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Running head: EMOTION REGULATION AND MOOD BRIGHTENING

Emotion regulation and mood brightening in daily life vary with depressive symptom levels

Vanessa Panaite^{1, 2}, Peter Koval^{3,4}, Egon Dejonckheere⁴, Peter Kuppens⁴

¹James A. Haley Veterans' Hospital

² University of South Florida

³ University of Melbourne

⁴KU Leuven - University of Leuven

Corresponding Author:

Vanessa Panaite, PhD, James A. Haley Veterans' Hospital, 8900 Grand Oak Circle, 132B, Tampa, FL, 33637, USA. E-mail: vanessap@mail.usf.edu

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ABSTRACT

Naturalistic studies of emotional reactivity in depression have repeatedly found larger decreases in negative affect among depressed individuals in response to daily positive events. This so-called “mood brightening” effect represents a theoretical and empirical oddity. The current study is a secondary analysis investigated whether the mood brightening effect is moderated by spontaneous use of emotion regulation strategies, which have been implicated in the maintenance and modulation of negative affect. Participants ($N = 95$) representing a large spectrum of depressive symptoms reported their experiences of negative affect and the occurrence of positive events in daily life for seven days using the experience sampling method. Our findings replicate and build upon those of prior studies relating to the mood brightening effect in the following ways: 1) we observed the mood brightening effect for specific negative emotions of sadness, anger, anxiety; and 2) we found evidence that the mood brightening effect is moderated by spontaneous use of rumination, distraction, and expressive suppression, which have been shown to enhance or dampen negative affect. The role of emotion regulation strategies in daily emotion reactivity to pleasant events is discussed.

KEYWORDS

Mood brightening; negative affect; depressive symptoms; emotional reactivity; positive events.

The mood brightening (MB) effect observed in ecological investigations of emotional reactivity in depression is a theoretical and empirical oddity. The MB effect refers to findings that depressed individuals report larger decreases in negative affect (NA) in response to positive events throughout daily life compared with their never-depressed peers (Peeters, et al., 2003; Bylsma, Taylor-Clift, & Rottenberg, 2011; Thompson et al., 2012). Thus, people with depression seem to “benefit” more from positive events than healthy individuals, at least in terms of the magnitude of change in NA following pleasant daily events. This seems to contradict common sense and a theoretical view of depression as involving inflexibly high levels of NA (e.g., Kashdan & Rottenberg, 2010; Koval et al, 2013). Moreover, it stands out against most lab-based studies investigating emotional reactivity in depression, which have largely found that depression is associated with blunted emotional responding, supporting the emotion context insensitivity theory of depression (ECI; Rottenberg, 2005, 2017). The current study aims to further our understanding of the MB effect by examining the moderating role of emotion regulation strategy use on emotional reactivity in daily life. This has promise for intervention and possibly prevention efforts to alleviate NA in dysphoric and depressed individuals.

Emotion regulation has been defined as the strategic use of cognitive or behavioral efforts to modify emotional responses. This may include dampening or enhancing the intensity or duration of emotions, or modifying how emotions are overtly expressed (Gross, 2002). Certain emotion regulation strategies have been especially linked to depression (see Aldao, et al, 2010 for a meta-analysis). Nolen-Hoeksema developed theoretical and empirical accounts establishing rumination as both a vulnerability and maintaining factor in depression. Rumination, the tendency to repeatedly focus on the cause and outcomes of negative life events and negative moods, has been linked to prolonged and intensified negative affect (Moberly & Watkins, 2008;

Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008). Rumination may foster depression by displacing more active emotion regulation strategies, such as problem solving and by increasing focus on negative self-referential information (Nolen Hoeksema et al., 2008). For example, inducing depressive rumination in an unselected sample leads to increased negative affect in the lab (Watkins, Moberly, & Moulds, 2008) and ruminative self-focus, especially brooding, is related to increased negative affect in daily life (Moberly & Watkins, 2008). This suggests that the MB effect may be stronger when positive events occur against a background of high levels of rumination since it further enhances NA. Events that are sufficiently positive to be appraised as such may help to break the cycle of rumination by shifting attention away from negative thoughts and feelings.

