes of emotion over time that lead to the persistence or recurrence of unwanted emotions over time.

**Table 1**
Study characteristics — cross-sectional and experimental designs.

| Author name | Sample details/moderators | Design | Psychopathology measures | Emotion-regulation measures |
|---|---|---|---|---|
| Allan and Gilbert (2002) | 197 college students | Experimental | CES-D | STAXI: anger-in |
| Ball, Smolin, and Shekhar (2002) | 12 adult patients with anxiety NOS, 45 patients with panic disorder, 13 patients with social anxiety, 12 patients with OCD, 21 patients with depression, and 17 healthy controls | Cross-sectional | ADIS for DSM-IV | TCI: harm avoidance |
| Borders, Barnwell, and Earleywine (2007) | 285 college students | Cross-sectional | Alcohol consumed in the last 6 months; alcohol-aggression expectancies; alcohol-related aggression index; alcohol-related hostility index | RRQ: rumination |
| Britton (2004) | 196 college students | Cross-sectional | Alcohol-related consequences; quantity frequency variability average number of drinking days; heavy drinks days | COPE: acceptance, positive reinterpretation |
| Burns, Bruehl, and Caceres (2004) | 53 college students | Experimental | STAI | AEI: anger-in |
| Cheung, Gilbert, and Irons (2004) | 125 college students | Cross-sectional | CES-D | RRS |
| Ciesla and Roberts (2007) | 263 college students | Experimental | BDI | RRS |
| Connolly, Rieger, and Caterson (2007) | 140 female college students | Cross-sectional | BES | BARQ: avoidance, rumination; STAXI-2: anger-in |
| Conway, Mendelson, Giannopoulos, Csank, and Holm (2004) | 201 college students | Cross-sectional | BDI | ROS |
| Cox, Stabb, and Hulgus (2000) | 161 children/adolescents 5th–9th graders | Cross-sectional | CDI | PAES-III: anger-in |
| Dennis (2007) | 67 college students | Cross-sectional | BDI; STAI | ERQ: reappraisal, suppression |
| Donaldson, Lam, and Mathews (2007) | 36 adult patients with depression and 36 healthy controls | Experimental | SCID for DSM-IV | RRS |
| Egloffet al. (2006) | 82 college students | Cross-sectional | STAI | ERQ: reappraisal, suppression |
| Erskine, Kvavilashvili, and Kornbrot (2007) | Sample 1: 84 college students; sample 2: 65 adult community members | Cross-sectional | BDI; STAI | RI; WBSI |
| Felsten (1998) | 240 college students | Cross-sectional | BDI | CSI: avoidance, problem solving |
| Flett, Hewitt, Blankstein, Solnik, and Van Brunschot (1996) | 114 college students | Cross-sectional | BAI; CES-D | SPSI: problem orientation, problem-solving skills, social problem solving |
| Flett, Madorsky, Hewitt, and Heisel (2002) | 65 college students | Cross-sectional | MASQ: anhedonic depression, anxious arousal, general distress anxious symptoms, general distress depressive symptoms | RSQ 10 rumination items |
| Fresco et al. (2007) | 61 college students | Cross-sectional | BDI; MASQ: anhedonic depression, anxious arousal | AAQ; ERQ: reappraisal, suppression; RSQ: brooding |
| Geller, Cockell, Hewitt, Goldner, and Flett (2000) | 21 adult female patients with anorexia nervosa and 21 healthy controls | Cross-sectional | EDE | STAXI-2 anger-in; STSS: silencing the self |
| Goldman and Haaga (1996) | 33 depressed adult patients and 41 healthy controls | Cross-sectional | Composite BDI + SCID for DSM-III | STAXI: suppression target others, suppression target spouse |
| Goldstein (2006) | Sample 1: 57 female adult community members; sample 2: 51 male community members | Cross-sectional | BDI; KAT | RRS |
| Good, Heppner, Debord, and Fischer (2004) | 260 male college students | Cross-sectional | BDI; STAI | PSI |
| Gross and John (2003) | 210 college students | Cross-sectional | BDI; CES-D; ZDS | ERQ: reappraisal, suppression |
| Haaga, Fine, Terrill, Stewart, and Beck (1995) | 115 college students | Cross-sectional | BAI; composite BDI + IDD | SPSI |
| Hankin, Lakdawalla, Carter, Abela, and Adams (2007) | Sample 1: 950 college students; sample 2: 431 college students | Cross-sectional | BDI | RRS brooding |
| Harrington and Blankenship (2002) | 199 college students | Cross-sectional | BAI; BDI | SMRI |
| Harris, Pepper, and Maack (2008) | 96 college students | Cross-sectional | BDI | RSQ: brooding |
| Haugh (2006) | 245 college students | Cross-sectional | BAI; BDI | SPSI-R: positive problem orientation |
| Hayes et al. (2004) | Sample 1: 202 college students; sample 2: 304 college students; sample 3: 205 college students | Cross-sectional | ASI; BAI; BDI | AAQ |
| Hewitt et al. (2002) | 114 children/adolescents; 10–15 years old | Cross-sectional | CDI; R-CMAS | PAES-III: anger suppression |

| | | | | |
|---|---|---|---|---|
| Joormann et al. (2006) | 20 adult patients with social anxiety, 64 with depression, and 91 healthy controls | Experimental | SCID for DSM-IV | RRS |
| Joormann and Gotlib (2008) | 23 adult patients with depression and 21 healthy controls | Experimental | SCID for DSM-IV | RRS brooding |
| Kant et al. (1997) | Sample 1: 100 adult community members; sample 2: 100 adult community members | Cross-sectional | BDI; STAI | SPSI-R |
| Kashdan, Barrios, Forsyth, and Steger (2006) | 382 college students | Cross-sectional | ASI; BSQ; SFS; STAI | AAQ; CSQ: avoidant, rational; ECQ |
| Kashdan and Breen (2007a,b) | 144 college students | Cross-sectional | BDI; MASQ: anhedonic depression, anxious arousal; SIAS | AAQ; DERS: non-acceptance; EACQ: emotional expression |
| Kashdan and Breen (2007, 2008) | 246 community members | Cross-sectional | ASI; MASQ: anhedonic depression, anxious arousal | DERS; EACQ: emotional expression |
| Knowles, Tai, Christensen, and Bentall (2005) | 528 college students | Cross-sectional | BDI | RSQ 23-item rumination scale; 4-item problem-solving scale |
| Kocovski, Endler, Rector, and Flett (2005) | 55 college students with social anxiety and 57 healthy controls | Experimental | EMAS — trait anxiety — social evaluation | CHIP |
| Koff and Sangani (1997) | 128 female college students | Cross-sectional | EAT | CISS: avoidance |
| Kopper and Epperson (1996) | 705 college students | Cross-sectional | BDI | AES: suppression |
| Krause, Mendelson, and Lynch (2003) | 127 adults from the community | Experimental | BAI; BDI | CSQ: chronic avoidance, current avoidance; WBSI |
| Kuyken and Brewin (1994) | 32 adult female patients with depression and 32 healthy controls | Cross-sectional | DSM-III criteria | WCQ: escape-avoidance, planful problem solving, positive reappraisal |
| Kuyken, Watkins, Holden, and Cook (2006) | 36 children/adolescents from the community with depression and 187 healthy controls; 14–18 years old | Cross-sectional | DSM-IV criteria | RRS |
| Lau, Christensen, Hawley, Gemar, and Segal (2007) | 32 adult patients with anxiety NOS, 43 adult patients with depression, and 36 healthy controls | Experimental | SCID for DSM-IV | RSQ |
| Londahl, Tverskoy, and D'Zurilla (2005) | 123 college students | Cross-sectional | BAI; BDI | SPSI-R: positive problem orientation |
| Luck, Waller, Meyer, Ussher, and Lacey (2006) | 43 adult female patients with anorexia nervosa restrictive type, 50 adult patients with bulimia nervosa, 28 patients with anorexia nervosa binge/purge type, and 345 healthy adult controls | Cross-sectional | DSM-IV criteria | YRAI |
| Luxton, Ingram, and Wenzlaff (2006) | 44 depressed college students and 178 healthy controls | Cross-sectional | Composite BDI + IDD | WBSI |
| Luxton and Wenzlaff (2005) | 78 depressed college students and 121 healthy controls | Cross-sectional | Composite BDI + IDD | WBSI |
| Maltby and Day (2000) | Sample 1: 188 female college students; sample 2: 172 male college students | Cross-sectional | BDI; STAI | RWCC avoidance, problem focus |
| Martin and Dahlen (2005) | 362 college students | Cross-sectional | DASS: anxiety, depression | CERQ: acceptance, positive reappraisal, refocus on planning, rumination |
| Mayhew and Edelmann (1989) | 49 college students | Cross-sectional | EDI: bulimia, drive for thinness | CI: avoidance coping, cognitive coping |
| McCabe, Blankstein, and Mills (1999) | 207 college students | Cross-sectional | CES-D | SPSI-R: positive problem orientation |
| McLean, Miller, and Hope (2007) | 160 female college students | Cross-sectional | BDI; EAT; SIAS | ERQ: reappraisal, suppression |
| Milligan and Waller (2000) | 83 college students | Cross-sectional | BITE | STAXI: suppression |
| Moulds, Kandris, Starr, and Wong (2007) | 104 college students | Cross-sectional | BAI; BDI | CBAS; RRS: brooding |
| Moulds, Kandris, and Williams (2007) | 93 college students | Experimental | BAI; BDI | RRS |
| Nezu (1986) | Sample 1: 37 depressed college students and 38 healthy controls; sample 2: 25 depressed adult patients and 21 healthy controls | Cross-sectional | BDI; SADS | PSI: personal control, confidence, approach-avoidance |

(*continued on next page*)

