

# The “Brightening” Effect: Reactions to Positive Events in the Daily Lives of Individuals With Major Depressive Disorder and Generalized Anxiety Disorder

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Depressed individuals are less reactive than healthy individuals to positive stimuli in the laboratory, but accumulating evidence suggests that they are more emotionally reactive to positive events in their daily lives. The present study probed the boundaries of this curious “mood brightening” effect and investigated its specificity to major depressive disorder (MDD) vis-à-vis generalized anxiety disorder (GAD), its closest boundary condition. We used ecological momentary assessment to measure reactions to positive events over one week in individuals with MDD ( $n = 38$ ), GAD ( $n = 36$ ), comorbid MDD-GAD ( $n = 38$ ), and no psychopathology ( $n = 33$ ). Depressed individuals responded to positive events with larger changes in affect, cognition, reported withdrawal (but not approach) behavior, and symptoms than healthy controls. More severe depression assessed before the sampling week predicted greater brightening. Altered reactivity to positive events was relatively specific to MDD when compared with GAD, similar to patterns found for

other positive emotional processes. The robustness, scope, and relative specificity of the brightening effect highlights the need to resolve conflicting findings across laboratory and non-laboratory studies to advance understanding of altered reactivity in emotional disorders.

*Keywords:* depression; anxiety; anhedonia; comorbidity; ecological momentary assessment

ANHEDONIA, A LOSS OF INTEREST or pleasure in previously enjoyed activities, is one of two cardinal symptoms of major depressive disorder (American Psychiatric Association, 2013). As a relatively homogeneous construct with an increasingly well-defined neural circuitry, anhedonia is viewed as a promising target for illuminating the etiology of MDD (Workshop Proceedings, 2011). Anhedonia is also an important clinical target, given its impact on daily functioning (Hopko, Lejuez, Ruggiero, & Eifert, 2003) and its prediction of poor treatment response (Spijker, Bijl, de Graaf, & Nolen, 2001), chronic course of illness (Moos & Cronkite, 1999), and risk for future depression (Wardenaar, Giltay, van Veen, Zitman, & Penninx, 2012).

In line with clinical descriptions of anhedonia, experimental research has generally found that depressed individuals are less reactive to positive stimuli than nondepressed individuals (Bylsma, Morris, & Rottenberg, 2008; Dichter, 2010).

Sections of this paper were included in a poster presented at the Anxiety and Depression Association of America Annual Conference in April 2016.

This work was supported in part by the National Institute of Mental Health [grant R01 MH094425 awarded to Ayelet Meron Ruscio].

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Assessed via self-report, behavioral, or neural measures, depressed individuals typically show reduced reactivity to positive images (e.g., Dunn, Dalgleish, Lawrence, Cusack, & Ogilvie, 2004), words (e.g., Canli et al., 2004), films (e.g., Rottenberg, Kasch, Gross, & Gotlib, 2002), and monetary rewards (e.g., Pizzagalli et al., 2009) presented in the laboratory (but see Swiecicki et al., 2009, for a study that did not find differences in reactivity to pleasant smells and tastes).

Surprisingly, studies outside the laboratory have found exactly the opposite: depressed individuals show *greater* emotional reactivity to positive experiences than nonclinical controls. In ecological momentary assessment (EMA) studies, depressed individuals report larger increases in positive affect (Peeters, Nicolson, Berkhof, Delespaul, & deVries, 2003) and larger declines in negative affect (Bylsma, Taylor-Clift, & Rottenberg, 2011; Peeters et al., 2003; Thompson et al., 2012) than nondepressed individuals following positive events in their daily lives. Past studies have also ruled out several methodological explanations for this unexpected “mood brightening” effect. Bylsma et al. (2011) and Thompson et al. (2012) controlled for average daily affect levels and for affect levels at the signal prior to the event,<sup>1</sup> respectively, ruling out differences in baseline affect between depressed and nondepressed individuals as an explanation for brightening. Bylsma et al. (2011) also utilized objective event coding to test whether depressed individuals had higher thresholds for rating events as positive than nonclinical controls, and found no differences in ratings thresholds between groups. Peeters et al. (2003) controlled for event frequency and found that the lower frequency of positive events among depressed relative to nondepressed individuals did not account for brightening. All three studies found that while depressed individuals’ responses to positive events were enhanced relative to controls, this difference did not extend to negative events (Bylsma et al., 2011; Peeters et al., 2003; Thompson et al., 2012), suggesting that brightening is specific to positive experiences.