Other regulation strategies that normatively lead to sustained or intensified negative affect may have similar augmenting effects on mood brightening. For instance, depression is also associated with a greater habitual use of suppression (D'Avanzato et al., 2013), which despite decreasing negative behavioral expression, it exacerbates NA experience (Gross & Levelinson, 1993, 1997). Curiously, emotion regulation strategies that tend to diminish NA, such as reappraisal, are less strongly associated with depression (Aldao, et al, 2010) yet are generally a predominant focus in major therapeutic interventions for depression (e.g., CBT). The observed lack of association between depression and reappraisal is likely because it is not knowledge and skill that are impaired among individuals with depression, but rather deployment of context appropriate emotion regulation strategies (see Rottenberg, 2017 for a review; Kovacs, et al, 2009; Haines et al., 2016). Finally, although distraction too has been reported to aid in alleviating NA (Nolen-Hoeksema & Morrow, 1993). it is possible that its use during positive opportunities may actually impede mood repair, via disruption of emotion processing, and so it may have a

less clear role in MB. It is clear that further understanding the moderating role of emotion regulation strategies in enhancing or diminishing the MB effect may help to strengthen intervention and prevention efforts to increase emotional flexibility in depression.

Considering both lab and daily life data showing that attempts to regulate emotions impacts both mean levels and dynamics of emotions (Brans et al, 2013; Gross, 1998; Hajcak & Nieuwenhuis, 2006; Jackson, Malmstadt, Larson, & Davidson, 2000; Koval et al., 2015), the key to understanding the MB effect may lie in looking at the role of emotion regulation in modulating emotional responses in depression. Depression has been associated with affective dysregulation, an umbrella term that encompasses limited efficiency and/or lack of context-sensitive deployment of regulatory strategies, despite evidence that knowledge and skill may not be significantly impaired among individuals with depression (see Rottenberg, 2017 for a review; Kovacs, et al, 2009). Depressed people do not necessarily perform poorly on performance-based emotion regulation tasks (Greening, Osuch, Williamson, & Mitchell, 2014) and do not consistently differ from healthy controls in their ability to “repair” sadness with cognitive reappraisal or distraction, when instructed (Ehring, Tuschen-Caffier, Schnulle, Fischer, & Gross, 2010; Joormann et al., 2007). However, individuals with depression seem to favor regulation strategies that generally maintain low mood, such as rumination and expressive suppression (Kovacs et al, 2009, see Aldao et al, 2010 for a meta-analysis) and show inflexible spontaneous deployment of emotion regulation strategies across contexts (e.g., Ehring, Tuschen-Caffier, Schnulle, Fischer, & Gross, 2010; Haines et al., 2016).

The current study is a first attempt to investigate the role of ER in the MB effect, using ecological momentary assessment (EMA). Specifically, a sample of individuals oversampled to represent a broad range of depression symptom levels reported their experiences of several

emotions, use of emotion regulation strategies, and occurrence of positive events in daily life 10 times per day for one week.

Our first aim was to investigate negative emotional reactivity to positive daily events. First, we expected to replicate prior findings that individuals with higher depressive symptoms would report, on average, larger decreases in NA in response to positive events (i.e., the MB effect) relative to participants with lower depressive symptoms. It is noteworthy that EMA studies of reactivity focused on NA, whereas laboratory studies focused on discrete emotions. Therefore, we propose an extension of prior EMA studies in depression by investigating whether the MB-effect is observed for discrete negative emotions such as sadness, anger, and anxiety.

Our second aim was to explore whether the use of certain emotion regulation strategies moderated the relationship between level of depressive symptoms and NA reactivity to positive events (i.e., the MB effect). Given extensive work supporting the idea that regulatory strategies such as rumination or suppression have NA-enhancing effects, we predicted that those individuals that ruminate the most would experience larger decreases in NA in response to positive events (i.e., a heightened MB effect). Although distraction generally alleviates NA, in positive contexts distraction may have an inhibitor effect by interfering with emotional processing, an effect to be explored in the current paper. Finally, we do not have robust predictions and therefore will explore the role of reappraisal NA reactivity during positive events, given weaker links between reappraisal and depression (e.g., Aldao et al, 2010).