**Table 1** (*continued*)

| Author name | Sample details/moderators | Design | Psychopathology measures | Emotion-regulation measures |
|---|---|---|---|---|
| Nezu, Nezu, Saraydarian, Kalmar, and Ronan (1986) | 462 college students | Cross-sectional | BDI | PSI |
| Papadakis, Prince, Jones, and Strauman (2006) | 223 female children/adolescents; 7th–12th graders | Cross-sectional | CDI | RSQ: brooding |
| Paxton and Diggens (1997) | 61 college students with eating restraint, 15 college students with bulimia nervosa, and 73 healthy controls | Cross-sectional | DEBQ-R; BUILIT binge | WCQ: emotional avoidance, problem solving, positive reappraisal |
| Peirson and Heuchert (2001) | 471 college students | Cross-sectional | BDI | TCI: harm avoidance |
| Perini, Abbott, and Rapee (2006) | 40 adult patients with social anxiety and 20 healthy controls | Experimental | ADIS for DSM-IV | RRS; WBSI |
| Piran and Cormier (2005) | 394 female adult community members | Cross-sectional | EAT; EDI: bulimia, drive for thinness | STAXI: anger-in |
| Rimes and Watkins (2005) | 30 adult patients with depression and 30 healthy controls | Experimental | SCID for DSM-IV | RRS |
| Riso et al. (2003) | 42 depressed adult patients and 24 healthy controls | Cross-sectional | SCID for DSM-IV | RSQ: rumination |
| Roberts et al. (1998) | Sample 1: 13 depressed college students and 19 healthy controls; sample 2: 8 depressed college students and 175 healthy controls; sample 3: 16 depressed college students and 181 healthy controls | Cross-sectional | IDD | RRS |
| Rude (2007) | 232 college students | Cross-sectional | BDI; STAI; ZDS | RRS: brooding |
| Rude, Wenzlaff, Gibbs, Vane, and Whitney (2002) | 399 college students | Experimental | BDI; IDD | WBSI |
| Rudolph, Flett, and Hewitt (2007) | 100 college students | Cross-sectional | CES-D | CERQ: acceptance, positive reappraisal, refocus on planning, rumination |
| Rudolph, Hammen and Burge (1994) | 57 children/adolescents from the community with depression and 61 healthy controls; 7–13 years old | Experimental | CDI | IPSQ: hostile, passive, sociable |
| Santanello and Gardner (2007) | 125 college students | Cross-sectional | BDI | AAQ |
| Schwarze, Oliver, and Handal (2003) | 43 adult female patients with binge eating and 164 healthy controls | Cross-sectional | Q-EDD | CISS: avoidance; WCQ: escape-avoidance |
| Siegle, Moore, and Thase (2004) | 349 college students | Cross-sectional | BDI | ROS; RRQ: rumination; SMRI: motivation |
| Sigmon et al. (2007) | 17 adult patients with depression and 17 healthy controls | Experimental | SCID for DSM-IV | COPE: acceptance, problem focused |
| Soukup, Beiler, and Terrell (1990) | 12 adult patients with anorexia nervosa, 33 with bulimia nervosa, and 26 healthy controls | Cross-sectional | DSM-III criteria | PSI |
| Sperberg and Stabb (1998) | 234 female college students | Cross-sectional | BDI | STAXI: anger-in |
| Stewart, Zvolensky, and Eifert (2002) | 182 college students | Cross-sectional | ASI | EAS |
| Tull and Gratz (2008) | 53 depressed college students and 53 healthy controls | Cross-sectional | DASS: depression | AAQ |

| | | | | |
|---|---|---|---|---|
| Verhaeghen, Joormann, and Khan (2005) | 99 college students | Cross-sectional | CES-D | RRS |
| Vickers and Vogeltanz-Holm (2003) | 84 college students with depression and 86 healthy controls | Experimental | BDI | RRS |
| Waller et al. (2003) | 20 adult female patients with anorexia nervosa restrictive type, 68 with bulimia nervosa, 39 with anorexia nervosa binge purge type, 13 with binge eating disorder, and 50 healthy controls | Cross-sectional | DSM-IV criteria | STAXI: suppression |
| Watkins (2004) | 140 college students | Cross-sectional | HADS: anxiety, depression | RSQ |
| Watkins and Baracaia (2002) | 32 adult patients with depression and 26 healthy controls | Experimental | SCID for DSM-III | RRS |
| Watkins and Brown (2002) | 14 adult patients with depression and 14 healthy controls | Experimental | DSM-III criteria | RRS |
| Wegner and Zanakos (1994) | Sample 1: 609 college students; sample 2: 405 college students; sample 3: 490 college students; sample 4: 199 college students; sample 5: 133 college students | Cross-sectional | ASI; BDI; MOCDI; STAI | WBSI |
| Weinstock and Whisman (2007) | 244 college students | Cross-sectional | BDI | RRS |
| Wenzlaff, Rude, and West (2002) | 22 college students with depression and 400 healthy controls | Cross-sectional | DSM-IV; IDD-L; BDI | WBSI |
| Wupperman and Neumann (2006) | 589 college students | Cross-sectional | CES-D | ROS; RRQ: rumination |
| Ziegert and Kistner (2002) | 205 children/adolescents; 4th–5th graders | Cross-sectional | CDI | CRSS: rumination |

Notes: AAQ: Acceptance and Action Questionnaire; ADIS for DSM-IV: Anxiety Disorders Interview Schedule for DSM-IV; AEI: Anger Expression Inventory; AES: Anger Expression Scale; ASI: Anxiety Sensitivity Index; BAI: Beck Anxiety Inventory; BARQ: Behavioral Anger Response Questionnaire; BDI: Beck Depression Inventory; BES: Binge Eating Scale; BITE: Bulimic Investigatory Test Edinburgh; BSQ: Body Sensations Questionnaire; BULIT: Bulimia Test; CBAS: Cognitive Behavioral Avoidance Scale; CDI: Children Depression Inventory; CERQ: Cognitive Emotion Regulation Questionnaire; CES-D: Center for Epidemiologic Studies Depression Scale; CHIP: Coping with Health Injuries and Problems; CI: Coping Index; CISS: Coping Inventory for Stressful Situations; COPE: Cope Inventory; CRSS: Children's Response Styles Scale; CSI: Coping Strategy Inventory; CSQ: Coping Styles Questionnaire; DASS: Depression and Anxiety Scales; DEBQ-R: Dutch Eating Behaviour Questionnaire; DERS: Difficulties with Emotion Regulation Questionnaire; DSM: Diagnostic and Statistical Manual of Mental Disorders; EACQ: Emotional Approach Coping Questionnaire; EAS: Experiential Avoidance Scale; EAT: Eating Attitudes Test; ECQ: Emotional Control Questionnaire; EDE: Eating Disorder Examination; EDI: Eating Disorders Inventory; EMAS: Endler Multidimensional Anxiety Scales; ERQ: Emotion Regulation Questionnaire; HADS: Hospital Anxiety and Depression Scale; IDD: Inventory to Diagnose Depression; IPSQ: Interpersonal Problem Solving Questionnaire; KAT: Khavari alcohol test; MASQ: Mood and Anxiety Symptom Questionnaire; MOCDI: Maudsley Obsessive–Compulsive Inventory; PAES-III: Pediatric Anger Expression Scale III; PSI: Problem-Solving Inventory; Q-EDD: Questionnaire for Eating Disorder Diagnoses; R-CMAS: Children's Manifest Anxiety Scale — Revised; RI: Rumination Inventory; ROS: Rumination on Sadness; RRQ: Rumination Reflection Questionnaire; RRS: Ruminative Response Scale; RSQ: Response Styles Questionnaire; RWOCC: Revised Ways of Coping Checklist; SADS: Schedule for Affective Disorders and Schizophrenia; SCID-IV: Structured Clinical Interview Diagnosis for DSM-IV; SCID-III-R: Structured Clinical Interview Diagnosis for DSM-III-R; SFS; Suffocation Fear Scale; SIAS: Social Interaction Anxiety Scale; SMRI: Scott–McIntosh Rumination Index; SPSI: Social Problem-Solving Inventory; STAI: Spielberg State–Trait Anxiety Inventory; STAXI: State–Trait Anger Expression Inventory; STAXI-2: State–Trait Anger Expression Inventory 2; STSS: Silencing the Self Scale; TCI: Temperament and Character Inventory; WBSI: White Bear Suppression Inventory; WCQ: Ways of Coping Questionnaire; YRAI: Young–Rygh Avoidance Inventory; ZDS: Zung Self-Rating Depression Scale.

**Table 2**
Study characteristics — longitudinal design.

| Author name | Sample details/moderators | Psychopathology measures | Emotion-regulation measures | Duration of follow-up | Summary of longitudinal results |
|---|---|---|---|---|---|
| Beevers and Meyer (2004) | 144 college students | MASQ: anhedonic depression, general distress depressive symptoms | WBSI | 7 weeks | Among those with low life stress, high thought suppression predicted lower depression over time. |
| Blalock and Joiner (2000) | 179 college students | BAI, BDI | CRI: cognitive avoidance | 3 weeks | Among those with higher life stress, higher cognitive avoidance predicted increases in both depression and anxiety for women only; no effects for behavioral avoidance. |
| Broderick and Korteland (2004) | 70 children/adolescents 4–6th graders | CDI | RSQ: 10 rumination items | Once per year for 3 years | Rumination predicted increases in depression. |
| Burwell and Shirk (2007) | 168 children/adolescents 8th graders | CDI; CDRS-R, MCC | RRS adapted for children | 1 year | Brooding predicted increases in depression, but not any other depression measures; reflection was not associated with change in any depression scores. |
| Butler and Nolen-Hoeksema (1994) | 199 college students | BDI | RRS:10 items | 2 weeks | Rumination predicted increases in depression, controlling for distraction and gender. |
| Calmes and Roberts (2007) | 543 college students | BAI, BDI | RRS | 6–8 weeks | Structural model showed significant paths from rumination to anxiety (controlling for initial anxiety and worry) but not to depression (controlling for initial depression and worry). |
| Ciarrochi and Scott (2006) | 163 college students | DASS: anxiety, depression | ECQ: rumination; SPSI: problem orientation | 1 year | Problem solving predicted increases in anxiety and depression. |
| Cooper et al. (2003) | 1978 children/adolescents, 13 to 19 years old | BSI: anxiety, depression | Avoidance composite from AES and HDL | 4.5 years | Avoidance coping predicted increases in problem behavior composite, but not in substance use when considered individually. |
| Engler et al. (2006) | 26 female college students who reported binge eating and 199 who did not | LHQ-R: binge eating | WCQ: escape-avoidance | 1 year | Avoidance predicted binge eating over time. |
| Gerard and Buehler (2004) | 5071 children/adolescents, 11–18 years old | CES-D | Problem-solving orientation assessed with 4 items | 1 year | Problem-solving orientation did not predict changes in depression scores, controlling for a number of other risk variables. |
| Grabe et al. (2007) | 299 children/adolescents, 11–13 years old | CDI; OBC-Y: body shame | RSQ: 5 rumination items | 2 years | Rumination predicted increases in depression, controlling for body shame and self-surveillance. |
| Hankin et al. (2005) | 210 college students | BDI | RRS | 35 days | Rumination did not predict daily depressive symptoms. |
| Holahan et al. (2005) | 1221 late–middle-aged adult community member | HDL: depressive features, depressive mood | CRI: cognitive avoidance | 10 years | Avoidance coping at predicted increases in depressive symptoms. |
| Hong (2007) | 241 college students | MASQ: anhedonic depression, anxious arousal, general distress anxious symptoms, general distress depressive symptoms | COPE: problem solving; RRS: 10 items | 1 month | Rumination predicted depression controlling for baseline worry and anxiety, but did not predict anxiety when controlling for baseline worry and depression. |