Although several EMA studies have now reported this surprising mood brightening effect, the boundaries of the effect are poorly understood. As prior studies focused on affective responses to positive events, it is unknown whether brightening extends to other aspects of internal experience, to overt behavior, or to clinical symptoms. Measuring reactivity across a wider range of outcomes would clarify the magnitude and extent of the brightening

effect, as well as shed light on types of responses that are particularly amenable to intervention efforts.

Prior studies also compared depressed participants only to nonclinical controls. As most individuals with MDD have a comorbid anxiety disorder (Ruscio & Khazanov, 2017), isolating abnormalities of emotional responding that are specific to MDD requires comparisons with anxiety. Generalized anxiety disorder (GAD), the disorder most strongly associated with MDD (Goldberg, Kendler, Sirovatka, & Regier, 2010), offers a particularly stringent test for specificity. Theoretical models and psychometric studies suggest that positive emotional processes should distinguish MDD from GAD (Watson, 2009; Watson & Naragon-Gainey, 2010). However, these models presume that overall levels of positive emotionality are diminished in depression, leaving open the question of whether heightened responding to positive stimuli distinguishes the disorders. No study has yet compared the reactions of depressed and anxious individuals to positive events in daily life, nor evaluated whether comorbid anxiety influences event responding in those with MDD. Establishing the specificity of the brightening effect is important for illuminating processes that are common across emotional disorders versus those that are uniquely important for understanding, and treating, depression.

### Study Aims

The present study used EMA to characterize reactions to positive events in the lives of individuals with MDD, GAD, comorbid MDD-GAD, or no psychopathology. The study had two aims. Our first aim was to broaden understanding of differences in positive event responding between depressed and nondepressed individuals by assessing affective responses alongside cognitive, behavioral, and symptomatic responses to daily events.

Our assessment of cognitive responses focused on rumination and worry. These cognitive processes are robust predictors of depressive and anxiety symptoms (Watkins, 2008) and share particularly close relationships with MDD and GAD (Ehring & Watkins, 2008; Kircanski, Thompson, Sorenson, Sherdell, & Gotlib, 2015). Promising early studies with nonclinical samples have reported larger declines in rumination (Takano, Sakamoto, & Tanno, 2013) and other depressive cognitions (Nezlek & Gable, 2001) following positive daily events in dysphoric undergraduates compared to less-dysphoric peers, hinting at the potential value of cognitive constructs for probing the brightening effect in MDD.

We also examined behavioral responses to daily positive events. As several prominent theoretical models organize behavioral responses into

<sup>1</sup>The analyses presented in this paper also control for levels of the outcome variable assessed at the signal prior to the event.

approach versus withdrawal behaviors (Davidson, 1998; Gray, 1994), we included measures of both behavior categories as reported by participants. Whereas depression and anxiety have both been linked to heightened withdrawal behaviors, only depressed individuals are thought to exhibit blunted approach behaviors (Shankman & Klein, 2003). Evidence of abnormal approach or withdrawal reactions in the wake of positive events could shed new light on how disorders are maintained in daily life, as well as identify behaviors with high potential as treatment targets.