Method

Participants

We analysed data from an existing study (see Koval et al., 2013 for details about the parent study), in which 100 undergraduates were recruited from an initial pool of 439 undergraduates at the University of Leuven who were screened for depression symptoms using

the Centre for Epidemiologic Studies Depression Scale (CES-D; Radloff, 1977; Range = 0 –52, $M = 16.39$, $SD = 10.27$, $\alpha = .92$). Our aim, in the original study, was to recruit a sample representing a wide, balanced, and uniform range of depressive symptoms. To this end, we used stratified sampling (Ingram & Siegle, 2009) to select a random sample of participants from each quintile of the CES-D pre-screening distribution. We contacted 241 eligible participants before reaching our target sample of 100 (CES-D pre-screening Range = 0 –50; $M = 19.27$; $SD = 12.53$). The final sample included 55 participants scoring above Radloff's (1977) clinical cut-off of 16, and 32 participants who scored above the more conservative cut-off score of 27 (Gotlib, Lewinson, & Seeley, 1995). Participants were reimbursed 70 EUR for their involvement. The sample size ($N=100$) was determined for the original study to ensure sufficient power to detect effect sizes of $r \approx .3$. One participant withdrew early, leaving a final sample of 99 (62 women; $M_{age} = 19.05$, $SD_{age} = 1.27$). However, EMA data from four other participants were excluded due to equipment malfunction ($n = 3$) or poor compliance with the EMA protocol (i.e., completion of less than 50% of scheduled survey; $n = 1$). No other data exclusions were used. The current study is an exploratory re-analysis of the available data and sample size.

Materials and Procedure

Participants attended an introductory lab session where they completed the CES-D for a second time along with other personality, well-being, cognitive control, and resting physiology measures (not reported here). No other measures or manipulations were administered during the initial lab session. Participants were then given a Palm Tungsten E2 palmtop running the Experience Sampling Program (Barrett & Barrett, 2001) and instructions for its use. Participants used the Palmtop to rate their momentary feelings, use of regulation strategies, and occurrence of events, 10 times per day over the following seven days. At the end of the week, participants

returned to the lab for a final session during which they completed an emotional film-clip task (not reported here).

Depressive symptoms. The CES-D was used to measure current depressive symptoms. This 20-item scale asks respondents to indicate how frequently they have experienced a range of depressive symptoms (e.g., “I had crying spells”) over the past week from 0 (*rarely or none of the time*) to 3 (*most or all of the time*). At the time of the study, 37 participants scored at or above Radloff’s (1977) clinical cut-off of 16, and 13 participants scored at or above Gotlib and colleagues’ (1995) more conservative clinical cut-off score of 27. The CES-D showed very good reliability ($\alpha = .92$) and correlated with CES-D at pre-screening at $r(99) = .72, p < .001$. All analyses reported below used CES-D scores measured at the time of the study rather than pre-screening CES-D scores.

EMA protocol. Participants were prompted to complete the EMA survey 10 times per day for 7 consecutive days, according to a stratified random interval scheme. On average, participants were beeped every 73.30 min (SD = 29.33). Compliance was high: participants responded to an average of 91.5% of the programmed beeps (SD = 6.2). At each beep, participants rated the intensity of four negative emotions (“sad”, “angry”, “anxious”, “depressed”) and two positive emotions (“happy”, “relaxed”). Participants also rated their use of six emotion regulation strategies. Each item began with “Since the last beep . . .”, and ended with “did you ruminate about your feelings” (rumination), “did you calmly reflect on your feelings?” (reflection), “did you see the event that caused your feelings from a different perspective?” (reappraisal), “did you try to distract yourself from your feelings?” (distraction), “did you talk with others about your feelings?” (social sharing), and “did you suppress the expression of your feelings?” (expressive suppression; see Brans, Koval, Verduyn, Lim, Kuppens, 2013). All

emotion and emotion regulation items were rated on a visual sliding scale with minimum labelled “not at all” and maximum labelled “very much so”. Ratings on the visual sliding scales were converted to values ranging between 1 and 100. Finally, participants were asked to report whether a positive or negative event had occurred since the prior beep, with two separate yes/no items.

