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Emotion Regulation of Events Central to Identity and their Relationship with Concurrent and Prospective Depressive Symptoms

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Abstract

Dispositional emotion regulation is related to the severity and maintenance of depressive symptoms. However, whether emotion regulation specific to an event highly central for an individual's identity is predictive of depressive symptoms has not been examined. Non-clinical participants ($N = 220$) reported the extent to which they employed a selection of emotion regulation strategies when recalling low and high-centrality events. Dispositional emotion regulation and depressive symptoms were also assessed. A seven-week follow-up was conducted. High-centrality events were associated with more emotion regulation efforts. Greater brooding and expressive suppression in relation to high-centrality memories predicted concurrent depressive symptoms after controlling for event valence and dispositional emotion regulation. Effects were absent for low-centrality memories. Emotion regulation in response to high-centrality memories did not predict depressive symptoms at follow up beyond baseline depressive symptoms. Overall the findings showed that maladaptive emotion regulation in response to memories of high-centrality events is important for explaining depressive symptomatology.

Keywords: centrality of event, life events, autobiographical memory, emotion regulation, depression

Emotion Regulation of Events Central to Identity and their Relationship with Concurrent and Prospective Depressive Symptoms

Appraising a negative event as central for our identity is related to posttraumatic stress and depressive symptom severity (e.g., Berntsen, Rubin, & Siegler, 2011; Boals, Schuettler, & South-Dobbs, 2015). However, empirical studies examining how individuals emotionally react to and regulate central events in general, and whether such emotional response is related to clinical symptoms, are lacking.

Individuals employ various strategies to influence their emotional experience. These emotion regulation strategies may be conscious or unconscious (Gross & John, 2003). A sizeable amount of work has been devoted to investigating dispositional or trait-like emotion regulation strategies as factors related to the onset, severity, and maintenance of psychological symptoms in general (e.g., Aldao & Nolen-Hoeksema, 2010; Aldao, Nolen-Hoeksema, & Schweizer, 2010), and depressive symptoms in particular (e.g., Berking, Wirtz, Svaldi, & Hofmann, 2014; Michl, McLaughlin, Shepherd, & Nolen-Hoeksema, 2013). This research has shown that some emotion regulation strategies have stronger maladaptive effects on negative mood and depression than others. For instance, greater use of strategies such as rumination, thought suppression, and expressive suppression is related to higher depressive symptoms and certain cognitive and affective biases (e.g., Borton, Markowitz, & Dietrich, 2005; Joormann, Dkane, & Gotlib, 2006). Brooding, a dimension of rumination characterized by a judgmental and negative self-evaluation, is related to a negative bias in information processing, dysphoric mood, and depressive symptoms (Joormann, et al., 2006; Treynor, Gonzalez, & Nolen-Hoeksema, 2003). In addition, both thought suppression and expressive suppression (i.e., inhibition of the behavioral expression of emotions) are related to increased physiological responses, greater negative mood, and depressive symptoms (Borton, et al., 2005; Campbell-

Sills, Barlow, Brown, & Hofmann, 2006; Ehring, Tuschen-Caffier, Schnulle, Fischer, & Gross, 2010; Gross & John, 2003).

In contrast to these potentially maladaptive regulation strategies, trait-like reflection and cognitive reappraisal are relatively benign regulation strategies (e.g., Aldao & Nolen-Hoeksema, 2010; Gross & John, 2003; Treynor, et al., 2003). Reflection, an active cognitive processing seeking an understanding and/or a solution to one's problems, has non-significant (Whitmer & Gotlib, 2011) or negative correlations to prospective depressive symptoms in non-clinical populations (Treynor et al., 2003). Reappraisal, which refers to interpreting emotion-eliciting events in a manner that changes its emotional impact (i.e., down-regulating the impact of a negative event; Gross & John, 2003), is positively correlated to well-being (Gross & John, 2003) and negatively correlated to depressive symptoms (Aldao & Nolen-Hoeksema, 2010).

Context and Event-Specific Emotion Regulation

More recent theorizations on emotion regulation and psychopathology (i.e., emotion regulation flexibility and sensitivity) propose a more dynamic approach for understanding the relationship between these concepts (Aldao, Sheppes, & Gross, 2015; Bonanno & Burton, 2013; Bonanno, Papa, Lalande, Westphal, & Coifman, 2004). Following these models, various aspects of the emotion regulation process are examined in relation to the context in which such processes take place. "Context" is a broad term that may refer to the situation in which emotions arise (McRae, Heller, John, & Gross, 2011), to the valence of emotions (O'Toole, Jensen, Fentz, Zachariae, & Hougaard, 2014), or to thinking of specific events (Coifman & Bonanno, 2010; Galatzer-Levy, Burton, & Bonanno, 2012).

Supporting evidence for this dynamic model on emotion regulation and psychopathology is rapidly accumulating (e.g., Galatzer et al., 2012; Levy-Gigi et al., 2016; Orcutt, Bonanno, Hannan, & Miron, 2014). However, one aspect that has been understudied

is that of the event-memory emotion regulation—that is, the emotion regulation strategies that individuals employ when thinking back on a particular event in memory. The evidence that specific regulation strategies connected to individual events in memory is important for psychopathology has primarily been investigated to understand emotion regulation in relation to posttraumatic stress symptoms (Bonanno, et al., 2004; Brewin, 1998; Michael, Ehlers, Halligan, & Clark, 2005; Orcutt, et al., 2014). However, whether and how specific emotion regulation strategies employed when remembering specific events is related to other symptom groups, such as depression, has received little attention.