The last outcomes we assessed were symptoms of MDD and GAD. Previous studies have examined differences in event responding between depressed and nondepressed groups, but have not examined within-person associations between positive events and momentary symptoms of depression. We were interested in testing the extent to which events “moved” symptoms of depression and anxiety experienced throughout the day. In addition to comparing depressed and nondepressed individuals on a variety of response dimensions, we sought to identify clinical characteristics that predict heightened responding in order to better understand which specific features of depression and anxiety are associated with mood brightening.

Our second aim was to evaluate the specificity of the brightening effect to MDD versus GAD. We examined whether responses to positive events distinguished individuals with MDD from those with GAD, and whether the responses of individuals with comorbid MDD-GAD differed from those with MDD alone. We tested both GAD and MDD severity as predictors of positive event responding, individually and when controlling for one another. In addition to disorder severity, we investigated other clinical characteristics of GAD and MDD as predictors of brightening. Lastly, we examined positive events as predictors of momentary GAD, as well as MDD, symptoms.

Based on the results of previous EMA studies, we hypothesized that depressed (MDD and comorbid) participants would rate their daily events as less positive and report fewer positive events than nondepressed (GAD and control) participants. Previous research also prompted us to hypothesize that affective reactions to events rated as positive by participants would be amplified in persons with MDD, although we lacked a strong basis for predicting whether the brightening effect would extend to cognitions, reported behavior, and symptoms. Finally, the work previously described suggesting that positive emotional processes distinguish depression from anxiety led us to speculate that the brightening effect would be specific to MDD vis-à-vis GAD.

## Materials and Methods

### PARTICIPANTS

The present study has previously been described (Ruscio et al., 2015). Participants were recruited from the community through electronic (Craigslis) and print (flyers) advertisements, and from the student body of a private university through a psychology department website. Participants first completed a screening survey online or by phone that included the Generalized Anxiety Disorder Questionnaire (GAD-Q-4; Newman, Zuellig, Kachin, Constantino, & Cashman, 2002) and the Diagnostic Inventory for Depression (DID; Zimmerman, Sheeran, & Young, 2003), self-report measures of DSM-IV symptoms of GAD and MDD. Those reporting MDD and/or GAD symptoms above diagnostic thresholds were eligible for the clinical groups. Those reporting symptom levels below diagnostic thresholds and no history of depression or anxiety that significantly interfered with their lives, as well as scoring below 56 on the Penn State Worry Questionnaire (Meyer, Miller, Metzger, & Borkovec, 1990; Molina & Borkovec, 1994), were eligible for the control group.

After providing informed consent, eligible participants were administered the Anxiety Disorders Interview Schedule for DSM-IV–Lifetime Version (ADIS-IV-L; DiNardo, Brown, & Barlow, 1994). Inclusion in one of the three clinical groups required a current, principal diagnosis of MDD or GAD, defined as the disorder currently causing the greatest interference and distress (Brown, Di Nardo, Lehman, & Campbell, 2001). Active psychosis and active suicidal intent, assessed during the ADIS, were exclusion criteria for all groups. Having a current substance use disorder was also an exclusion criterion for all groups, given its high potential to compromise the accuracy of participants’ responses. Approximately 65% of interviewed participants met these eligibility criteria and were enrolled in the study. Of the 151 participants who began the study, two participants’ data were lost due to technical problems, one participant did not return the electronic diary, and three participants withdrew due to time conflicts.

The final sample included 145 participants (127 community residents and 18 students) from the following four groups: (1) an MDD group ( $n = 38$ ) diagnosed with MDD but not GAD, (2) a GAD group ( $n = 36$ ) diagnosed with GAD but not MDD, (3) a comorbid group ( $n = 38$ ) diagnosed with both GAD and MDD, and (4) a control group ( $n = 33$ ) with no current or past psychopathology and no mental health treatment. Consistent with patterns in representative community samples (Ruscio & Khazanov, 2017) and with the episodic course of MDD, most participants (61%) in the GAD group

had past MDD, whereas one participant (4%) in the MDD group had past GAD. The four groups did not differ in education, marital status, or race/ethnicity (see Table 1). They did, however, differ in age and sex: the MDD group was older than the control group, and the GAD group included more females than the comorbid group. We therefore included age and sex as covariates in all multilevel models.