Negative Affect. For each episode recorded during the 7-day EMA study, an NA score was computed by adding the 4 negative emotion adjectives. An average across all episodes was computed to determine the overall daily NA. Multilevel reliability accounting for within-person changes over time resulted in an estimate of .90 for NA (estimate similar to those reported by Bylsma et al, 2011; Thompson et al., 2012).

Statistical methods

Given that time points (i.e., EMA surveys) were considered non-independent and clustered within each participant, analyses of daily life emotional reactivity to positive events were performed using Hierarchical Linear Modeling (HLM). Analyses were implemented using SPSS statistical software package Version 22 (IBM, 2013). HLM can accommodate within person clustering of time points by accounting for non-independence of clustered data and estimating variance at all levels (Nezlek, 2001). In all models described below, (continuous) predictors were person-mean centered, implying that Level-1 parameters represent purely within-person effects (Enders & Tofghi, 2007). An unstructured covariance matrix was used for the random effects and maximum likelihood was used in the current analyses.

The MB effect. Using multilevel modeling, negative affective reactivity to positive events was modeled by regressing each person i 's NA level at the current time-point t , onto a dummy variable indicating the occurrence of positive events “since the previous survey”

(positive event_{*ti*}: 0=no event; 1=event), while controlling for person *i*'s level of NA at the prior time-point (NA_{*t-1i*}), as shown in the Level-1 model equation, below.

Level 1 Model:

$$NA_{ti} = \beta_{0i} + \beta_{1i}(NA_{t-1i}) + \beta_{2i}(\text{positive event}_{ti}) + r_{ti}$$

At Level-2, the Level-1 intercept and slopes were allowed to vary randomly across participants and modeled as a function of individual differences in depressive symptoms (i.e., CES-D scores), as shown in the Level-2 model equations, below.

Level 2 Model:

$$\beta_{0i} = y_{00} + y_{01}(\text{CES-D}_i) + u_{0i}$$

$$\beta_{1i} = y_{10} + y_{11}(\text{CES-D}_i) + u_{1i}$$

$$\beta_{2i} = y_{20} + y_{21}(\text{CES-D}_i) + u_{2i}$$

Of particular relevance to the current study, β_{2i} is a slope reflecting each person *i*'s NA reactivity to positive events, and y_{21} reflects how individual differences in NA reactivity to positive events are related to depressive symptoms (a direct test of the MB effect).

Moderation of the MB effect by ER strategies. Next, to investigate whether the MB effect was moderated by state use of ER strategies, we ran six additional models including each ER strategy and its interaction with positive events at Level-1, with separate models per strategy, as shown in the Level-1 model equation below.

Level 1 Model:

$$NA_{ti} = \beta_{0i} + \beta_{1i}(NA_{t-1i}) + \beta_{2i}(\text{positive event}_{ti}) + \beta_{3i}(ER_{ti}) + \beta_{4i}(\text{positive event}_{ti} * ER_{ti}) + r_{ti}$$

In the Level-1 equation, β_{4i} reflects the interaction between use of a given ER strategy and event reactivity, indicating whether the use of a certain ER strategy in the time-interval between *t-1* and *t* modulated NA reactivity to positive events. As before, the Level-1 intercept

and slopes were allowed to vary randomly across participants and modeled as a function of CES-D scores, as shown in the Level-2 model equations below.