In the context of depression, research has focused on rumination and thought suppression (also referred to as avoidance of memories), as a means of dealing with intrusive memories (Meiser-Stedman, Dalgleish, Yule, & Smith, 2012; Williams & Moulds, 2008). These studies may be viewed as addressing event-specific emotion regulation as they examine emotion regulation in response to unwanted involuntary memories of a specific negative event. These studies have yield mixed results. For example, Williams and Moulds (2008) found in a non-clinical sample that ruminating over intrusive memories (e.g., “I think of what I would have done differently”) was associated with greater depressive symptoms, even after controlling for memory frequency. In the same study, suppressing intrusive memories (e.g., “I think of something else”) was associated with higher depressive symptoms in simple correlations, but not in partial correlations controlling for memory frequency. Similarly, Meiser-Stedman et al. (2012) found in a sample of adolescents that suppression of intrusive memories did not explain unique variance when other variables, such as the intensity of negative emotions (e.g., shame, sadness), were taken into account.

An equally mixed picture has emerged when prospectively predicting depressive symptoms by measures of rumination and thought suppression. Suppressing intrusive memories at a given point did not predict future depressive symptoms among clinically

depressed patients (Brewin, Watson, McCarthy, Hyman, & Dyson, 1998), and non-clinical individuals (Newby & Moulds, 2011a) in two studies. However, in another study (Brewin, Reynolds, & Tata, 1999) greater suppression of intrusive memories at baseline predicted symptom severity six months later among clinically depressed patients. Conversely, in another study, lower suppression of intrusive memories predicted greater depressive symptoms at six months in a non-clinical sample (Newby & Moulds, 2011b). Overall, predicting concurrent and prospective depressive symptom severity in relation to the employment of rumination and thought/memory suppression of intrusive memories has produced mixed findings. Further, to our knowledge, other emotion regulation strategies, such as expressive suppression or reflection, have not been examined in relation to individual remembered events and depressive symptoms.

Centrality of Event and Depressive Symptoms

We speculate that one factor contributing to the inconsistencies found in the research on event-specific emotion regulation in depression may be the lack of assessment of other relevant event-related variables. Specifically, how central the remembered events are for the individuals' identity may be of particular importance. Memories of important events often become central to identity by serving as anchor points for the individual's identity, turning points in the life story and by coloring the interpretation of other experiences (Berntsen & Rubin, 2006). Such central events may be either positive or negative. The judgment of the centrality of an event for one's identity constitutes a subjective appraisal. In non-clinical populations, highly central events are typically positive and often refer to normative transitional events, such as getting married (e.g., Berntsen, et al., 2011). Perceiving a negative event as highly central is related to elevated symptoms of posttraumatic stress disorder (for reviews see Berntsen et al., 2011; Boals et al. 2015), and depression (Berntsen et al., 2011; Boals, 2014; Boelen, 2012; Robinaugh, & McNally, 2010).

When integrating findings on emotion regulation of intrusive memories (e.g., Williams & Moulds, 2008), and findings on event centrality and depressive symptoms (e.g., Boals, 2014), a potential triangulation between event centrality, event-specific emotion regulation, and depressive symptoms emerges. In the context of posttraumatic stress symptoms, Berntsen and Rubin (2006) have suggested that perceiving memories of stressful events as highly central are associated with the engagement of rumination that, in turn, may exacerbate symptom severity. In the present study, we examine whether this proposition may apply to depressive symptoms and other emotion regulation strategies.

To our knowledge, only one study has investigated the interplay between emotion regulation strategies employed for specific events, their centrality for an individual's identity, and depressive symptoms. In this seminal study, Newby and Moulds (2011c) compared the centrality of intrusive memories of depressed, recovered, and never depressed individuals, along with rumination and thought suppression of such memories. Newby and Moulds (2011c) failed to find differences in the centrality of event among the three groups. However, higher memory centrality was related to elevated rumination. Greater rumination over the memories was in turn related to depressive symptoms. These findings suggest that (1) the emotion regulation of intrusive memories (e.g., rumination) is related to symptom severity, and that (2) the centrality of event may be indirectly related to depressive symptoms via emotion regulation. Newby and Moulds' (2011c) findings are in accord with Berntsen and Rubin's (2006) suggestion that highly central events may require greater effort for emotion regulation and that this in turn may be associated with heightened symptoms. These findings are also in line with our current thesis that assessing event-specific emotion regulation strategies is relevant for the prediction of depressive symptoms. However, these propositions have not yet been empirically tested.

Current Study

The aim of the present study was to examine emotion regulation strategies in response to memories of personal events that vary in centrality. We first examined whether the use of a selection of emotion regulation strategies differs for high versus low-centrality events within the same participants. Second, we examined whether specific emotion regulation strategies of high-centrality events (in comparison with low-centrality events) predicted concurrent and prospective depressive symptoms. For this purpose, we asked individuals to report their emotional reactions when experiencing memories of a low- and a high-centrality event. Further, because the interplay between centrality, event-specific emotion regulation, and psychological symptoms has exclusively focused on negative events, it was important to examine the role of valence, centrality, and emotion regulation separately (i.e., positive events may also be stressful; Holmes & Rahe, 1976).