| | | | | | |
|---|---|---|---|---|---|
| Kashdan and Breen (2008) | 148 college students | BDI; SIAS | ERQ: suppression | 3 months | Suppression at baseline predicted social anxiety symptoms and lower level of positive emotions at follow-up. |
| Matheson and Anisman (2003) | 177 college students | BAI BDI | SCOPE: cognitive restructuring, emotional expression, problem solving, rumination subscale | 6 months | Increases in depression associated with low problem solving and high emotional containment and rumination. |
| Nezu and Ronan (1988) | 150 college students | BDI | PSI | 3 months | Low problem solving on both measures predicted increases in depression. |
| Nolen-Hoeksema (2000) | 1122 adult community members | BAI; composite BDI + HRSD | RRS | 1 year | Rumination predicted increases in depression. |
| Nolen-Hoeksema and Harrell (2002) | Sample 1: 599 female adult community members; sample 2: 523 male adult community members | DSM-IV criteria | RRS | 1 year | Rumination predicted increases in alcohol abuse problems in women but not men. |
| Nolen-Hoeksema et al. (2007) | 478 female children/ adolescents, 14–17 years old at time 1 | DSM-IV criteria, substance abuse items from Stice et al. (1998); EDE: bulimic symptoms; SADS for school-aged children | RRS: 6 items | Measures taken each year for 4 years | Lagged models showed rumination predicted increases in depression, substance abuse, and eating disorder symptoms; girls with higher rumination were more likely to show onsets of MDE, and to meet diagnostic criteria for bulimia nervosa. |
| O'Connor et al. (2007) | Sample 1: 224 adult community members; sample 2: 279 college students | CES-D; GHQ: anxiety, depression | RRS:10 items | 8 weeks | Brooding predicted increases in depression after controlling for social perfectionism. |
| Priester and Clum (1993) | 303 college students | BDI | PSI | 1–2 weeks | Low problem-solving confidence interacted with poor test scores to predict increases in depression |
| Sarin et al. (2005) | 86 college students | MASQ: anhedonic depression, anxious arousal, general distress anxious symptoms, general distress depressive symptoms | RRS | Immediately after exam, 4–8 h later, and 4 days later | Rumination predicted increases in depression and mixed anxiety/depression at 4 days post-exam only; rumination predicted anxiety 4–8 h and 4 days after exam. |
| Segerstrom et al. (2000) | 110 college students | BDI; CCL: anxiety, depression | GRS; RDS: rumination modified: 11 items | 1 week | Full scale rumination predicted increases in depression but not anxiety when controlling for worry; short form of rumination did not predict increases in depression or anxiety when controlling for worry. |
| Wenzlaff and Luxton (2003) | 225 college students | BDI | RS: 10 items; WBSI | 10 weeks | Suppression predicted increases in depression but rumination did not. |

Notes: AES: Anger Expression Scale; BAI: Beak Anxiety Inventory; BDI: Beck Depression Inventory; BSI: Brief Symptom Inventory; CCL: Cognitive Checklist; CDI: Children Depression Inventory; CDRS-R: Children's Depression Rating Scale – Revised; CES-D: Center for Epidemiologic Studies Depression Scale; COPE: Cope Inventory; CRI: Cognitive Responses Inventory; DASS: Depression and Anxiety Scales; DSM: Diagnostic and Statistical Manual of Mental Disorders; ECQ: Emotional Control Questionnaire; EDE: Eating Disorders Examination; ERQ: Emotion Regulation Questionnaire; GHQ: General Health Questionnaire; GRS: Global Rumination Scale; HDL: Health and Living Daily Form; HRSD: Hamilton Rating Scale for Depression; LHQ-R: Life History Questionnaire – Revised; MASQ: Mood and Anxiety Symptoms Questionnaire; MCC: Mood and Conduct Checklist; OBC-Y: Objectified Body Consciousness Scale for Youth; PSI: Problem-Solving Inventory; RDS: Responses to Depression Scale; RRS: Ruminative Responses Scale; RS: Rumination Scale; RSQ: Response Styles Questionnaire; SADS: Schedule for Affective Disorders and Schizophrenia; SCOPE: Survey of Coping Profile Endorsement; SIAS: Social Interaction Anxiety Scale; SPSI: Social Problem-Solving Inventory; WBSI: White Bear Suppression Inventory; WCQ: Ways of Coping Questionnaire.

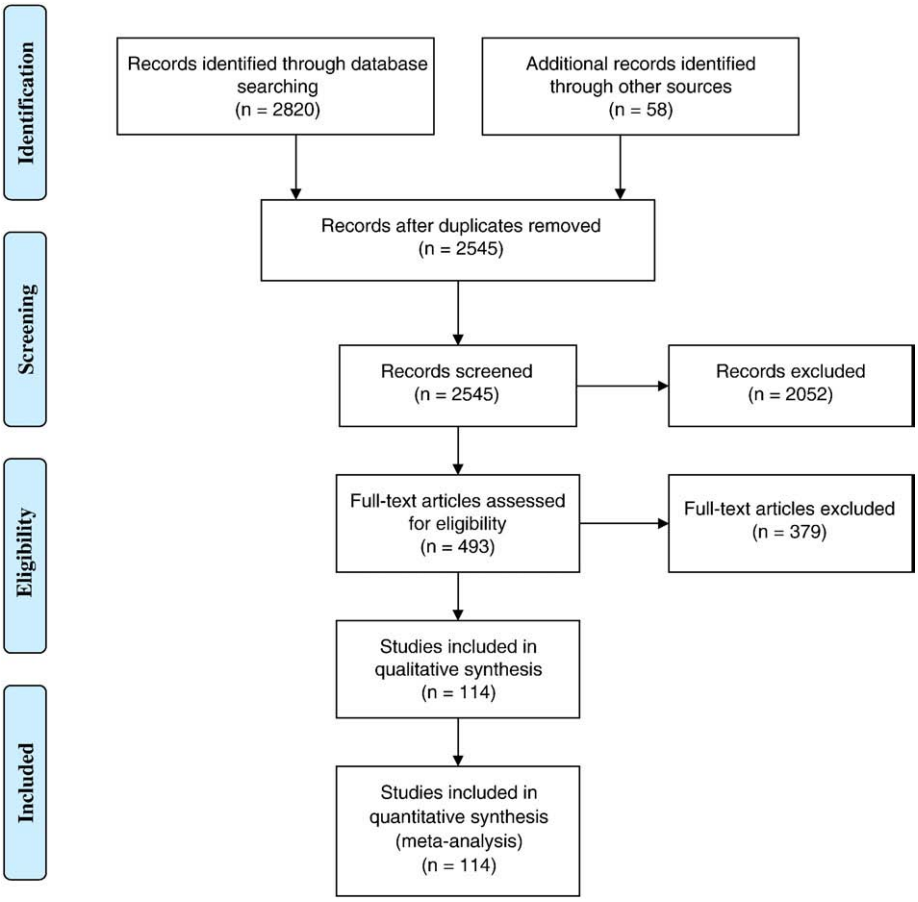


**Fig. 1.** Information flow on study selection.

On the other hand, the extent to which individuals can accurately self-report on their emotion-regulation strategies can be questioned (Robinson & Clore, 2002). For example, such reports may require more insight and meta-cognition than individuals are capable of. These reports may also be influenced by negative moods or self-presentation biases. Self-reports of emotion regulation may confound the experience of emotion with its regulation (Cole et al., 2004). Finally, some measures of emotion regulation may have substantial content overlap with psychopathology criterion measures (Treynor, Gonzalez, & Nolen-Hoeksema, 2003). We return to these measurement issues in the Discussion section.