Level 2 Model:

$$\beta_{0i} = y_{00} + y_{01}(\text{CES-D}) + u_{0i}$$

$$\beta_{1i} = y_{10} + y_{11}(\text{CES-D}) + u_{1i}$$

$$\beta_{2i} = y_{20} + y_{21}(\text{CES-D}) + u_{2i}$$

$$\beta_{3i} = y_{30} + y_{31}(\text{CES-D}) + u_{3i}$$

$$\beta_{4i} = y_{40} + y_{41}(\text{CES-D}) + u_{4i}$$

Of particular relevance to the current study, y_{41} reflects the three-way (cross-level) interaction between positive events (Level-1), use of a particular ER strategy (Level-1) and depressive symptoms (Level-2). In other words, y_{41} reflects the degree to which use of a particular ER strategy moderates the potential MB effect.

Graphs. Figures were developed in R with the ggplot2 package. For all plots, continuous predictors were within-person centered at the momentary level, and grand-mean centered at the person level. Interactions were visualized for the case where the average person reported an average emotion at the previous moment (i.e., lagged emotion is zero). For emotion regulation, we used -1 SD and +1 SD to visualize “low” and “high” strategy use, respectively. At the between-person level, participants’ CESD scores were grand-mean centered, with “low” and “high” representing -1 SD and +1 SD around the grand mean for depressive symptoms, respectively.

Results

Preliminary analyses

Before testing our primary hypotheses, we first examined whether level of depressive symptoms predicted overall negative affect, negative emotions (angry, sad, anxious), and mean use of ER strategies in daily life. Each of these variables was entered separately as an outcome in means-as-outcomes multilevel models, with depressive symptoms entered as a Level 2 predictor.

Mean daily negative affect, negative emotions, and positive events

Individuals with higher depressive symptoms reported higher mean levels of NA ($\beta = 13.87$, $SE = 1.76$, $p < .001$), including higher mean levels of sadness ($\beta = 15.96$, $SE = 2.10$, $p < .001$), anger ($\beta = 8.26$, $SE = 8.26$, $p < .001$), and anxiety ($\beta = 10.52$, $SE = 1.94$, $p < .001$) in daily life, relative to those reporting fewer depressive symptoms. Those with higher depressive symptoms also reported fewer positive events relative to individuals with fewer depressive symptoms (see Table 2).

State emotion regulation

We investigated use of six emotion regulation strategies: rumination, reflection, reappraisal, expressive suppression, social sharing, and distraction. Findings revealed that those higher in depressive symptoms were more likely to report using rumination ($\beta = 18.89$, $SE = 2.98$, $p < .001$), expressive suppression ($\beta = 12.17$, $SE = 3.01$, $p < .001$), and distraction ($\beta = 7.91$, $SE = 3.35$, $p = .021$). Reflection ($\beta = 2.85$, $SE = 2.58$, $p = .273$), reappraisal ($\beta = .04$, $SE = 2.43$, $p = .987$), and social sharing ($\beta = -1.55$, $SE = 2.54$, $p = .543$) were not reliably related to level of depressive symptoms (see Table 2 displaying correlations between CES-D and emotion regulation strategies).

Affective reactivity to positive events. Is the mood brightening effect specific to sadness?

Results of our first main analyses (described above) showed that NA reactivity to positive events varied as a function of depressive symptoms, such that those with higher depressive

symptoms experienced larger decreases in NA ($y_{21} = -5.24$, $SE = .98$, $p < .001$), sadness ($y_{21} = -5.66$, $SE = 1.39$, $p < .001$), anger ($y_{21} = -4.43$, $SE = 1.20$, $p < .001$), and anxiety ($y_{21} = -5.51$, $SE = 1.16$, $p < .001$) following positive events (see Figure 1), replicating the MB effect reported in previous EMA studies, and extending previous findings to demonstrate the MB effect for specific high-arousal (anger, anxiety) and low-arousal (sadness) negative emotions.

Does use of emotion regulation strategies moderate the mood brightening effect?