We formulated three main hypotheses. First, we hypothesized that regulating high-centrality events would require greater involvement of emotion regulation processes than regulating low-centrality events. Specifically, we assessed the employment of brooding, memory suppression, expressive suppression, reappraisal, and reflection, when recalling low- and high-centrality events. We expected a greater employment of all five strategies to regulate high-centrality memories relative to low-centrality memories (Hypothesis 1). However, in an extension of previous findings on trait-like emotion regulation (e.g., Aldao et al., 2010), we expected that only brooding, memory suppression, and expressive suppression (i.e., maladaptive strategies) of high-centrality events would predict depressive symptoms (Hypothesis 2). We did not expect high-centrality event reappraisal or reflection to be significant predictors of depressive symptoms (Gross & John, 2003; Treynor et al., 2003). In contrast, we expected strategies employed in association with low-centrality events to be only weakly related to depressive symptoms, if at all. Third, we expected maladaptive emotion regulation of high-centrality events to be associated with depressive symptoms, irrespective of

the valence of the event. Finally, we explored whether the relationship between the regulation of high-centrality memories and depressive symptoms would hold after controlling for trait-like emotion regulation, and whether such regulation would predict prospective depressive symptoms in a short-term follow-up.

Method

Participants

Data were collected from 220 college students with a mean age of 22.64 years ($SD = 2.00$). Seventy-nine percent were women ($n = 173$). Eighty-nine percent ($n = 197$) identified themselves as Caucasians, 6% of other ethnic background ($n = 13$), 1 % as Middle Eastern ($n = 3$), 1% as Asian ($n = 2$), 0.5% as African ($n = 1$), and the ethnic background was not reported for 2% ($n = 4$). One hundred and forty-one participants (64 % of the full sample) completed a follow-up (T2) seven weeks after the initial assessment (T1).¹ Independent *t*-tests revealed no significant differences between follow-up completers and non-completers for any relevant variable including age, gender distribution, depressive symptoms, trait-like emotion regulation (e.g., rumination, expressive suppression), and event characteristics ($ps > .14$).

Design Overview and Procedure

Participants were recruited in Denmark through ads in social media, posters across the university campus, in-class announcements, and a participant pool at the research center. All recruitment materials invited persons aged 18-30 who had experienced recent life changes or stressful events. The project was described as an online study about how young adults think about and react to their life experiences and their overall psychological well-being.

Participants obtained a participant code and a link to a secure website after contacting the researchers. Informed consent was obtained electronically. The study had three parts. First, in an initial assessment, participants completed an online battery containing self-report questionnaires measuring trait-like emotion regulation and depressive symptoms (T1).

Second, participants completed a life events checklist requiring participants to mark all the events that they experienced in the prior six months. Then participants answered several questions regarding the centrality, valence, and emotion regulation of a self-nominated low-centrality event and a high-centrality event. Third, participants that agreed to be contacted again received an email for a follow-up seven weeks after their initial participation, when depressive symptoms were re-assessed (T2; $M = 6.81$, $SD = 2.64$ weeks). All measures were collected online. Participants collected their compensation (a 100 DKK/USD\$14 gift card) in person, at which point they were debriefed. The study was approved by the local review board for research ethics.

Materials

Emotion Regulation Questionnaire (ERQ; Gross & John, 2003). The ERQ is a widely used 10-item self-report questionnaire assessing a general tendency to engage in two emotion-regulation strategies, cognitive reappraisal (e.g., “When I’m faced with a stressful situation, I make myself think about it in a way that that helps me stay calm”) and expressive suppression (e.g., “I control my emotions by not expressing them”). Each item is rated on a seven-point Likert scale going from 1 = *Strongly Disagree* to 7 = *Strongly Agree*. The ERQ’s test re-test reliability for a 20-day interval is good ($r = .71$; Batistoni, Ordonez, da Silva, do Nascimento & Cachioni, 2013). The ERQ’s convergent and discriminant validity is adequate by showing moderate correlations with other measures of emotional processing (e.g., mood repair, emotional expression), and low correlations with personality traits (Cabello, Salguero, Fernandez-Berrocal, & Gross, 2012). The internal consistency in the current study for both the Reappraisal and the Expressive Suppression subscales was satisfactory, $\alpha = .76$.

The White Bear Suppression Inventory (WBSI; Wegner & Zanakos, 1994). This 15-item self-report questionnaire measures individual differences in the reported tendency to suppress thoughts (e.g., “There are things that I try not to think about”). Item responses are on a five-

point Likert scale going from 1 = *Strongly Disagree* to 5 = *Strongly Agree*. The inventory's test re-test reliability for a 12-week interval is high ($r = .80$). The questionnaire correlates moderately with measures of intrusive thinking, anxiety, and depressive symptoms, thus supporting both its convergent and divergent validity ($r_s = .35$ to $.57$; Muris, Merckelbach, & Horselenberg, 1996). The internal consistency of this inventory in the current study was high (Cronbach's $\alpha = .90$).

The Ruminative Response Scale (RRS; Nolen-Hoeksema, Larson, Grayson, 1999). The 22-item version of this widely used questionnaire was employed to assess general tendencies to reflect (e.g., "Go away by yourself and think about why you feel this way) and to brood (e.g., "Think 'Why can't I get going?'") when experiencing low mood (Treyner, et al., 2003). Response options are given in a four-point Likert scale going from 1 = *Almost Never* to 4 = *Almost Always*. The RRS's stability over a one-year period is adequate (.60 and .67 for Brooding and Reflection, respectively) (Treyner et al., 2003). The two-factor solution of this inventory has been replicated in factor analytic studies (Schoofs, Hermans, & Raes, 2010). Likewise, the questionnaire's convergent and discriminant validity has been empirically supported (Kasch, Klein, & Lara, 2001). The internal consistency in the current study was satisfactory for both sub-scales (Cronbach's $\alpha = .72$ for Reflection and $\alpha = .73$ for Brooding).