#### PROCEDURE

During the first laboratory visit, participants were interviewed by a diagnostician with a master's or bachelor's degree in psychology who had received extensive training on the clinical interviews and achieved a high level of interrater agreement with the supervising licensed psychologist. The clinical assessment team discussed each interview and reached decisions about diagnosis and clinical severity by consensus. A second rater who was blind to initial diagnoses independently rated a random subset of the audio-recorded interviews ( $n = 32$ ). Interrater reliability was high for both GAD ( $K = 1.00$ ) and MDD ( $K = 0.88$ ) diagnoses.

Participants attended an orientation session, after which they completed the experience sampling protocol for 1 week. During this week, participants responded to prompts from an electronic device (Palm Pilot Z22) eight times daily during the 12-hour period they selected (typically 10 A.M.–10 P.M.; 12% of participants chose a different 12-hour

period that better fit their schedules). They were signaled using a time-stratified random sampling strategy in which one signal occurred at a random time within each 90-minute block, with signals separated by at least 20 minutes. Upon receiving the auditory signal, participants had 15 minutes to respond before the signal was coded as missed. Participants could set the device not to signal for one hour if they were entering a situation in which responding was infeasible or unsafe.

At each assessment, participants first rated their thoughts, feelings, behaviors, and symptoms at the time they were signaled (Time 1, or T<sub>1</sub>). They then described the most significant positive or negative event that occurred since the previous signal, defined as the event that had the biggest impact on them. After answering questions about the event, participants rated the thoughts and feelings they experienced immediately after the event (Time of Event, or T<sub>E</sub>). Behaviors and symptoms were not rated again at this time to minimize participant burden. T<sub>1</sub> and T<sub>E</sub> ratings consequently were made at the same assessment, with T<sub>1</sub> ratings describing the participant's current state and T<sub>E</sub> ratings reporting retrospectively on an event that occurred since the previous signal (0 to 180 minutes earlier). T<sub>1</sub> ratings preceded T<sub>E</sub> ratings to avoid the potential impact of recalling emotionally evocative events on ratings of current experience.

Participants were introduced to these procedures and completed two practice assessments during the

Table 1  
Demographic and Clinical Characteristics of the Sample by Group

Variable	Control ( $n = 33$ )	MDD ( $n = 38$ )	Comorbid ( $n = 38$ )	GAD ( $n = 36$ )
Demographic characteristics				
Age*	28.61 (10.42) <sub>a</sub>	36.38 (12.33) <sub>b</sub>	33.60 (11.35) <sub>a,b</sub>	31.62 (9.24) <sub>a,b</sub>
% Female*	66.7 <sub>a,b</sub>	71.1 <sub>a,b</sub>	52.6 <sub>a</sub>	83.3 <sub>b</sub>
% Caucasian	54.5	54.1	56.8	63.9
Marital Status				
Never married	75.0	60.5	71.1	47.2
Married or cohabiting	15.6	26.3	18.4	44.4
Previously married	9.4	13.2	10.5	8.3
Education				
High school or lower	6.1	10.8	10.5	8.3
Some college	48.5	29.7	31.6	27.8
College degree or higher	45.5	59.5	57.9	63.9
Clinical characteristics				
GAD severity <sup>***</sup>	0.54 (0.91) <sub>a</sub>	3.99 (1.60) <sub>b</sub>	4.95 (1.02) <sub>c</sub>	4.89 (0.66) <sub>c</sub>
MDD severity <sup>***</sup>	0.18 (0.53) <sub>a</sub>	5.16 (0.82) <sub>b</sub>	5.16 (0.74) <sub>b</sub>	2.19 (1.13) <sub>c</sub>
Current comorbid disorders <sup>a***</sup>	0.00 (0.00) <sub>a</sub>	0.90 (0.96) <sub>b</sub>	1.33 (1.36) <sub>b</sub>	0.81 (0.82) <sub>b</sub>
Past comorbid disorders <sup>a**</sup>	0.00 (0.00) <sub>a</sub>	0.77 (1.10) <sub>b</sub>	1.00 (1.12) <sub>b</sub>	0.88 (1.56) <sub>b</sub>

Note. MDD = major depressive disorder. GAD = generalized anxiety disorder;  $M (SD)$  are presented for dimensional variables; all other values represent percentages. Values in the same row that do not share subscripts differ at  $p < .05$ .