Some researchers have used observational methods to study emotion regulation, usually by instructing participants to engage in a particular emotion-regulation strategy in response to an emotion-eliciting stimulus and then observing the effects on participants' subsequent emotions, cognitions, or physiological responding (e.g., Gross, 1998; Gross & John, 2003; Nolen-Hoeksema et al., 2008). Although these studies are invaluable in testing hypotheses about the short-term effects of emotion-regulation strategies, they do not assess whether the tendency to engage in certain emotion-regulation strategies is associated with clinically significant levels of psychopathology. In this respect, recent evidence suggests that when some participants are instructed to use specific strategies, they have a hard time doing so (Demaree, Robinson, Pu, & Allen, 2006). In addition, these studies have typically focused only on one or two emotion-regulation strategies at a time (usually either expressive or thought suppression, reappraisal, or rumination), and are highly heterogeneous in design and outcome variables, making it difficult to compare findings across them. Furthermore, reviews of these experimental studies are available elsewhere: Gross and Thompson (2007) review studies of the effects of experimental manipulations of reappraisal and expressive suppression on emotion and physiology. Extensive reviews and meta-analyses of experimental manipulations of rumination on depression or anxiety are provided by Mor and Winquist (2002) and Watkins (2008). Effects of experimental manipulations of thought suppression on subsequent thought frequency in clinical and non-clinical samples are reviewed narratively by Purdon (1999) and Rassin, Merckelbach, and Muris (2000) and in a meta-analysis by Abramowitz, Tolin, and Street (2001). Thus, the present meta-analysis will focus only on self-report measures of dispositional tendencies toward specific emotion-regulation strategies.

## 3. Methods

### 3.1. Literature searches

We searched for studies that provided data on at least one of the disorders and one of the regulatory strategies of interest, regardless of whether the study of these constructs was a central focus of the study. We conducted systematic searches for articles published between 1985 and July 2008 using PsycInfo and Medline. We chose 1985 as a beginning point because, with the exception of some studies of problem solving and avoidance coping, there was little work on what is now referred to as emotion regulation prior to the mid-1980s. Also, many of the emotion-regulation measures that are widely used now did not exist prior to the mid-80s, and as the lists of studies in Tables 1 and 2 indicate, the vast majority of the work on emotion regulation has been done since the mid-1990s. We searched with keywords for every combination of individual emotion-regulation strategy and type of psychopathology. Specifically, we searched for combinations of these terms across all fields (i.e., title, abstract, keywords): acceptance, avoidance, reappraisal, problem solving, rumination, suppression, emotion, regulation,

emotion regulation, mindfulness, depression, anxiety, eating, binge, anorexia, bulimia, alcohol, and substance. We searched for truncated versions of these terms. In addition, we searched for authors who have published substantially in the topics of interest. We supplemented our searches by looking for articles in Google Scholar. In addition to the online databases, we checked reference sections of published articles, examined tables of contents of relevant journals, consulted with colleagues, and contacted authors in the field in order to obtain information regarding the existence of studies we might have left out. Two of the authors conducted independent searches that yielded similar results. We organized these references and identified duplicates using Endnote X1. Fig. 1 shows this search process in more detail.

#### 3.1.1. Inclusion/exclusion criteria for the study

We included a study if it reported at least one cross-sectional relationship between an emotion-regulation strategy and one of the disorders of interest regardless of their specific aims. For example, we included studies that focused on the experimental manipulation of emotional states (e.g., mood induction paradigms) or on scale development as long as they provided baseline data for the relationship between dispositional emotion regulation and psychopathology. When studies measured psychopathology dichotomously either by using a diagnostic interview or cutoff in a continuous self-report measures, we only included the study if it provided an appropriate control group that would allow us to compute mean difference effect sizes. In terms of populations, we included studies recruiting adults and children, as well as from various demographic groups, such as college students, community residents, and clinical patients (in most cases, outpatients).

Emotion regulation was assessed via self-report questionnaires and psychopathology was assessed via self-report questionnaires or diagnostic interviews. Most studies provided data on more than one relationship between regulation strategy and disorder. Additionally, for each relationship, studies tended to provide data from various measures, thus resulting in several effect sizes per relationship per sample. Because utilizing multiple effect sizes from a sample results in a violation of the assumption of independence of meta-analyses (Lipsey & Wilson, 2001), we sought to correct for this problem. First, we defined each combination of emotion-regulation strategy and disorder as a construct (see Augustine & Hemenover, 2008; Pole, 2007; Thomas, Vartanian, & Brownell, 2009). Second, for each of these constructs, we calculated one effect size by averaging across the data provided by that study on that construct (Rosenthal & DiMatteo, 2001). We chose this approach over randomly selecting data in order to avoid losing information.

If a study did not report data that had been collected or presented in a form that we could use, we contacted the authors. Twenty-four authors provided data for 11 studies. The remaining authors indicated that too much time had elapsed since the time of data collection and thus data were no longer available.

We utilized several exclusion criteria. First, we excluded dissertations, master's theses, or conference presentations because restricting our analyses to studies published in peer-reviewed journals increased the likelihood that studies would be of acceptable quality. Also, most studies of interest included in this meta-analyses administered several measures and reported many correlations, including many non-significant ones, thus reducing the concern for file-drawer effects. This decision is consistent with several recent meta-analytic reviews that have not included dissertations, master's theses, or conference presentations (e.g., Hagedoorn, Sanderman, Bolks, Tuinstra, & Coyne, 2008; Kurtz & Mueser, 2008; Papadatou-Pastou, Martin, Munafo, & Jones, 2008; Pole, 2007; Tarbox & Pogue-Geile, 2008). Second, we excluded studies that recruited non-English speaking populations because the validity of most translated measures of emotion regulation in other cultures has not been established. Third, we excluded studies that recruited the following populations: diagnosed with medical conditions (e.g., cancer, HIV, cardiac problems), special groups (e.g., caregivers, bereaved individuals, traumatized individuals) in order to increase the representativeness of the samples included in the meta-analysis. Fourth, for similar reasons, we excluded studies that preselected individuals based on variables other than clinical diagnosis (e.g., cognitive factors). Fifth, we excluded studies with clinical samples consisting only of recovered patients (as opposed to patients with a current diagnosis) because we wanted to maximize differences between those with and without psychopathology and because most emotion-regulation theories do not make predictions about differences between recovered patients (who may have received psychotherapy that changed their emotion-regulation strategies) and other groups. Sixth, we excluded treatment studies because they did not have a healthy control group that would allow us to draw comparisons with the psychopathology groups. Seventh, we excluded the few studies that only provided data on generalized anxiety disorder (GAD) in order to minimize contamination between worry and rumination (Watkins, Moulds, & Mackintosh, 2005). Eighth, we excluded studies that only provided data on Posttraumatic Stress Disorder (PTSD), which had a lot of variability in the type of trauma and the time elapsed from trauma until assessment. Ninth, we excluded the longitudinal portion of longitudinal studies since there was much variability in the length of time that elapsed between assessments, although we did use baseline data from these studies. Following the Results section, we provide a qualitative review of the longitudinal findings.

Applying these exclusion criteria resulted in 114 studies being retained (71 cross-sectional, 18 experimental, and 25 longitudinal). Of the cross-sectional studies, 54 provided correlations and 17 provided mean difference data. Of the experimental studies, 6 provided correlational data and 12 provided mean differences data. Of the longitudinal studies, 24 provided correlational data and 1 provided mean difference data. The 114 included studies provided a total of 241 effect sizes (198 from correlations, and 43 from mean differences). For more information see Tables 1 and 2.

### 3.2. Coding procedures

The first author and two research assistants coded studies independently and achieved adequate levels of agreement, with kappa coefficients ranging from .83 to 1 (Landis & Koch, 1977). We coded the following potential moderator variables: sample type (clinical or non-clinical) and sample age group (children/adolescents versus adults). Children and adolescents were recruited from either schools or the community, so none of them was recruited from clinical populations. In terms of dependent variables, we coded information on the emotion-regulation strategies measures (acceptance, avoidance, reappraisal, rumination, problem solving, and suppression) and the disorder measures (anxiety, depression, eating, or substance). If a study provided several subscales for a measure, we included all of them and then averaged across all subscales for each combination of strategy and disorder. When studies provided continuous data for a combination of strategy and disorder, we extracted relevant data to calculate effect sizes of the r-family. When necessary, we reversed coded correlation coefficients, so that positive scores would indicate a stronger use of the strategy. When studies provided dichotomous data, such as scores for an emotion-regulation strategy in a clinical and control group, we extracted relevant data to calculate effect sizes of the d-family (Lipsey & Wilson, 2001). If a study administered continuous measures and also used a cutoff to dichotomize the sample, we favored the continuous (i.e. correlational) over the dichotomized data, because splitting a variable into categories results in loss of information and might increase the probability of type II errors (Altman & Royston, 2006; Rosenthal & DiMatteo, 2001; Streiner, 2002).

**Table 3**
Emotion-regulation strategies across psychopathology groups.

| Emotion-regulation strategy | Mean | 95% CI | 95% CI | *p*-value | *k* | *Q*-statistics *p*-value |
|---|---|---|---|---|---|---|
| Acceptance | −.19 | −.40 | .05 | ns | 7 | <.001 |
| Avoidance | .38 | .33 | .44 | <.001 | 37 | <.001 |
| Problem solving | −.31 | −.36 | −.25 | <.001 | 42 | <.001 |
| Reappraisal | −.14 | −.20 | −.07 | <.001 | 15 | <.001 |
| Rumination | .49 | .45 | .52 | <.001 | 89 | <.001 |
| Suppression | .34 | .28 | .39 | <.001 | 51 | <.001 |

### 3.3. Effect size calculation and corrections

Because the coded studies provided effect sizes of the r- and d-families, we put them in the same metric for the purposes of our analyses. Rosenthal and DiMatteo (2001) suggest converging d's to r's given that r's are more easily interpreted in terms of practical importance. Additionally, converting continuous r into dichotomous d results in loss of information. Thus, when studies provided effect sizes of the d-family, we first calculated Cohen's *D* standardized mean difference by subtracting the means of control group from those of the clinical group and then dividing this difference by the pooled variance (Cohen, 1988). Then, we transformed Cohen's *D* into an effect size of the r-family, the correlation coefficient *r* (Rosenthal, & DiMatteo, 2001). Once all the effect sizes were on the r-metric, we calculated the appropriate corrections. Correlation coefficients have a problematic standard error formulation (Lipsey & Wilson, 2001), which can be solved by using the Fisher's Zr-transform (Hedges & Olkin, 1985; Lipsey & Wilson, 2001), so we applied this transformation. Given that larger samples are more precise, we also corrected for sample size by multiplying the effect sizes by their inverse variances (Hedges & Olkin, 1985; Hedges & Vevea, 1998; Lipsey & Wilson, 2001). We then back transformed the Fisher's *z* coefficients to raw correlation coefficients for ease of interpretation. We utilized Cohen's guidelines (1988) to interpret correlation effect sizes: above .40 as large, around .25 as medium, and below .10 as small.