Beck Depression Inventory – II (BDI-II; Beck, Steer, & Brown, 1996). The BDI-II is the gold standard self-report questionnaire to assess behaviours, attitudes, and feelings that characterize depression within the last two weeks. It consists of 21 symptom items each with four corresponding response options that reflect increasing symptom frequency or severity. The BDI-II test-retest reliability for two and five-week intervals with university students is high ($r_s > .75$; Huprich & Roberts, 2012). The BDI-II's convergent validity is well supported, with moderate to high correlations with other measures of depressive symptom (Beck et al., 1996; Dozois, Covin, & Brinker, 2004; Osman et al., 1997; Sprinkle et al., 2002). The BDI-

II's discriminant validity has been also supported (Beck et al., 1996; Harris & D'Eon, 2008).

The internal consistency in the current study was high (Cronbach's $\alpha = .90$).

Recent Events Checklist (See supplementary e-materials). A checklist of recent life events was developed for the purpose of the current study. Thirty-eight events were selected from studies assessing the most frequent stressful life events reported by young adults (Jackson & Finney, 2002; Smyth, Hockemeyer, Heron, Wonderlich, & Pennebaker, 2008; Wang, Sareen, Bolton, Johnson, & Bolton, 2012), and supplemented with events from the Stressful Life Experiences Scale (ISLES; Holland, Currier, Coleman, & Neimeyer, 2010), and the Social Readjustment Rating Scale (Holmes & Rahe, 1967). The checklist included a wide variety of both positive and negative events as we were interested in understanding the role of centrality of events of either valence. As a first step, participants marked all events from the list that they experienced in the prior six months. In the case that none or only one event was endorsed, participants added one or two events from the prior six months that they had considered important for their identity. Therefore, each participant reported at least two events. Second, they rated the valence of each event endorsed (in the analyses, positive valence is coded as 1 and negative valence as 2). Third, participants nominated the most central (hereafter high-centrality event, HCE) and the least central event (hereafter low-centrality event, LCE) for their identity and life story. Later, participants completed the Centrality of Event Scale (Berntsen & Rubin, 2006) and a series of questions described below regarding the emotional response to their memories of each event.

Centrality of Event Scale (CES; Berntsen & Rubin, 2006). The CES is a measure assessing how central an event is to a person's identity and life story. Items are rated on a five-point scale ($1 = \textit{totally disagree}$; $5 = \textit{totally agree}$). The seven-item version was employed in the current study, which retains the same good psychometric properties of the 20-item version (Berntsen & Rubin, 2006). In the current study, the original instructions were

slightly re-worded to direct participants to complete one CES for the self-nominated high-centrality event (HCE-CES), and another for the low-centrality event (LCE-CES). The CES's test re-test is good for a four-week interval ($r = .68$; Matos, Pinto-Gouveia, & Gomes, 2010). Previous studies support the convergent and divergent validity of the CES (Berntsen & Rubin, 2006; Matos, et al., 2010). The internal consistency for both CESs was high, with HCE-CES Cronbach's $\alpha = .88$, and LCE-CES Cronbach's $\alpha = .93$.

Emotional Responses to Memories. A set of questions developed for the current study were answered for each event separately. The following definition was provided to the participants for answering to the questions: *Sometimes we have spontaneous recollections of an event from the past. These are memories that pop-up in one's thoughts without one trying to think about them. When we have a spontaneous memory, we often experience immediate emotions. To what extent do you experience the following emotions when you have spontaneous memories of "HCE/LCE" event?* Then participants rated fear, happiness, sadness, and anger from 1 = *Not at all* to 5 = *A great deal*. To assess emotion regulation participants were then asked: *Right after you have spontaneous memories of "HCE/LCE event" how much do you do any of the following?: I think: "Why do I always react this way?"* (brooding); *I try not to think about it* (thought/memory suppression), *I analyze the memory to understand my feelings* (reflection), *I change the way I was thinking about the situation* (reappraisal), and *I control my emotion by not expressing it* (expressive suppression). Each emotion regulation item was rated from 1 = *Not at all* to 5 = *A great deal*. The items were taken (and slightly adjusted for the context of memory retrieval) from validated questionnaires assessing trait-like emotion regulation described above including the ERQ (Gross & John, 2003) for expressive suppression and reappraisal, the WBSI (Wegner & Zanakos, 1994) for thought/memory suppression, and the RRS (Treyner et al., 2003) for brooding and reflection. The selected items possessed high face validity of the corresponding

strategy, and a high factor loading ($> .59$) in their corresponding factor in previous validity studies (Gross & John, 2003; Treynor et al., 2003; Wegner & Zanakos, 1994). Further, most of the individual items correlated with the total score of the corresponding inventory in our sample ($r_s > .20$, $p_s < .05$).²

Finally, participants also responded to the following questions for each event: *How long ago did "HCE/LCE event" happen?* (age of event), *When this event took place, how stressful was it for you?* (stressfulness; 1 = *Not at all* to 5 = *A great deal*), and *How often do you have spontaneous memories of this event?* (frequency; 1 = *Never* to 5 = *Once an hour or more*).

The materials were administered in Danish. All the questionnaires in Danish had been employed in other studies (Christensen, et al., 2009; Finnbogadottir & Berntsen, 2011; Harris, Rasmussen, & Berntsen, 2014; Rasmussen & Berntsen, 2010), with the exception of the RRS which was translated and back-translated following standard procedures for the present study. Two other self-report questionnaires were administered as a part of a larger project, but not analyzed for the present study.