Results of the models including interactions with ER strategies revealed that the MB effect was moderated by rumination, expressive suppression, and distraction (see Table 3 for results of all six models including ER strategies). Specifically, for general NA, the MB effect was stronger when individuals with higher depressive symptoms reported greater use of rumination ($y_{41} = -.09$, $SE = .03$, $p = .015$). An opposite effect was observed for distraction ($y_{41} = .16$, $SE = .03$, $p < .001$) and expressive suppression ($y_{41} = .13$, $SE = .03$, $p < .001$), such that individuals with higher levels of depressive symptoms reported smaller decreases in NA following positive events when engaging in above average levels of distraction or suppression. A similar pattern of results was observed for sadness specifically (see Table 3 and Figure 2).

Different patterns were noted for anxiety and anger. Greater use of reappraisal ($y_{41} = -.23$, $SE = .06$, $p < .001$) and rumination ($y_{41} = -.16$, $SE = .05$, $p = .005$) were associated with a larger decrease in anger in response to positive events among those with high depressive symptoms. However, higher use of distraction was associated with a larger increase in anger ($y_{41} = .11$, $SE = .04$, $p = .022$) and anxiety ($y_{41} = .11$, $SE = .04$, $p = .019$) during positive events among those with high depressive symptoms (see Table 3).

Discussion

Previous daily life studies have demonstrated that depressed individuals and those with elevated depressive symptoms experience larger decreases in NA following positive events, relative to healthy individuals and those with lower depressive symptoms (Peeters, et al., 2003; Bylsma, Taylor-Clift, & Rottenberg, 2011; Thompson et al., 2012). The current study replicated this MB effect and extended these findings in two ways: First, in addition to observing mood brightening for overall NA, we also observed the MB effect for each of the specific negative emotions, including low-arousal sadness and high-arousal anger and anxiety. Second, we found moderation of this effect by state use of ER strategies that have been shown to enhance or dampen NA.

First of all, our findings confirm that individuals with higher levels of depressive symptoms derive greater emotional benefits from positive events, reporting larger momentary decreases in anxiety, sadness, and anger. Although a focus on discrete emotions is the norm in laboratory studies (e.g., Rottenberg, Kasch, Gross, Gotlib, 2002), previous EMA studies have not demonstrated the MB effect in relation to specific low-arousal negative states (sadness) and high-arousal negative emotions (anxiety and anger). Studies on negative emotional reactivity to positive probes in the lab have primarily focused on mood repair paradigms, such as recalling positive memories, which have generally resulted in negative findings (e.g., Joormann, Siemer, & Gotlib, 2007). However, this may be a result of substantive differences between in vivo emotional experiences and biased memory consolidation, which has been shown to favor negative material (e.g., Everaert, Duyck, & Koster, 2014).

Second, the current study was the first to investigate the possible moderation of the MB effect by the use of specific ER strategies in daily life. Our findings show that the MB effect was moderated by the use of three emotion regulation strategies that have been associated with

depression: rumination, distraction and expressive suppression. A core current finding was that presence of momentary rumination enhanced the mood brightening effect both when looking at NA and discrete emotions, specifically anger and sadness. A large body of work has linked low mood and rumination (Moberly & Watkins, 2008; Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008) through repetitive focus on sources and outcomes of NA, especially sadness and dysphoria. However, anger related rumination has also been linked to depression in young adults (see Gilbert, Cheung, Irons, McEwan, 2005). Our findings suggest that the MB effect is stronger when positive events occur against a background of high levels of rumination, a strategy shown to maintain or even intensify NA. One speculation is that temporally, the MB effect is preceded by an increase in NA due to rumination. Although our study was not equipped to delineate mechanistic pathways, it is possible that positive events have a direct impact on lowering rumination and consequently have the strongest implications for mood repair among those at highest depression risk.

Although the MB effect was also observed when participants reported using expressive suppression, which like rumination, often leads to enhanced NA, the MB effect was attenuated when positive events occurred in conjunction with high levels of suppression. These findings possibly reflect an inhibiting role of suppression for hedonic experience. It is possible that expressive suppression elicits increasing efforts to persistently keep the expression of NA at bay, but which may interfere with opportunities for affective repair offered by positive events. This is especially problematic given that depressed individuals are less likely to attend to and faster to disengage from positive stimuli relative to healthy individuals (Joormann & Gotlib, 2007; Levens & Gotlib, 2010). Another possibility may be that expressive suppression is generally implemented late in the emotion generation process (Gross & John, 2003), when emotions have

been fully developed and possibly less likely to respond to environmental changes, such as positive events.