### 3.4. Data analytic plan

#### 3.4.1. Random-effect models

We assumed that the effect sizes included in the meta-analyses were sampled from a universe of possible effect sizes, and as such we make unconditional inferences to generalize beyond them (Hedges & Vevea, 1998; Hunter & Schmidt, 1990). In order to model these unconditional inferences, we ran random-effect models, as they assume that effect sizes differ from the population by sampling error plus random variability among the studies (Field, 2003; Lipsey & Wilson, 2001; Rosenthal & DiMatteo, 2001). Another advantage of using random-effect models is that because they model variability from both between- and within-studies, they tend to produce larger standard errors, which means they reduce the risk of type I errors and are thus more conservative (for a review of the risks resulting from failing to use random-effect models when appropriate, see Field, 2003).

According to Hedges and Vevea (1998), using less than five effect sizes might result in random-effect tests that can only be regarded as approximate. However, for many combinations of regulatory strategy and disorder less than five effect sizes were found in the literature. Our best solution to address this problem was to still run the models with two, three, and four effect sizes. However, we do suggest caution in the interpretation of these effect sizes. Additionally, in order to address the low power in analyses with such few observations, when evaluating our results we took two additional steps: we included relationships that were marginally significant ($p<.10$) and we pointed at the magnitude of their effect sizes.

#### 3.4.2. Moderator analyses

We were interested in the proportion of the variance in the association between emotion-regulation strategy and psychopathology that is accounted for by sample type or age group among the reviewed studies. We focused on these two moderators because they can introduce substantial variability in assessments of psychopathology. First, we were interested in evaluating sample type as a potential moderator, given that individuals from a clinical population would be expected to have higher levels of psychopathology than those from a non-clinical population. In this respect, we were curious to see whether the relationship between emotion regulation and psychopathology was greater when more clinically severe participants were included in the analyses.

Second, emotion regulation is considered key to mental health in children, but the ability to regulate emotions develops over childhood and adolescence (Eisenberg, Spinrad, & Eggum, in press). Thus, the relationships between emotion regulation and psychopathology may be less strong in children than in adults. We evaluated whether age influenced the relationship between emotion-regulation strategies and psychopathology.

In order to examine moderators, we first calculated the *Q* statistics (Hedges & Olkin, 1985) to determine the degree of heterogeneity among our effect sizes. However, many researchers find this metric to be problematic as it tends to be overly sensitive to detecting heterogeneity in larger samples (Hunter & Schmidt, 1990). In the case of this investigation we ran the risk of not detecting significant heterogeneity, given the small sample size of many of our combinations of strategies and disorders. For this reason, in addition to calculating the *Q* statistics, we decided to follow the suggestions of Rosenthal and DiMatteo (2001) to evaluate our moderators even in the absence of a significant *Q*.[3] We ran these analyses using SPSS version 13.0 for Mac (SPSS Inc, Chicago, Illinois, USA) and the macros written by Lipsey and Wilson (2001). Similarly to our approach with the random-effect models, we ran mixed effects analyses in which there were at least 2 effect sizes per level.

## 4. Results

### 4.1. Regulation strategies across disorders

We calculated the random-effect model of the correlation coefficients for each regulatory strategy collapsed across the disorder clusters (see Table 3). As predicted, the following strategies were positively associated with psychopathology: avoidance ($r=.38$; $k=37$; 95% CI = [.33; .44]) and suppression ($r=.34$; $k=51$; 95% CI = [.28; .39]), both with magnitudes from medium to large, and rumination with a large magnitude ($r=.49$; $k=89$; 95% CI = [.45; .52]). Conversely, the following strategies were negatively associated with psychopathology: problem solving, with a medium to large effect size ($r=-.31$; $k=42$; 95% CI = [−.36; −.25]), and reappraisal, with a small to medium effect size ($r=-.14$; $k=15$; 95% CI = [−.20; −.07]). Surprisingly, acceptance was not significantly associated with psychopathology ($r=-.19$; $k=7$; 95% CI = [−.40; .05]), although it is worth mentioning that this effect is small to medium in magnitude and in the predicted direction.

[3] This was only the case for eating and avoidance and sample type as the moderator.

**Table 4**
Emotion-regulation strategies and psychopathology groups.

| Emotion-regulation strategy | Psychopathology | Mean | 95% CI | 95% CI | *p*-value | *k* | *Q*-statistics *p*-value |
|---|---|---|---|---|---|---|---|
| Acceptance | Anxiety | −.25 | −.70 | .35 | .42 | 2 | <.001 |
| Acceptance | Depression | −.20 | −.52 | .16 | .27 | 4 | <.001 |
| Acceptance | Substance | .00 | | | | 1 | |
| Avoidance | Anxiety | .37 | .26 | .48 | <.001 | 13 | <.001 |
| Avoidance | Depression | .48 | .40 | .55 | <.001 | 16 | <.001 |
| Avoidance | Eating | .18 | .13 | .24 | <.001 | 7 | .91 |
| Avoidance | Substance | .26 | | | | 1 | |
| Problem solving | Anxiety | −.27 | −.36 | .16 | <.001 | 14 | <.001 |
| Problem solving | Depression | −.33 | −.40 | −.26 | <.001 | 26 | <.001 |
| Problem solving | Eating | −.29 | −.53 | −.01 | <.05 | 2 | .08 |
| Reappraisal | Anxiety | −.13 | −.28 | .02 | .09 | 5 | <.01 |
| Reappraisal | Depression | −.17 | −.29 | −.06 | <.05 | 7 | <.01 |
| Reappraisal | Eating | −.05 | −.21 | .1 | .5 | 2 | .56 |
| Reappraisal | Substance | −.08 | | | | 1 | |
| Rumination | Anxiety | .42 | .37 | .48 | <.001 | 23 | <.001 |
| Rumination | Depression | .55 | .51 | .59 | <.001 | 56 | <.001 |
| Rumination | Eating | .26 | .20 | .32 | <.001 | 3 | .87 |
| Rumination | Substance | .21 | .11 | .31 | <.001 | 7 | <.001 |
| Suppression | Anxiety | .29 | .19 | .39 | <.001 | 19 | <.001 |
| Suppression | Depression | .36 | .29 | .43 | <.001 | 26 | <.001 |
| Suppression | Eating | .36 | .24 | .47 | <.001 | 6 | <.05 |

As Table 3 shows, all these effects but acceptance had significant *Q* statistics, suggesting important variability. We then conducted moderator analyses by running mixed models. In the first mixed model, we evaluated the role of sample type in moderating avoidance, problem solving, rumination, and suppression, since they all had at least two effect sizes per level of this variable. Sample type significantly moderated rumination ($Q$ (1) = 24. 54, $p$<.001) with studies including clinical participants showing larger effect sizes ($r$ = .87; $k$ = 11; 95% CI [.73, 1]) than studies including only non-clinical participants ($r$ = .49; $k$ = 78; 95% CI [.44, .54]). It also moderated suppression ($Q$ (1) = 6.76, $p$<.01), with studies including clinical participants having larger effect sizes ($r$ = .68; $k$ = 4; 95% CI [.42, .93]) than those without clinical participants ($r$ = .33; $k$ = 47; 95% CI [.26, .40]). However, sample type was only a marginally significant moderator of avoidance ($Q$ (1) = 3.21, $p$ = .07), with studies including clinical participants having larger effect sizes ($r$ = .57; $k$ = 6; 95% CI [.38; .76]) than those without clinical participants ($r$ = .38; $k$ = 31; 95% CI [.30; .46]) and problem solving ($Q$ (1) = 2.94, $p$ = .09), with studies including clinical participants having larger effect sizes ($r$ = −.53; $k$ = 4; 95% CI [−.78, −.28]) than those without clinical participants ($r$ = −.31; $k$ = 38; 95% CI [−.38, −.24]).

In the second mixed model, we evaluated the role of age group as a moderator of problem solving, rumination, and suppression, since these were the only strategies with more than two effect sizes in each level of this variable. Age group did account for a significant portion of the variability in problem solving ($Q$ (1) = 4, $p$<.05) with adults having a stronger relationship ($r$ = −.34; $k$ = 40; 95% CI [. −.41; −.27]) than children/adolescents ($r$ = −.03; $k$ = 2; 95% CI [−.32; .26]) and of suppression ($Q$ (1) = 11. 98, $p$<.001), with adults again having a stronger relationship ($r$ = .38; $k$ = 48; 95% CI = [.32; .44]) than children/adolescents ($r$ = −.08; $k$ = 3; 95% CI = [−.34, .17]). However, age group did not moderate the relationship between rumination and psychopathology ($Q$ (1) = .83, $p$ = .36).