Results

Data Analysis Strategy for Hypothesis Testing

Hypothesis 1, concerning differences in emotion regulation of HCEs and LCEs, was tested by conducting paired *t*-tests. For Hypotheses 2 and 3, concerning the relationship between depressive symptoms and HCE and LCE valence and emotion regulation strategies, a separate multiple hierarchical regression analysis was conducted to predict symptom severity at T1 and T2. Gender was entered first as it represents a variable that chronologically precedes event occurrence. Event centrality and valence were then entered to give them priority to explain variance due to their well-documented relationship to depressive symptoms. Event-specific emotion regulation strategies were entered last to examine their potential additional contribution to explain depressive symptoms variance. Only the emotion

regulation strategies correlating with depressive symptoms at T1 or T2 were entered in the regression models.³ Two exploratory analyses were conducted to clarify the contribution of event/state versus trait-like emotion regulation. For sake of parsimony, supplementary analyses are only summarized.

Descriptive Statistics

Number and characteristics of recent events. Participants reported an average of 5.45 important events ($SD = 2.72$, range 2 – 14) happening to them in the preceding six months. Consistent with other studies with non-clinical populations (e.g., Walker, Skowronski, Thompson, 2003), participants reported more positive events ($M = 3.36$, $SD = 2.91$) than negative events ($M = 2.32$, $SD = 1.78$), $t(219) = 4.49$, $p < .001$. All 38 events from the checklist were endorsed at least once, except for “becoming a parent.” See the supplementary *e*-materials for the frequency of each event, and the frequency of events nominated as either HCE or LCE.

Hypothesis 1: Differences between high and low-centrality events. The centrality (CES score) of the HCEs was greater than the centrality of LCEs, thus validating the participants' selection for high and low-centrality events. The proportion of HCEs ($n = 105$, 50%) rated as negative was higher than for LCEs ($n = 84$, 40%), $Z = 1.97$, $p = .049$. Memories of HCEs were rated as more stressful and were associated with more intense fear, sadness, and anger. Consistent with Hypothesis 1, HCEs were associated with significantly greater regulation of emotions across all strategies: memory suppression, expressive suppression, brooding, reflection, and reappraisal (with a Bonferroni correction for five comparisons, $p = .01$) (see Table 1).

Hypotheses 2 & 3: Event Valence, Emotion Regulation, and T1 Depressive Symptoms

Depressive symptoms and simple correlations with event variables. The sample scored within the mild range of depressive symptoms at T1 ($M = 10.37$, $SD = 8.36$, range 0-

47) (Beck et al., 1996). The sample scored significantly lower at T2, $t(1, 140) = 5.26, p < .001$, with a mean in the non-clinical range of depressive symptoms ($M = 7.93, SD = 8.08$, range 0-50). As Table 2 shows, T1 depressive symptoms correlated reliably with most HCE emotion regulation strategies including, memory suppression, brooding, reflection, and expressive suppression, but not for reappraisal. LCE expressive suppression correlated positively with T1 depressive symptoms as well. The pattern for T2 depressive symptoms was the same, with the addition that LCE-Brooding also correlated significantly with T2 depressive symptoms. HCE's negative valence was correlated to greater T1 depressive symptoms, whereas LCE valence was not.

Prediction of T1 depressive symptoms. A multiple hierarchical regression analysis was conducted to determine whether emotion regulation for low and high-centrality events would predict depressive symptom severity, after controlling for event valence and centrality (i.e., although events were either high or low in centrality, the actual centrality for an individual's identity was variable between subjects). Gender was controlled in Step 1. Step 2 consisted of HCE and LCE valence (where 1 = *positive* and 2 = *negative*) and CES scores for the HCE and LCE. Step 3 consisted of the event-specific emotion regulation strategies that were significantly correlated to depressive symptoms in simple correlations (see Table 2).

The final model was significant, $F(10, 177) = 5.29, R = .48, p < .001$, by accounting for 23% of the T1 variance in depressive symptoms (see Table 3). Gender was not a significant predictor in Step 1. Step 2 explained additional variance. In Step 2, HCE's negative event valence and higher centrality were unique predictors of higher depressive symptoms. In contrast, the LCE's valence and centrality were not significant. The last step was also significant. In the final model, HCE negative valence, greater HCE centrality, and greater HCE-Brooding and HCE-expressive suppression were significant predictors of higher

depressive symptoms. Other HCE emotion regulation strategies and LCE-related variables were not significant.

The results supported Hypothesis 2 inasmuch only HCE emotion regulation was uniquely related to T1 depressive symptoms. Furthermore, the results were consistent with Hypothesis 3 given that HCE emotion regulation predicted variance in addition to the events' (negative) valence with similar effect sizes.

Controlling for trait emotion regulation. A more stable tendency to regulate emotions (i.e., trait) may influence the employment of any regulation strategy for a given event or in a given moment (i.e., state). Thus, we examined whether HCE-Brooding and HCE-Expressive suppression would remain significant after controlling for *trait-like* brooding (RRS Brooding; $M = 9.98$, $SD = 3.16$) and expressive suppression (ERQ-ES; $M = 12.69$, $SD = 5.19$), respectively. The analyses were conducted separately for brooding and expressive suppression. HCE-Brooding ($\beta = .13$, $p = .04$) remained significant after controlling for RRS Brooding ($\beta = .48$, $p < .001$). Similarly, HCE-Expressive suppression ($\beta = .16$, $p = .03$) hold significant after controlling for ERQ-Expressive suppression ($\beta = .22$, $p < .001$). These results suggest that brooding and suppressing emotions in relation to highly central events is associated with depressive symptoms beyond a general tendency to employ such emotion regulation strategies. (For full analyses, see supplementary *e*-material).

Prospective Analyses

A hierarchical multiple regression was conducted to determine whether T1 event emotion regulation would prospectively predict depressive symptoms at T2. The variable sequence in the regression model was the same as in T1 analyses, except that T1 BDI-II was entered as a predictor in the first step. Step 2 consisted of gender, followed by Step 3(event valence and centrality). In Step 4, the emotion regulation strategies for both the HCE and LCE that had been significant in simple correlations were entered as predictors.