Finally, our findings suggesting a dampened yet present MB effect during high levels of distraction among those reporting high depressive symptoms, partially supporting prior findings reflecting that distraction, although not considered an “adaptive” emotion regulation, aids in mood repair (e.g., Ehring, Tuschen-Caffier, Schnulle, Fischer, & Gross, 2010; Joormann et al., 2007). However, these findings have been generated by work on negative emotion regulation in negative contexts. Our findings may extend prior results showing that distraction from depressed mood helped with the generation of less negative autobiographical memories (Lyubomirsky, Caldwell, & Nolen-Hoeksema, 1998), which possibly subsequently aided in mood repair. It is possible, however, that use of distraction in a context insensitive manner may lead to hedonic dysregulation via limited emotion processing of positive contexts, which may have manifested as a dampened MB effect in the current study. Although this explanation is speculative and awaits further investigation.

Despite the novelty and strengths of our study, findings should be interpreted with a few limitations in mind. First, a depression diagnosis was not verified in the sample. To mitigate this concern, a stratified sampling approach was used to ensure that a wide range of depressive symptom levels were represented. This increases the generalizability of our findings to clinical depression (Ingram & Siegle, 2009) since depression is more likely to be dimensional than categorical (Haslam, Holland, & Kuppens, 2012), suggesting that there is value in examining associations between affect dynamics and depressive symptom severity even in a non-clinical sample. Second, while the MB effect has been demonstrated in previous EMA studies (e.g., Peeters et al, 2003; Bylsma et al, 2011; Thompson et al, 2012), examining reactivity of discrete

negative emotions to positive events in daily life is new and should be replicated in future work. A noteworthy limitation of EMA studies is the lack of precision in recording temporal development of events and emotion regulation strategies. However, the temporal unfolding of emotions and emotion regulation strategies is, in fact, a theoretical point of contention in the literature given that these processes can only be directly assessed with self-reports, which are limited in their precision recording dynamic processes. The capacity to report the temporal development of affect and emotion regulation strategies also rely on other processes such as meta-cognition and self-awareness. Finally, we did not record the intensity of positive events and therefore were unable to investigate the impact of a dose response on the MB effect, although other work has discussed the importance of intensity of events in the amplitude of the MB effect (e.g., Panaite et al, 2017).

Despite these limitations, several strengths are worth noting. The study employed a well-defined sample to represent a large variety of depressive symptom severity and hence strengthen generalizability of current findings across the continuum of depressed states. Our replication of prior findings regarding NA reactivity to positive events described by an MB effect gives further credence to the set of results describing findings of a similar effect when investigating discrete emotions. Finally, the current study provided initial evidence that the MB effect is moderated by state use of emotion regulation strategies in daily life. Overall, this is a first investigation of the role of uninstructed deployment of emotion regulation strategies on affective response to hedonic opportunities among a sample of individuals with a large range of depressive symptom levels.

The current findings also have implications for depression prevention and intervention efforts. Given that depression has been conceptualized as a disorder of emotion and emotion regulation (Rottenberg, 2017), understanding the contexts in which hedonic opportunities (such

as the MB effect) are augmented or diminished in daily life is crucial for the design of effective clinical interventions for depression. For example, current therapies could increase focus on teaching patients to recalibrate appraisal of positive events so that less intense events are also valued. Additionally, helping patients rebalance awareness of mood repair to integrate value of both decrease in negative emotions and increases in a variety of positive emotions, not just high intensity positive emotions, is very much in line with new work establishing the role of eudaimonic well-being (e.g., Steger, Kashdan, Oishi, 2008). Our findings may also speak to mechanisms of change in habitual use of emotion regulation to increase well-being (see Houben, et al., 2015; Kashdan & Rottenberg, 2010 for reviews).