### 4.2. Emotion-regulation strategies in each disorder

We calculated the random-effect model of the correlation coefficients for each combination of regulatory strategy and disorder (e.g., avoidance + anxiety; rumination + depression). As Table 4 shows, avoidance was positively associated with anxiety ($r$ = .37; $k$ = 13; 95% CI [.26, .48]), depression ($r$ = .48; $k$ = 16; 95% CI [.40, .55]), and eating ($r$ = .18; $k$ = 7; 95% CI [.13, .24]); rumination was positively associated with anxiety ($r$ = .42; $k$ = 23; 95% CI [.37, .48]), depression ($r$ = .55; $k$ = 56; 95% CI [.51, .59]), eating ($r$ = .26; $k$ = 3; 95% CI [.20, .32]), and substance ($r$ = .21; $k$ = 7; 95% CI [.11, .31]); and suppression was positively associated with anxiety ($r$ = .29; $k$ = 19; 95% CI [.19, .39]), depression ($r$ = .36; $k$ = 26; 95% CI [.29, .43]), and eating ($r$ = .36; $k$ = 6; 95% CI [.24, .47]). Conversely, problem solving was negatively associated with anxiety ($r$ = −.27; $k$ = 14; 95% CI [−.36, −.16]), depression ($r$ = −.33; $k$ = 26; 95% CI [−.40, −.26]) and eating ($r$ = −.29; $k$ = 2; 95% CI [−.53, −.01]); and reappraisal was marginally negatively associated with anxiety ($r$ = −.13; $k$ = 5; 95% CI [−.28, .02]; $p$ = .09), negatively associated with depression ($r$ = −.17; $k$ = 7; 95% CI [−.29, −.06]), and not associated with eating ($r$ = −.05; $k$ = 2; 95% CI [−.21; .10]). Acceptance was not significantly associated with anxiety ($r$ = −.25; $k$ = 2; 95% CI [−.70, .35]) or depression ($r$ = −.20; $k$ = 4; 95% CI [−.52, .16]). Lastly, there was only one sample providing data to calculate effect sizes for the following combinations of strategies and disorders: acceptance and substance (Britton, 2004; $r$ = 0; $n$ = 196), avoidance and substance (Cooper et al., 2003; $r$ = .26, $n$ = 1978), and reappraisal and substance (Britton, 2004; $r$ = −.08; $n$ = 196).

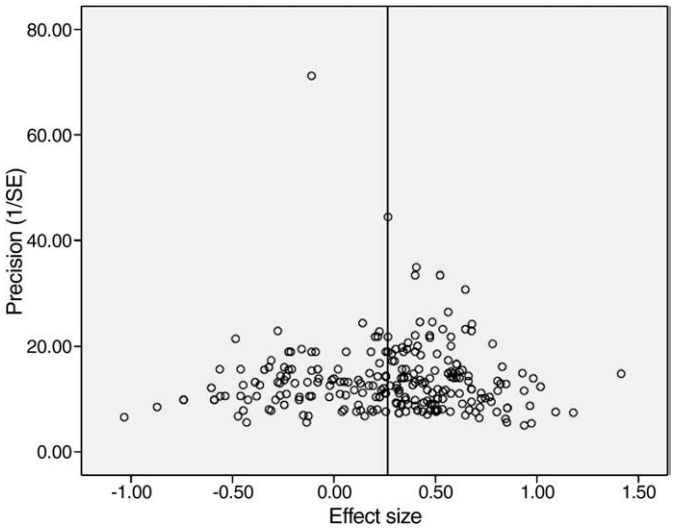


**Fig. 2.** Funnel plot of all effect sizes in this study.

We then conducted moderator analyses for the effect sizes with significant *Q* statistics (see Table 4). We examined age group in the combinations of strategy and disorder that had at least two effect sizes in each level of the moderator variable: problem solving, rumination + depression, and suppression + depression. Age group was a significant moderator of problem solving + depression ($Q$ (1) = 5.81, $p$<.05) with adults showing larger effect sizes ($r$ = −.38; $k$ = 24; 95% CI [−.46, −.30]) than children/adolescents ($r$ = −.04; $k$ = 2; 95% CI [−.30, .23]) and of suppression + depression ($Q$ (1) = .9.27, $p$<.01), with adults having larger effect sizes ($r$ = .42; $k$ = 24; 95% CI [.34, .50]) than children/adolescents ($r$ = −.06; $k$ = 2; 95% CI [−.36, .23]). However, age group was not a significant moderator of rumination + depression ($Q$ (1) = .28, $p$ = .60).

We then examined our second moderator variable, sample type, in the combinations of strategy and disorder with at least two effect sizes in each level of the moderator variable: avoidance + depression; problem solving + depression; rumination + anxiety; rumination + depression; and suppression + eating. Sample type was a significant moderator of avoidance + depression ($Q$ (1) = 4.55, $p$<.05) with studies including clinical participants showing larger effect sizes ($r$ = .77; $k$ = 3; 95% CI [.52, 1]) than those without clinical patients; rumination + anxiety ($Q$ (1) = 8.51, $p$<.01) with studies including clinical patients having a larger effect size ($r$ = .70; $k$ = 3; 95% CI [.52, .86]) than those

without clinical participants ($r = .42$; $k = 20$; 95% CI [.36, .48]); rumination + depression ($Q(1) = 16.60$, $p < .001$), with studies including clinical participants having a larger effect size ($r = .92$; $k = 8$; 95% CI [.77, 1.08]) than those without clinical participants ($r = .57$; $k = 48$; 95% CI [.51, .63]); and suppression + eating, with studies including clinical participants having a larger effect size ($r = .59$; $k = 2$; 95% CI [.42, .77]) than those without clinical participants ($r = .30$; $k = 4$; 95% CI [.23, .37]). However, sample type was not a significant moderator of problem solving + depression ($Q(1) = 2.13$, $p = .15$). Lastly, we examined sample type in avoidance + eating, an effect size with non-significant $Q$ statistics. It was non-significant ($Q(1) = 1.18$, $p = .28$).

It is also important to keep in mind that critics of measures of rumination such as the Response Styles Questionnaire (RSQ) have argued that the items on this measure overlap substantially with symptoms of distress, inflating correlations between rumination and depression and anxiety (e.g., Roberts, Gilboa, & Gotlib, 1998; Segerstrom, Tsao, Alden, & Craske, 2000). To address the item overlap between the rumination scale of the RSQ and depression, Treynor et al. (2003) removed items from the rumination scale that most obviously overlapped with depression (e.g., "I think about my feelings of sadness") and then submitted the remaining 10 items to a factor analysis to determine if these 10 items represented one scale or multiple subscales. This analysis yielded two subscales, brooding (e.g., "I think, 'What am I doing to deserve this?'") and pondering (e.g., "I analyze my personality to try to understand why I am depressed."). The brooding items did not include references to depressive symptoms, but did capture theorists' definitions of rumination as abstract, self-evaluative self-focus (i.e., Nolen-Hoeksema, 1991; Watkins, 2008), while the pondering items capture a less self-evaluative, more problem-solving form of self-reflection. Treynor et al. (2003) found that the brooding subscale showed very similar relationships to depression cross-sectionally and longitudinally as did the full rumination scale, whereas the pondering subscale was less reliably associated with depression (see also Joormann, Dkane, & Gotlib, 2006).

In this meta-analysis, we sought to address this issue empirically, by showing that the correlation coefficients between rumination and psychopathology were comparable regardless of whether we had calculated them using only the brooding subscale from the RSQ, only other rumination scales (i.e., non-RSQ), or all the scales available for this meta-analysis. Indeed, this is what we found. We averaged 27 effect sizes from scales other than the RSQ and found the following relationships: medium to large for rumination and psychopathology ($r = .37$; $k = 27$; 95% CI [.30; .45]), large for rumination and depression ($r = .41$; $k = 15$; 95% CI [.32; .49]), medium to large for rumination and anxiety ($r = .32$; $k = 10$; 95% CI [.27; .36]), and medium for rumination and eating ($r = .23$; $k = 1$) and rumination and substance ($r = .22$; $k = 1$). We then averaged the 19 effect sizes from the brooding subscale of the RSQ and the results paralleled those obtained using non-RSQ rumination scales and all the rumination scales combined. We found the following relationships: medium to large for brooding and psychopathology ($r = .39$; $k = 19$; 95% CI [.33; .45]), large for brooding and depression ($r = .44$; $k = 11$; 95% CI [.38; .50]), medium to large for brooding and anxiety ($r = .38$; $k = 5$; 95% CI [.23; .52]), medium for brooding and eating ($r = .27$; $k = 2$; 95% CI [.20; .33]) and brooding and substance ($r = .2$; $k = 1$).

### 4.3. Publication bias

Meta-analyses are subject to the file-drawer problem (Rosenthal, 1979). In the present study, however, because most effect sizes were obtained from large tables examining correlations between several measures, it is likely that null results were actually reported. However, it is also problematic that for some regulatory strategies (i.e., reappraisal and acceptance), the number of available effect sizes was much smaller. To address presence of publication bias in our sample, we constructed a funnel plot, which is a scatterplot with an index of study size plotted against a measure of effect size (Rothstein, 2007). Larger studies appear at the top and cluster around the mean, whereas smaller studies appear at the bottom and show more dispersion around the mean. If the plot looks symmetric (i.e., a funnel), this indicates that effect sizes hone in on the true mean as the sample size increases. Conversely, if the plot is asymmetric, this suggests the presence of publication bias (Rothstein, 2007). As Fig. 2 shows, we plotted precision (1-SE; a measure of sample size) against effect size. Visual inspection indicates a funnel shape, and thus, suggests no publication bias. However, because of the subjective nature of visual inspections, we conducted a statistical analyses based on the rank correlation (Kendall's tau) between precision and effect size (Begg & Mazumdar, 1994, Rothstein, 2007). We found this correlation to be non-significant ($\tau_b = -.01$, $p = .77$), thus providing converging evidence to support the inferences made from the visual inspection of the funnel plot.[4]

Despite their wide use, funnel plots have been the source of much criticism (e.g., Lau, Ioannidis, Terrin, Schmidt & Olkin, 2006), we have also assessed publication bias by calculating Orwin's (1983) fail-safe N for each of the five regulatory strategies that showed significant results across psychopathology with .10 as the absolute value (corresponding to small effect sizes according to Cohen and lower than our lowest effect size −.14 which was significant). These analyses indicate that the number of additional studies with effect sizes of 0 required to make our results non-significant would be: 3.9 times as many for rumination (351 studies), 2.4 times as many for suppression (122 studies), 2.8 times as many for avoidance (104 studies), twice as many for problem solving (88 studies), and half as many for reappraisal (6 studies). Thus, this supports the notion that publication bias was unlikely in this study.

## 5. Review of longitudinal studies

As noted earlier, there was a great deal of heterogeneity in the designs of longitudinal studies examining the relationships between one of the emotion-regulation variables of interest and one of the outcome variables of interest. Further, with the exception of rumination, there was a small number of longitudinal studies of any of the other emotion-regulation variables. We included the baseline bivariate correlations between emotion-regulation variables and psychopathology variables from these studies in the meta-analysis where possible. Below, we describe the longitudinal results of these studies. See Table 2 for details about the studies.