The model accounted for 68% of the variance in T2 depressive symptoms, $F(11, 111) = 21.26, p < .001$. The first step including T1 depressive symptoms ($\Delta R^2 = .62, p < .001$) explained the most variance. None of the remaining steps added significantly to the amount of variance explained by the first step (although trends were observed, see Table 3). The final model showed a trend-wise effect for higher HCE-Brooding as prospective predictor of greater depressive symptoms at T2 (Table 3), which might be seen as consistent with the effect of brooding in the analyses predicting concurrent depressive symptoms. However, the lack of statistical significance and small effect size render implications unclear.⁴

Discussion

The primary objective of the current study was to examine whether the emotion regulation strategies employed in response to memories of events highly central to an individual's identity were related to depressive symptoms. We aimed to bridge the extensive research supporting the relationship between maladaptive trait-like emotion regulation and depression (Aldao & Nolen-Hoeksema, 2010; Gross & John, 2003; Michl et al., 2013) with research on centrality of event and depressive symptoms (e.g., Berntsen, et al., 2011; Boals, 2014; Robinaugh & McNally, 2010). We addressed a gap in the literature by investigating the employment of a selection of emotion regulation strategies when recalling low versus high-centrality events, and their relation to concurrent and prospective depressive symptoms.

There were three primary findings in this study. First, individuals engaged in greater emotion regulation when remembering high-centrality events (HCEs) relative to less central events (low-centrality events, LCEs). Individuals reported higher employment of reflection, brooding, memory suppression, expressive suppression, and reappraisal for HCEs. Although this possibility had been suggested for rumination (Berntsen & Rubin, 2006), this is the first study to empirically support this contention and to extend this notion to other emotion regulation strategies.

Second, both brooding and suppressing emotions in relation to highly central events were related to greater concurrent depressive symptoms. These relationships were robust as they held significant after controlling for event valence and dispositional brooding and expressive suppression. Regarding brooding, the results are in agreement with the well-established findings for a positive relationship between ruminating over intrusive memories, negative mood (Williams & Moulds, 2010), and depressive symptoms (Williams & Moulds, 2008). Regarding expressive suppression, this is, to our knowledge the first study investigating suppression of emotions triggered by memories in the context of depressive symptoms. Therefore, no direct comparison with other studies may be made. Nonetheless, the significant relationship between higher HCE-Expressive suppression and depressive symptoms is consistent with experimental studies assessing other forms of state expressive suppression. For instance, two studies have found that holding back or suppressing one's emotional expression during a negative mood induction was related to greater negative affect post-intervention compared with acceptance of the emotion (Campbell-Sills, et al., 2006) or the employment of reappraisal (Ehring et al., 2010). Our findings suggest that an appraisal for centrality is related to how individuals employ brooding and expressive suppression. Moreover, when this is done to a large extent for memories of highly central events, a unique relationship with concurrent depressive symptoms is observed.

As expected, HCE-Reappraisal and HCE-Reflection were not significantly related to concurrent depressive symptoms. However, contrary to expectation, HCE-Memory Suppression was also not a significant predictor of concurrent depressive symptoms. Findings with memory suppression have been mixed, thus our findings are partly in line with previous studies in which the suppression of intrusive memories did not predict symptom severity (Meiser-Stedman, et al., 2012; Williams & Moulds, 2008). However, in extension to the studies with intrusive memories, the present study suggests that this may be the case

irrespective of how central the memories are to an individual. Therefore, memory suppression, in its state version, may not be as relevant for explaining depressive symptoms as its trait-like counterpart (Aldao et al., 2010). Taken together, the variability in the result pattern for the various strategies and depressive symptoms suggests that certain HCE regulation strategies are better predictors of depressive symptom severity than others.

Third, in the present study, both the centrality and (negative) valence of high-centrality events were related to depressive symptoms. A negative bias in autobiographical memory in depression has been documented before (e.g., Watkins, Grimm, Whitney, & Brown, 2005), the same has a relationship between high (negative) centrality and depressive symptoms (e.g., Boals, 2014; Pinto-Gouveia & Matos, 2011). In the present study, these effects were independent from each other (i.e., centrality was related to depressive symptoms irrespective of event valence). Additionally, event emotion regulation was at least as strong as event valence in predicting depressive symptoms (i.e., similar effect sizes).

Why would brooding or suppressing emotions of positive central events be related to depressive symptoms? We offer some speculative explanations. For instance, events qualified as positive may be also stressful (e.g., Holmes & Rahe, 1976). In addition, self-criticism, a key characteristic of brooding (Treyner et al., 2003), may extend to judging one's past or current reactions to past events. In this case, (negative) evaluations of oneself may be associated with negative mood, even if the outcome of the event could be judged as positive. Regarding expressive suppression, an experimental study found that remembering positive memories actually worsened the mood in depressed participants (Joormann, Siemer, & Gotlib, 2007). This finding was interpreted as being the result of contrasting current and past states of the self, in which the present suggested deterioration of the self (i.e., a negative, self-devaluating difference between past and current self). Lastly, more recently, a study found

that a fear for positive emotions may be related to depressive symptoms (Vanderlind, Stanton, Weinbrecht, Velkoff, & Joormann, 2017).

Results from the prospective analyses were not as straightforward as those for concurrent symptoms. Prospective analyses suggested at most a marginal effect of HCE-Brooding predicting higher depressive symptoms at a seven-week follow-up when controlling for baseline depressive symptoms. Previous prospective studies assessing rumination and memory suppression in the context of intrusive memories have also yield non-significant findings (Brewin et al., 1998; Newby & Moulds, 2011a). Therefore, it is less clear what the role of emotion regulation in response to memories may play in the maintenance of symptoms (but see Brewin et al., 1999, and Rottenberg, Joormann, Brozovich, & Gotlib, 2005). Future research should aim at resolving this issue.