\* $p < .05$ . \*\* $p < .01$ . \*\*\* $p < .001$ .

<sup>a</sup> Number of anxiety, mood, and substance-related disorders, excluding MDD and GAD.

orientation session, with the week-long study beginning the next morning. Participants were contacted on the second day to confirm that study procedures were being followed and were debriefed in person after 7 days. A research ethics committee approved all procedures.

## MEASURES

### EMA Variables

*Event Valence.* Participants rated the valence of each  $T_E$  event on a Likert scale ranging from 1 (*very negative*) to 7 (*very positive*), with 4 being neutral. Similar to a previous study of positive events among individuals with depression (Bylsma et al., 2011), we considered positive events to be events rated moderately or highly positive (6 or 7 on the 7-point scale) and compared these to all other events (rated 1-5 on the 7-point scale).

### Outcomes

*Affect.* Emotions experienced immediately after the  $T_E$  event and at the signal were rated using three positive affect (PA; happy, proud, determined) and three negative affect (NA; sad, anxious, dissatisfied with myself) terms capturing dimensions of emotional experience important for MDD and GAD. The terms were drawn from the basic emotion scales of the expanded Positive and Negative Affect Schedule (PANAS-X; Watson & Clark, 1994), although we used “anxious” instead of a term from the Fear scale given the greater relevance of anxiety than fear for GAD (Roemer, Orsillo, & Barlow, 2002). Each emotion was rated on a Likert scale ranging from 1 (*not at all*) to 5 (*very much*). Items were averaged at each time point to form momentary PA (within-person  $\omega = .63-.72$ ) and NA ( $\omega = .75-.77$ ) variables. Within-person  $\omega$  was calculated using estimates from the within-subjects portion of a confirmatory factor model; these coefficients can be interpreted like Cronbach’s alpha (Bolger & Laurenceau, 2013). Between-person correlations, calculated from pseudo  $R^2$  extracted from hierarchical models (Bryk & Raudenbush, 1992), were high between momentary PA and trait PA assessed by the Positive and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988; .60–.63). Momentary NA correlated highly with trait NA on the PANAS (.60–.61).

*Cognition.* Participants rated the extent to which they were ruminating and worrying immediately after the  $T_E$  event and at each signal. Worry was assessed with the items “I was worrying about how things will turn out” and “I kept thinking about something bad that COULD happen.” Rumination was assessed with the items “I kept thinking about something negative that has happened” and “I was dwelling on my

mistakes, failures, or losses.” These items were based on theoretical and empirical descriptions of negative, repetitive thought as central to both worry and rumination, with worry focusing on future events and rumination focusing on past events and themes of personal failure (Ehring & Watkins, 2008; Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008). Items rated on separate 1–5 scales were averaged to form momentary worry (within-person  $\alpha = .84-.85^2$ ) and rumination ( $\alpha = .82-.85$ ) variables. Within-person  $\alpha$  was also calculated using estimates from the within-subjects portion of a confirmatory factor model and can be interpreted like Cronbach’s alpha (Geldhof et al., 2014). Momentary worry correlated highly with trait worry assessed by the Penn State Worry Questionnaire (.58–.59). Momentary rumination correlated highly with trait rumination assessed by the Ruminative Responses Scale (Nolen-Hoeksema & Morrow, 1991; .57–.58).