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Disclosure of interest

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Tables*Table 1.* Descriptive statistics of baseline and EMA variables.

Variables	Total (N = 97)	High CESD* (n = 51)	Low CESD* (n = 46)
Age	19 (1.2)	19.1 (1.3)	18.9 (1.2)
Gender (% Male)	37%	31%	43%
CESD	14.4 (9.6)	21.2 (8.2)	6.8 (3.0)
NA	15.4 (10.7)	19.9 (12.0)	10.4 (5.9)
Sadness	17.7 (12.6)	23.1 (14.3)	11.7 (6.5)
Anger	14 (9.5)	16.9 (10.8)	10.8 (6.5)
Anxiety	13.2 (10.5)	16.6 (12.5)	9.4 (5.8)
Rumination	27.1 (16.9)	33.5 (19.2)	20.0 (10.1)
Reflection	23.5 (12.4)	23.5 (12.3)	23.4 (12.5)
Reappraisal	18.4 (11.6)	17.6 (10.8)	19.3 (12.4)
Expressive suppression	23.7 (15.4)	28.0 (16.5)	18.9 (12.7)
Sharing	21.4 (12.1)	21.4 (12.7)	21.5 (11.5)
Distraction	29.6 (16.4)	34.8 (16.7)	23.9 (14.2)
Positive events (sum)	17.9 (11.2)	14.9 (9.6)	21.3 (12.1)

Note: CESD = Centre for Epidemiologic Studies Depression; Low and High CESD groups were computed using median split.

Table 2. Pearson's *rs* showing strength of relationships between depressive symptoms, average (across beeps) number of positive events, NA, negative emotions, and use of emotion regulation strategies.

	Depressive symptoms (CES-D)
	<i>r</i>
Number of positive events	-.327**
NA	.625***
Sadness	.611***
Anger	.420***
Anxiety	.482***
Rumination	.541***
Reflection	.110
Reappraisal	.001
Expressive suppression	.380***
Social Sharing	-.062
Distraction	.232*

NA = negative affect; * $p < .05$, ** $p < .01$, *** $p < .001$

Table 3. Emotion regulation moderators of NA and specific negative emotion reactivity during positive events as a function of CES-D scores.

	NA reactivity		Sadness reactivity		Anger reactivity		Anxiety reactivity	
Moderators	y ₄₁	SE	y ₄₁	SE	y ₄₁	SE	y ₄₁	SE
Rumination	-.09*	.04	-.13*	.06	-.16**	.06	-.01	.05
Distraction	.16	.03	.17**	.05	.11*	.05	.11*	.05
Expressive suppression	.13***	.03	.14*	.05	.05	.05	.03	.05
Social sharing	.02	.03	.07	.05	.02	.05	.07	.05
Reappraisal	-.06	.05	-.10	.08	-.23**	.07	.10	.07
Reflection	-.01	.04	.02	.06	-.04	.05	.02	.05

NA = negative affect; * $p < .05$, ** $p < .01$, *** $p < .001$

Figures

Figure 1. Combined negative affect (Panel A), sadness (Panel B), anger (Panel C), and anxiety (Panel D) reactivity to positive events as a function of depressive symptoms. At the within-person level, all emotions were within-person centered with zero reflecting the average emotionality for a particular person. At the between-person level, participants' CESD scores were grand-mean centered, with "low" and "high" representing -1 SD and +1 SD around the grand mean for depressive symptoms, respectively.

Figure 2. The role of rumination (Panel A), suppression (Panel B), and distraction (Panel C) in sadness reactivity to positive events as a function of depressive symptoms. At the within-person level, levels of sadness and emotion regulation strategy use were within-person centered, with zero reflecting the average sadness and use of that strategy for a particular person. For strategy use, we used -1 SD and +1 SD to visualize “low” and “high” emotion regulation, respectively. At the between-person level, participants’ CESD scores were grand-mean centered, with “low” and “high” representing -1 SD and +1 SD around the grand mean for depressive symptoms, respectively.