### 5.1. Rumination and depression

In studies of adults, rumination, as measured with a version of the Response Styles Questionnaire (RSQ; Treynor et al., 2003) predicted increases in self-reported depressive symptoms across periods of a few days to weeks (Butler & Nolen-Hoeksema, 1994; Hong, 2007; O'Connor, O'Connor & Marshall, 2007; Sarin, Abela & Auerbach, 2005; Segerstrom et al., 2000) to one year later (Nolen-Hoeksema, Larson, & Grayson, 1999). RSQ rumination also predicted onsets of major depression across one year (Nolen-Hoeksema, 2000). A few studies found that RSQ rumination did not predict increases in self-reported depression over periods of 5 to 10 weeks (Calmes & Roberts; 2007; Hankin, Fraley, & Abela, 2005; Wenzlaff & Luxton, 2003). Matheson and Anisman (2003) found that a different measure of rumination (Survey of Coping Profile Endorsement, SCOPE) predicted increases in self-reported depression over 6 months. Ciarrochi and Scott (2006) used the rumination scale of the Emotional Control Questionnaire (ECQ; Ciarrochi, Scott, Deane, &

[4] We found similar results when doing visual inspection and calculating correlations for each of the six regulatory strategies.

Heaven, 2003) and found that it did not predict increases in depressive symptoms but did predict decreases in positive mood.

In studies of children, rumination, as measured by the adult rumination scale (RSQ; Treynor et al., 2003), predicted increases in self-reported depression across a span of three years (Broderick & Korteland, 2004). Among adolescents, rumination as measured by the CRSQ or RSQ has been found to predict increases in self-reported depression one to four years (Burwell & Shirk, 2007; Grabe, Hyde, & Lindberg, 2007; Nolen-Hoeksema et al., 2007). Rumination predicted new onsets of major depression across four years as assessed by structured clinical interview in one study of 496 adolescents (Nolen-Hoeksema et al., 2007), but did not predict clinician-rated or mother-rated depressive symptoms over one year in another study of 168 adolescents (Burwell & Shirk, 2007).

### 5.2. Rumination and other psychopathology

Three studies of adults have found that RSQ rumination predicted increases in self-reported anxiety symptoms (Calmes & Roberts, 2007; Nolen-Hoeksema, 2000; Sarin et al., 2005) while two studies found that it did not predict anxiety symptoms (Hong, 2007; Segerstrom et al., 2000, who controlled for worry in the same equation). RSQ rumination predicted increases in alcohol abuse problems in women but not men (Nolen-Hoeksema & Harrell, 2002). One study of adolescent females found that RSQ rumination predicted increases in substance abuse symptoms and eating disorder symptoms, and the onset of bulimia nervosa over a four-year period (Nolen-Hoeksema et al., 2007).

### 5.3. Avoidance

Two studies of adults used the avoidance subscales of the Coping Responses Inventory (CRI: Moos, 1993). Holahan, Moos, Holahan, Brennan, and Schutte (2005) found that a combination of the cognitive avoidance and emotional discharge scales predicted increases in depressive symptoms over 10 years in a sample of late-to-middle aged adults. Blalock and Joiner (2000) found that cognitive avoidance predicted increases in both depressive and anxiety symptoms over 3 weeks in college students, but for women only; there were no significant effects for behavioral avoidance. Engler, Crowther, Dalton, and Sanftner (2006) found significant differences for the Ways of Coping escape-avoidance scale between chronic and recent-onset binge-eaters versus non-binge-eaters over a one-year period of female college students. In a study of female adolescents, Cooper, Wood, Orcutt, and Albino (2003) found that the CRI avoidance coping subscale predicted increases in a composite measure of problem behaviors, but not increases specifically in substance use.

### 5.4. Suppression

Scores on the White Bear Suppression Inventory (WBSI; Wegner & Zanakos, 1994) predicted increases in depressive symptoms over 7 weeks among college students with low life stress (Beevers & Meyer, 2004) and over 10 weeks in another sample of college students (Wenzlaff & Luxton, 2003). Scores on the emotional containment subscale of the SCOPE predicted increases in self-reported depression across 6 months in college students (Matheson & Anisman, 2003). Similarly, a study examining expressive suppression with the Emotion Regulation Questionnaire (ERQ; Gross & John, 2003) found that low emotional suppression and low social anxiety interacted to predict heightened positive affect three months later (Kashdan & Breen, 2008).

### 5.5. Problem solving

Two studies using the Problem-Solving Inventory (PSI; Heppner & Petersosn, 1982) found that this scale predicted changes in self-reported depression across periods ranging from 1–2 weeks (Priester & Clum, 1993) to 3 months (Nezu & Ronan, 1988). Other measures of problem solving have also predicted changes in depressive symptoms over one month (COPE problem solving; Hong, 2007), 6 months (SCOPE problem solving; Matheson & Anisman, 2003), and one year (SPSI; Ciarrochi & Scott, 2006) in college students.

In a study of adolescents, Gerard and Buehler (2004) found that a 4-item measure of problem-solving orientation did not predict change in self-reported depressive symptoms over 1 year when controlling for a number of other risk factors.

## 6. Discussion

In the present meta-analytic review, we evaluated the relationship between six widely studied emotion-regulation strategies and four psychopathology groups. Each emotion-regulation strategy was associated with overall psychopathology in the predicted direction: maladaptive strategies (i.e., rumination, avoidance, suppression) were associated with more psychopathology and adaptive strategies (i.e., acceptance, reappraisal, and problem solving) with less psychopathology. However, interesting patterns emerged when examining the magnitude of these relationships, suggesting that the relationship between emotion-regulation strategies and psychopathology might vary by strategy and type of psychopathology.

Some emotion-regulation strategies were more strongly related to overall psychopathology (i.e., collapsing across symptom types) than others. We found that the effect size for rumination was large; the effect sizes for avoidance, problem solving, and suppression were medium to large; and the effect sizes for reappraisal and acceptance were small to medium (non-significant for the latter). Thus, in general, the maladaptive strategies were more strongly related to psychopathology than the adaptive strategies. This may indicate that presence of a maladaptive emotion-regulation strategy is more deleterious than the relative absence of particularly adaptive emotion-regulation strategies. The exception may be problem solving; not having a strong problem-solving orientation may have wide-ranging negative effects on well-being, and open the door for the development of maladaptive emotion-regulation strategies such as rumination, suppression, and avoidance (Nolen-Hoeksema et al., 2008; Zelazo & Cunningham, 2007).

When we examined the relationships between each emotion-regulation strategy and each psychopathology separately, some patterns emerged. The relationships between certain emotion-regulation strategies were stronger for depression and anxiety than for substance use and eating disorders. Specifically, for rumination, effect sizes were large for anxiety and depression and medium for eating and substance. For avoidance, the effect size was large for depression, medium to large for anxiety, and medium for eating and substance. Lastly, for reappraisal, the effect sizes were small to medium for depression and anxiety, and small for eating and substance. These patterns suggest that, not surprisingly, mood-related disorders (i.e., depression and anxiety) are more closely related to certain problems in emotion regulation than externalizing disorders (i.e., substance use and eating disorders).

Emotion dysregulation, in the form of rumination, avoidance, and difficulties with reappraisal, may show a weaker association with substance use and eating disorders because these relationships might be more complex. Several potential explanations come to mind. For example, the relationship between these strategies and substance and eating disorders might be moderated by reward sensitivity. In this respect, individuals with these emotion-regulation difficulties who also are high on reward sensitivity may be more likely to turn to substances (alcohol, drugs, food) to ameliorate their unregulated distress, and thus, will be more prone to develop substance use disorders and/or eating disorders (Carver, Johnson, & Joormann, 2008). Evidence supporting this notion comes from studies showing

that higher reward sensitivity is correlated with earlier age of onset of drinking alcohol in young adults (Pardo, Aguilar, Molinuevo, & Torrubia, 2007), alcohol use and abuse in non-clinical samples (Loxton & Dawe, 2001), with craving and positive affect responses in response to alcohol cues in young adult hazardous drinkers (Zisserson & Palfai, 2007). Similarly, some studies find that individuals with symptoms of binge eating or eating disorders show elevated levels of reward sensitivity (Davis, Strachan, & Berkson, 2004; Kane, Loxton, Staiger, & Dawe, 2004; Loxton & Dawe, 2001, 2007). Thus, people high in reward sensitivity may be drawn toward the reinforcing properties of substances, such as alcohol and/or palatable foods (Davis et al., 2004; Dawe & Loxton, 2004). Emotional distress, which is chronically higher in people with emotion dysregulation, appears to potentiate reward systems in the brain (Brady & Sinha, 2005), and this potentiation may be even greater in individuals high in reward sensitivity, increasing the chances they will turn to alcohol or binge eating. Intake of alcohol or food will be reinforced both by the satisfaction of high appetitive drives and by the reduction of negative emotions these individuals otherwise cannot regulate. Thus, the combination of emotional dysregulation and high reward sensitivity should be a potent risk factor for the development and/or maintenance of substance abuse and eating disorders.

Alternately, some theories of eating and substance disorders propose that binge eating and substance use function as emotion-regulation strategies in their own right (Macht, Haput, & Ellgring, 2005; Polivy & Herman, 2002; Sher & Grekin, 2007). Individuals regulating their emotions by binge eating or substance use may be less likely to resort to other forms of emotion-regulation strategies, because the binge eating and substance use fill the individuals' emotion-regulation needs. This suggests that binge eating or substance use would be relatively weakly associated with other emotion-regulation strategies, and indeed our meta-analysis showed they were less correlated with emotion-regulation strategies than were depression or anxiety. Still, binge eating and substance use were correlated somewhat with emotion-regulation strategies. Perhaps binge eating and substance use tend to be part of a cluster of maladaptive emotion-regulation strategies used by some individuals. Future research is needed to determine if binge eating (and other eating-related pathology) and substance use actually do serve to down-regulate emotions in some individuals, and how they fit into possible clusters of maladaptive emotion-regulation strategies.