*Reported Behavior.* At each signal, participants rated the extent to which they were currently engaging in approach and withdrawal behaviors on a 1–5 scale. Approach behaviors included social engagement (“seeking out or connecting with other people”) and productive activity (“keeping active and busy”). Withdrawal behaviors included social withdrawal (“distancing or isolating myself from others”) and inactivity (“unable to make myself get up and do things”). We assessed social interaction and activity level to represent the domains typically included in measures of behavioral approach and withdrawal (Ball & Zuckerman, 1990; Carver & White, 1994). Reported behaviors were analyzed separately as they did not form reliable composites.

*Symptoms.* At each signal, participants were presented with each *DSM-IV* symptom of MDD and GAD and indicated which, if any, they were currently experiencing. Given the large number of symptoms included, dichotomous responses were collected to minimize participant burden. Endorsement of suicidal ideation branched to emergency referrals. The symptom ratings were summed into MDD (15 items) and GAD (9 items) composites, which correlated strongly with ADIS clinical severity ratings for MDD (.58) and GAD ( $r = .52$ ), respectively.

### Clinical Variables

*Clinical Predictors.* Clinical predictors were derived from the ADIS. Interviewers rated the overall severity of MDD (ICC = 0.97) and GAD (ICC = 0.97) using 0–8 scales. MDD and GAD

<sup>2</sup> Within-person  $\alpha$  was calculated as it is an acceptable alternative when the model for calculating within-person  $\omega$  fails to converge (Geldhof, Preacher, & Zyphur, 2014).

severity were strongly related, but differentiable ( $r = .63, p < .001$ ). They also assessed the course of MDD and GAD, including current episode duration among current cases, age of onset of the first episode, history of single vs. recurrent episodes, and lifetime persistence (total months in episode over the lifetime) among lifetime cases. Other clinical predictors included the number of current and past comorbid disorders (out of 13 anxiety, mood, and substance-related disorders other than MDD and GAD), history of mental health treatment (pharmacotherapy or psychotherapy), and family history of psychopathology (any mental health diagnosis or treatment in first- or second-degree relatives).

#### STATISTICAL ANALYSES

Means were compared across diagnostic groups using SPSS v23 (IBM SPSS Statistics for Macintosh, 2015). Multilevel analyses were performed using Hierarchical Linear and Nonlinear Models 7.01 (Raudenbush, Bryk, & Congdon, 2013). All models included age and sex as covariates. For outcome variables demonstrating significant time-of-day effects, time was included as a covariate as well. EMA assessments were in a two-level model, nested within individuals. Continuous level 1 variables were centered around each individual's mean and continuous level 2 variables were centered around the overall sample mean. One type of model tested associations between event valence rated retrospectively ( $T_E$ ) and outcomes occurring either immediately after the event ( $T_E$ ) or one signal after the event ( $T_1$ ); these event and outcome variables were assessed at the same signal. The second type of model examined time-lagged associations between event valence ( $T_E$ ) and outcomes two signals after the event ( $T_2$ ); these event and outcome variables were assessed at consecutive signals, always within the same day. The role of positive events was investigated by including in models a dichotomous variable in which positive events were contrasted with all other events. As described below, most analyses presented controlled for the outcome variable assessed at the previous signal.

Multilevel regression models were performed using assessments from the entire sample. As we were interested in comparing the event responses of depressed individuals to controls and to individuals with GAD, some models tested whether the relationships of interest differed for the MDD and comorbid groups versus the control group, or for the MDD and comorbid groups versus the GAD group. To test whether comorbid GAD influenced responsiveness to positive events, we also tested whether the relationships of interest differed for the MDD group versus the comorbid group. To test for moderation by diagnoses, we constructed dichotomous variables for each

contrast. For example, depressed participants were compared to controls using a dummy variable in which MDD and comorbid participants were coded 1 and controls were coded 0. As an illustration of the analyses performed, the relationship between positive events and rumination can be described by the following equation:

$$\text{rumination}_{ij} = \beta_{0j} + \beta_{1j}(\text{positive event}_{ij}) + r_{ij}$$

where  $\text{rumination}_{ij}$  is the rumination rating for individual  $j$  at observation  $i$ ; intercept  $\beta_{0j}$  is the expected rumination rating for non-positive events; slope  $\beta_{1j}$  is the expected change in rumination with the occurrence of a positive event for individual  $j$ ;  $\text{positive event}_{ij}$  is whether a positive or non-positive event was reported at observation  $i$  for individual  $j$ ; and  $r_{ij}$  is the error term associated with observation  $i$  for individual  $j$ . These Level 1 intercepts and slopes for individual  $j$  can then be predicted at Level 2 by the following equations:

$$\begin{aligned} \beta_{0j} = & \gamma_{00} + \gamma_{01}(\text{Age}) + \gamma_{02}(\text{Sex}) \\ & + \gamma_{03}(\text{MDD and comorbid groups vs. controls}) \\ & + u_{0j} \end{aligned}$$

$$\begin{aligned} \beta_{1j} = & \gamma_{10} + \gamma_{11}(\text{Age}) + \gamma_{12}(\text{Sex}) \\ & + \gamma_{13}(\text{MDD and comorbid groups vs. controls}) \\ & + u_{1j} \end{aligned}$$

where  $\gamma_{00}$  is the intercept for control participants; coefficients  $\gamma_{01}$  through  $\gamma_{03}$  indicate the expected change in the average intercept attributable to between-person variance in age, sex, or MDD status;  $u_{0j}$  is the unique increment to the intercept associated with individual  $j$ ;  $\gamma_{10}$  is the regression slope for control participants; coefficients  $\gamma_{11}$  through  $\gamma_{13}$  indicate the expected change in the average regression slope attributable to between-person variance in age, sex, or MDD status; and  $u_{1j}$  is the unique increment to the slope associated with individual  $j$ . This model was then repeated with the inclusion of a covariate adjusting for the status of rumination at the previous observation ( $\text{rumination}_{i-1}$ ). We focused on the average within-person regression coefficients ( $\gamma_{10}$ ) and their moderation by diagnostic status ( $\gamma_{13}$ ). We then tested MDD and GAD severity, as well as other clinical predictors (e.g., number of current comorbid disorders) as moderators of within-person regression coefficients in the full sample.

In some analyses, we included group variables as covariates. In these analyses, we constructed three dichotomous variables comparing the MDD, GAD, and comorbid groups to the control group and included all three diagnostic variables in the model.

## Results

### PRELIMINARY ANALYSES

Participants received 7,988 signals and completed 5,724 assessments. The mean response rate was 72% ( $SD = 12.7$ , range 41–98%), similar to response rates in validation studies with depressed and anxious samples (Husky et al., 2010; Johnson et al., 2009). The proportion of completed assessments did not differ by signal, diagnostic group, or any clinical predictor.

### FREQUENCY OF POSITIVE EVENTS IN MDD AND GAD

A total of 5,700 events were reported during the sampling week. Events ranged in valence from 1 (*very negative*) to 7 (*very positive*), with the average event rated about neutral ( $M = 4.30$ ,  $SD = 0.72$ ). Mean event positivity was significantly lower in the three clinical groups (MDD = 4.00 [.65]; Comorbid = 4.05 [.62]; GAD = 4.33 [.62]) than the control group ( $M = 4.88$ ,  $SD = 0.68$ ),  $F(3, 141) = 13.78$ ,  $p < .001$ . In total, 22% of events were rated as moderately or highly positive. The MDD and comorbid groups each reported significantly fewer positive events (16%) than controls (32%), with the GAD group intermediate (24%),  $F(3, 141) = 5.01$ ,  $p = .002$ . While this pattern held when comparing positive to only neutral events,  $F(3, 141) = 3.33$ ,  $p = .021$ , power was reduced and the MDD and comorbid groups differed from controls only at a trend level (both  $p < .058$ ).