Finally, an important question arises regarding the direction of the relationship between the emotional response associated to an event in memory and event centrality. In the present study it is not possible to disentangle whether an event was appraised as central due to the emotional consequences that followed it (and thus, perhaps requiring greater efforts for emotional regulation), or whether it was the appraisal of high centrality that generated a greater need for emotion regulation. Appraisal models suggest that the appraisal is the initiator of the emotional response (Ellsworth & Scherer, 2003). However, the emotional response feeds back to influence potential new appraisals. Therefore, a dynamic relationship between appraisal and emotion regulating may be at hand. Methods capturing the temporal dynamics for centrality appraisal and emotion processing would be necessary to address this issue.

There are various limitations to our study. First, we relied on self-reports, which may be affected by reporting biases. Experimental or experience sampling studies exploring the impact of each regulation strategy when remembering central events would represent stronger

evidence for the relationships found. Second, although we made a careful selection of items assessing the emotion regulation strategies, we had only one item for each emotion regulation strategy, precluding more systematic psychometric and conceptual analyses of their properties. Third, our follow-up was short-term. A more extended follow-up period, or multiple assessment points, would enhance our understanding regarding the role of event emotion regulation and centrality in the maintenance of depressive symptoms. Fourth, employing a sample of well-educated, non-clinical adults limits the generalizability of our findings to other groups. Future research should test whether the present findings replicate in clinical depression. Fifth, we asked participants to report events that had taken place within the last six months to ensure that low-centrality events would still be relevant to the individuals' lives. However, in so doing, more remote, and maybe more highly central, events, were not examined.

Despite these limitations, the relationship between HCE-Brooding, HCE-Expressive suppression, and depressive symptoms was robust. Therefore, an important direction for future research is to achieve a better understanding of the intersection of event centrality and emotion regulation. The present findings may also suggest that event-specific emotion regulation is important in psychological syndromes in which a single event may become a central part of a person's identity, such as posttraumatic stress disorder (Berntsen & Rubin, 2006) or prolonged grief (Boelen, 2012). This knowledge may be employed to further advance cognitive and affective models of depression.

Conclusion

The current study contributes to the understanding of the contingencies for the relationship between emotion regulation in response to autobiographical memories and the severity and maintenance of depressive symptoms. Notably, we found that emotion regulation strategies employed for high-, but not low-centrality events, were positively related to

concurrent depressive symptoms. The findings identified brooding and expressive suppression as particularly maladaptive strategies when remembering events highly central to identity and/or life story. These strategies were related to higher concurrent depressive symptoms even after accounting for event valence. Potentially, these findings may generalize to other symptom groups in which specific events may become a central part of a person's identity.

Disclosure statement. The authors declare no conflict of interest.

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Footnotes

¹ A small portion of participants from this sample participated in a memory diary study (Del Palacio-Gonzalez, Berntsen, & Watson, 2017). To verify that their participation did not affect T2 findings, we conducted an analysis controlling for diary participation. The overall results were the same as presented in Table 3, with diary participation *ns*.

² Correlations of the individual HCE items with the total score of their corresponding inventories' total score were $r_s = .20$ to $.40$, $p_s < .05$ for brooding, reflection, memory suppression, and expressive suppression, and non-significant for reappraisal. Correlations for the LCE reflection, memory suppression, and expressive suppression items and their corresponding inventories were $r_s = .14$ to $.25$, $p_s < .05$. The correlations between the LCE reappraisal and brooding items and the inventories' total score were non-significant.

³ Full models with all the emotion regulation items for both HCE and LCE were also tested, but not reported for space reasons. Both T1 and T2 results were largely replicated. In T1 analyses, higher HCE-Brooding, higher HCE-Expressive Suppression, and lower HCE-Reappraisal were significant predictors of greater T1 depressive symptoms. T2 analyses were highly similar to those presented in Table 3, except that the p values were slightly higher.

⁴ We note that the prospective analyses present a potential suppression effect as indicated by a negative correlation between event valence and symptoms (i.e., positive valence related to higher symptoms). The non-significant effect is primarily driven by the inclusion of T1 BDI as control variable (i.e., highly correlated to T2 BDI), and the two event valences. Specifically, T2 BDI and H-Valence have a zero-order correlation of $r = .04$, that becomes $pr = -.18$ when controlling T1 BDI. We preserved the model on the basis of hypothesis testing. Multicollinearity was also assessed for, and both tolerance and variance inflation factors were within acceptable ranges. However, the analysis should be interpreted cautiously.

Table 1
Paired t-tests Contrasting Event and Memory Characteristics of High and Low-Centrality Events

	High-Centrality Event		Low-Centrality Event		<i>df</i>	<i>t</i>	<i>p</i>
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>			
<i>Event characteristics</i>							
CES	28.97	6.51	15.47	7.65	218	21.80	<.001
Stressfulness ^r	3.68	1.26	2.44	1.40	216	10.50	<.001
Memory Frequency ^r	2.82	0.97	2.00	0.90	216	10.33	<.001
<i>Event Emotional Intensity^r</i>							
Fear	2.52	1.31	1.70	1.08	204	7.31	<.001
Sadness	2.69	1.49	1.91	1.20	203	5.69	<.001
Happiness	2.80	1.63	2.74	1.57	205	0.55	.655
Anger	1.92	1.32	1.68	1.15	203	1.98	.049
<i>Event Emotion Regulation^r</i>							
Brooding	1.87	1.22	1.49	0.95	205	3.53	<.001
Reflection	2.73	1.38	1.90	1.28	204	7.46	<.001
Memory Suppression	2.36	1.36	1.96	1.34	205	3.02	.001
Expressive Suppression	2.12	1.19	1.76	1.14	205	3.69	<.001
Reappraisal	2.21	1.22	1.76	1.12	205	4.37	<.001

Note. *N* = degrees of freedom (*df*) + 1. Variations on *df* are due to missing data for pairwise analyses. CES = Centrality of Event Scale.