Further, it should be noted that there were substantially more studies of the relationships between rumination, avoidance, and reappraisal, on the one hand, and depression and anxiety, on the other hand, than there were of the relationships between these emotion-regulation strategies and substance use or eating disorders. Thus, effect sizes for substance use and eating disorders may be less reliable than the effect sizes for depression and anxiety.

In contrast, effect sizes for the relationships between problem solving and suppression and depression, anxiety and eating disorders were all medium. Again, the number of studies of these emotion-regulation strategies and eating disorders limits the reliability of our findings, and no studies of the relationships between substance use and suppression or problem solving met the inclusion criteria for this meta-analysis. If these findings hold up in future studies, however, they suggest that suppression and problem solving have similar effects across at least two types of internalizing symptoms and one type of externalizing symptom.

### 6.1. Moderators

We evaluated sample age and type as moderators of the relationships between strategies and disorders. In many cases, we were not able to conduct moderation analysis because of lack of sufficient studies in each cell. Sample type was a significant moderator of avoidance, problem solving, rumination, and suppression, with studies including clinical participants showing stronger relationships between these strategies and psychopathology than studies without clinical participants. Thus, the relationship between dispositional emotion-regulation strategies and psychopathology may be stronger when more extreme groups are compared, suggesting that the strength of this relationship may be a function of clinical severity. This is consistent with research showing that emotion regulation plays a central role in the etiology and maintenance of clinical levels of psychopathology (Berenbaum et al., 2003; Greenberg, 2002; Kring & Bachorowski, 1999; Mennin & Farach, 2007).

These findings underscore the importance of simultaneously studying both normative and clinical populations when conducting research in psychopathology. Specifically, our results indicate that direct comparisons between normative and clinical populations can be critical in our endeavor to delineate how and when normative processes become pathological. However, it is worth keeping in mind that only a minority of our effect sizes (27) came from studies that used a clinical sample and barely any studies used a multi-sample method with a direct comparison of clinical and normative samples. Thus, we recommend that future studies of psychopathology-related processes utilize a multi-sample approach.

Age moderated the relationship between psychopathology and problem solving and suppression, with adults showing stronger relationships than children/adolescents. Conversely, age did not moderate the relationship between psychopathology and rumination. Children may be less able than adults to report on their use of problem solving and suppression because awareness of one's use of these strategies takes a greater degree of meta-cognition than children are capable of (Eisenberg et al., in press). Children may also be less likely to use suppression or problem solving than adults because they require a degree of executive control over emotional reactions that children have not yet developed (Steinberg et al., 2006). In contrast, rumination may be a more primitive, automatic response to negative emotion than problem solving or suppression, which makes it easier for children both to engage in and report on.

### 6.2. Implications

Our findings suggest that certain strategies (i.e., rumination, suppression, avoidance, problem solving) might be more strongly related to mental health than others (acceptance and reappraisal). The relatively small relationships between psychopathology and acceptance and reappraisal are surprising, given the prominent role of these constructs in two major therapeutic approaches: acceptance-based treatments and cognitive-behavioral therapy, respectively (see Hofmann & Asmundson, 2008). First, we note that acceptance-based treatments promote acceptance in order to reduce experiential avoidance (Eifert & Forsyth, 2005; Hayes et al., 1999). This meta-analysis showed that avoidance had a medium size relationship to psychopathology, a finding in accordance with the goals of acceptance-focused treatments. Second, in addition to reappraisal, another important regulatory strategy to CBT is problem solving (Beck et al., 1979; D'Zurilla, 1988; Marlatt et al., 1988), which showed moderate relationships to psychopathology. Thus, our findings provide some support for the foci of CBT.

Given the nature of a meta-analytic review, we examined strategies independently of one another. Thus, we could not model the relationships among strategies, which for example, might have helped clarify the relationship between avoidance and acceptance. Utilizing multivariate structural models (i.e., structural equation modeling, SEM, Arbuckle, 2007; Kline, 1998) would allow researchers to model the relationship between dispositional strategies and psychopathology and to incorporate latent factors (e.g., personality traits, attentional factors) that might account for differential relationships between strategies and disorders. Additionally, it will be important to examine strategies at the state level. In this respect, researchers could model the temporal course of emotion-regulation strategies, since people may use different strategies over the course of an emotional event. For example, it

might be possible that individuals with eating or substance use disorders will try regulatory strategies for a short period of time before using eating or substances to regulate their moods.

Another necessary step would be to examine the relationships between emotion-regulation strategies and psychopathology simultaneously at the dispositional and state level. The importance of examining both levels simultaneously is underscored by studies showing that dispositional and state affect interact to produce different outcomes (e.g., Egloff & Hock, 2001), that dispositional regulatory strategies (i.e., avoidance) moderate the effects of instructed state suppression (Feldner, Zvolensky, Eifert, & Spira, 2003), and that regulatory strategies interact with dispositional affective style to affect mood (Dennis, 2007). In order to examine the interaction between dispositional and state level, we recommend the assessment of spontaneous emotion regulation, since allowing people to regulate their emotions in whichever way comes naturally to them will likely parallel assessment of emotion regulation at the dispositional level (see Berkman & Liberman, 2009; Egloff, Schmukle, Burns & Schwerdtfeger, 2006). Support for this notion comes from studies showing that when participants are instructed to use specific strategies, they have a hard time doing so (Demaree et al., 2006) and work on automatic processes suggesting that people engage in automatic emotion regulation (Williams & Bargh, 2007). A few recent studies have examined spontaneous regulation in the lab (e.g., Egloff et al., 2006; Liverant, Brown, Barlow, & Roemer, 2008), but the extent of regulatory strategies examined and of modeling with dispositional levels of emotion regulation and affect has been limited, so we suggest a more comprehensive pursue of this line of work. Additionally, we recommend the assessment of spontaneous emotion regulation in ecologically valid contexts of assessment such as those that elicit emotions in a social context.

### 6.3. Limitations of the literature

When examining the literature on emotion-regulation strategies and psychopathology, we found that, despite all the interest that emotion regulation and strategies have received in the last decade, the number of effect sizes for some combinations of strategies and disorders was, indeed, small. Thus, the small size of some of our cells might have limited the generalizability of our findings. However, by using random-effect models, we sought to maximize the generalizability of our results. Additionally, it is worth keeping in mind that the two strategies with the weakest relationship to psychopathology (i.e., acceptance and reappraisal) also had the smallest number of effect sizes, suggesting that more comprehensive assessment of these strategies is necessary to better understand asymmetry. When examining the longitudinal data, most focused on rumination and not on other strategies or the interaction between strategies. In this respect, we hope that in future years, the examination of specific emotion-regulation strategies in disorders becomes more systematic.

Finally, some theorists have argued that the adaptiveness of specific emotion-regulation strategies depends on the context, 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). Similarly, some theorists have argued that it is most adaptive to be able to flexibly move between coping strategies depending on the context of a situation (Aldwin, 1994; Barrett & Gross, 2001; Bonanno, 2001; Cole et al., 2004; Compas, Malcarne, & Fondacaro, 1988; Thompson, 1994; Gratz & Roemer, 2004). Unfortunately, only a handful of studies have specifically tested these context-specificity and flexibility hypotheses (e.g., Bonanno, Papa, Lalande, Westphal & Coifman, 2004; Cheng, 2001, 2003). This will be an important focus for future work. We speculate that one reason that studies of non-clinical populations showed less of a relationship between specific emotion-regulation strategies and psychopathology than clinical populations is because the non-clinical populations are more likely to move flexibly between emotion-regulation strategies, and this skill is at least as important as the use of any one strategy in determining psychopathology.

### 6.4. Study limitations

Our investigation had some limitations. First, we examined emotion regulation with self-report data, which allowed us to assess emotional experience in the subjective domain. Examining only one domain is problematic because biases specific to that domain might not be corrected. In the case of the subjective domain, the biases that might have introduced include people's difficulties with reporting emotional phenomena (e.g., Koster, Soetens, Braet, & De Raedt, 2008; Robinson & Clore, 2002), especially in the case of children (Eisenberg et al., in press); confounding items that measure psychopathology with items that measure emotion, and confounding the assessment of emotion generation with regulation (e.g., Cole et al., 2004).

Second, the relationship between emotion-regulation strategies and psychopathology might be inflated because of item overlap. For example, assessment of eating disorder symptomatology might be confounded by the presence affective items in measurement instruments of eating symptoms (Barker & Galambos, 2009), the assessment of emotion might be confounded by distress (Stanton, Dannof-Burg, Cameron, & Ellis, 1994), or the assessment of rumination might be confounded with depressive symptomatology (Nolen-Hoeksema et al., 2008). We addressed the latter issue by showing similar patterns of results when using measures of rumination that did not contain symptom overlap. Still, the possibility of item overlap between self-reports of emotion regulation and psychopathology calls for the use of other methods, including experimental methods, to test the relationships between emotion-regulation strategies and psychopathology symptoms. As noted earlier, experimental methods have confirmed the relationships between some emotion-regulation strategies and symptoms (e.g., rumination and depression), but most relationships remain untested.

In the present meta-analytic review, we evaluated the relationship between six widely studied emotion-regulation strategies and four psychopathology groups. We found that maladaptive strategies were more strongly associated with psychopathology than adaptive strategies, and that rumination, avoidance, and difficulties with reappraisal were more strongly associated with depression and anxiety than with eating and substance use disorders. Conducting an empirical review of this magnitude was possible because of the recent growth in interest in affective phenomena in psychopathology and the resulting flourishing of research examining regulatory strategies. At the same time, this review reflects critical omissions in the study of regulatory strategies in psychopathology. Specifically, regulatory strategies should be comprehensively assessed transdiagnostically and modeled in relation to one another both at the state and dispositional level. We hope that future work on emotion regulation in psychopathology follows a systematic course that carefully addresses these issues.

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