^rRating scales ranging from 1 to 5.

Table 2

Correlation of Depressive Symptoms with Centrality, Valence, and Regulation of High and Low Centrality Events

	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	
1 T1 BDI	.76**	.18**	.18**	.33**	.19**	.25**	.28**	.11	.19**	.06	.12	.10	.06	.17*	.04	
2 T2 BDI [^]		.27**	.04	.38**	.11	.25**	.19*	.03	.12	.05	.25**	.13	.10	.22*	.04	
3 H-CES			-.19**	.06	-.12	.10	-.09	-.02	.16*	.25**	.09	.21**	.15*	.15*	.15*	
4 H-Valence [†]				.32**	.57**	.24**	.30**	.37**	.06	.00	.00	-.11	-.11	-.01	-.05	
5 H-Brooding					.48**	.38**	.41**	.38**	.13	.06	.18**	.07	.22**	.20**	.17*	
6 H-M. Sup.						.41**	.50**	.53**	.12	.07	.13	.15*	.12	.15*	.15*	
7 H-Ref.							.30**	.51**	.15*	.07	.13	.11	.35**	.14	.18*	
8 H-E. Sup.								.44**	.08	-.02	.15*	.03	0.04	.31**	.08	
9 H- Reap.									.12	-.04	.02	.06	.16*	0.06	.17*	
10 L-CES										.03	.19**	.08	.29**	.21**	.15*	
11 L-Valence [†]											.33**	.52**	.34**	.31**	.37**	
12 L-Brooding												.55**	.49**	.43**	.48**	
13 L-M. Sup.													.44**	.50**	.46**	
14 L-Ref.														.37**	.57**	
15 L-E. Sup.															.39**	
16 L-Reap.																--

Note. $N = 220$. BDI = Beck Depressive Inventory-II; CES = Centrality of Event Scale; ERQ = Emotion Regulation Questionnaire; E. Sup. = Expressive Suppression; H = High-Centrality Event; M. Sup. = Memory Suppression; L = Low-Centrality Event; Reap. = Reappraisal; T1 = Time 1; T2 = Time 2.

[†] 1 = positive and 2 = negative.

[^] $n = 141$.

* $p < .05$. ** $p \leq .01$.

Table 3
Hierarchical Multiple Regression Predicting T1 and T2 Depressive Symptom from High-Centrality and Low-Centrality Event Variables

	T1 Depressive Symptoms (N = 220)					T2 Depressive Symptoms (N = 141)				
	ΔR^2	B	<i>t</i>	<i>sr</i> ²	<i>p</i>	ΔR^2	β	<i>t</i>	<i>sr</i> ²	<i>p</i>
Step 1	.03				.452	Step 1	.62			<.001
Sex ²		.06	0.75	<.01	.452	T1 BDI-II	.79	13.98	.62	<.001
Step 2	.11				<.001	Step 2	.01			.096
Sex ²		.01	0.16	<.01	.876	T1 BDI-II	.78	13.86	.61	<.001
H-CES		.23	3.05	.05	.003	Sex ²	.09	1.68	.02	.096
H-Valence [†]		.26	3.60	.07	<.001	Step 3	.03			.069
L-CES		.08	1.13	.01	.259	T1 BDI-II	.79	13.38	.61	<.001
L-Valence [†]		.03	0.35	<.01	.725	Sex ²	.10	1.82	.03	.071
Step 3	.12				<.001	H-CES	.09	1.47	.02	.143
Sex ²		.05	0.71	<.01	.481	H-Valence [†]	-.12	-1.98	.03	.050
H-CES		.18	2.51	.03	.013	L-CES	-.02	-0.33	<.01	.743
H-Valence [†]		.19	2.28	.03	.024	L-Valence [†]	<.01	0.06	<.01	.950
L-CES		.01	0.20	<.01	.843	Step 4	.03			.134
L-Valence [†]		-.01	-0.07	<.01	.944	T1 BDI-II	.76	11.42	.54	<.001
H-Brooding		.22	2.63	.04	.009	Sex ²	.09	1.52	.02	.132
H-Reflection		.08	1.04	.01	.299	H-CES	.06	1.05	.01	.295
H-Mem. Sup.		-.19	-1.88	.02	.062	H-Valence [†]	-.17	-2.60	.06	.011
H-Exp. Sup.		.22	2.53	.03	.012	L-CES	-.04	-0.74	<.01	.460
L-Exp. Sup.		.12	1.53	.01	.128	L-Valence [†]	-.03	-0.55	<.01	.583
						H-Brooding	.14	2.14	.04	.035
						H-Reflection	.02	0.38	<.01	.702
						H-Exp. Sup.	-.04	-0.54	<.01	.589
						L- Brooding	.11	1.55	.02	.123
						L- Exp. Sup.	-.05	-0.77	.01	.442

Note. BDI-II = Beck Depressive Inventory-II; CES = Centrality of Event Scale; Exp. Sup. = Expressive Suppression H = High-Centrality Event; L = Low-Centrality Event; Mem. Sup. = Memory Suppression; T1 = Time 1; T2 = Time 2.

²1 = man and 2 = woman.

[†]1 = positive and 2 = negative.