### ASSOCIATIONS AMONG OUTCOME VARIABLES

We examined within-person relationships among the outcome variables by calculating the proportion of variance explained in one variable when the other variable was added to the hierarchical model (see Supplemental Table 1; Bryk & Raudenbush, 1992), although these effect size estimates should be interpreted with caution (Kreft & De Leeuw, 1998). The largest associations were observed between MDD and GAD symptoms (which shared 57% of their variance) and between NA, worry, and rumination (which shared 31%–61% of their variance); these outcomes overlapped substantially yet were still differentiable. The associations were lower for symptoms with affective and cognitive outcomes (11%–38% shared variance) and lowest for PA and reported behaviors with all other outcomes (2%–23% shared variance).

### POSITIVE EVENTS AS PREDICTORS OF LEVEL OF, AND CHANGE IN, OUTCOMES

Mean levels of the outcome variables (1) over the week and (2) following positive events are shown in Supplemental Tables 2 and 3, respectively. To

predict overall levels of functioning following positive events, we ran a first set of models using the occurrence of a positive event to predict the level of each outcome (see Table 2, left column). Positive events predicted higher levels of all positive outcomes (positive affect and approach behaviors) and lower levels of all negative outcomes (negative affect, rumination, worry, withdrawal behaviors, and symptoms of MDD and GAD). The associations were observed mainly within one signal (up to 180 minutes) after the event, although associations with three of four reported behaviors were still evident two signals later, all  $\gamma > -0.33$ , all  $p < .040$ . The occurrence of a positive event predicted higher levels of positive outcomes and lower levels of negative outcomes more strongly for depressed (MDD and comorbid) participants than controls (all  $\gamma > 0.24$ , all  $p < .008$ ), with the exception of PA two signals after the event and reported approach behaviors at all time points. Positive events predicted lower levels of a few negative outcomes more strongly for depressed (MDD and comorbid) participants than GAD participants, and for participants with comorbid rather than pure depression, but these differences were inconsistent across time points.

We reran each model controlling for the level of the outcome variable at the signal prior to the event in order to evaluate positive events as a predictor of change in these outcomes and to rule out the possibility that baseline differences in outcome variables could account for the findings (T0; see Table 2, right column). Positive events predicted increases in PA and reductions in NA, rumination, and worry immediately after the event and at the next signal, all  $\gamma > -0.51$ , all  $p < .001$ . Positive events also predicted increased approach and decreased withdrawal behaviors, and decreased symptoms of MDD and GAD up to one signal later, all  $\gamma > -0.42$ , all  $p < .031$ . These changes largely dissipated two signals after the event, although reductions in inactivity persisted two signals (up to 4.5 hours) after the event.

Replicating the mood brightening effect, depressed (MDD and comorbid) participants showed larger increases in PA and larger reductions in NA than controls after experiencing a positive event, both immediately after the event and at the next signal, all  $\gamma > 0.17$ , all  $p < .039$ . Extending the mood brightening effect, depressed participants also reported larger reductions in rumination and worry immediately after the event and larger reductions in rumination, social withdrawal, inactivity, and MDD and GAD symptoms at the next signal than controls, all  $\gamma > -0.25$ , all  $p < .010$ . While most of these differences did not extend past

Table 2  
Occurrence of Positive Events Predicting Level of, and Change in, Outcomes

Outcome	Level of outcome			Change in outcome		
	$\gamma$	SE	Contrasts	$\gamma$	SE	Contrasts
Immediately after event (T <sub>E</sub> )						
Positive affect	1.20	.20***	Dep > Cont***	1.08	.21***	Dep > Cont**
Negative affect	-0.60	.17***	Dep > Cont***, Dep > GAD*	-0.60	.15***	Dep > Cont***, Dep > GAD*
Rumination	-0.62	.17***	Dep > Cont***, Dep > GAD*	-0.65	.16***	Dep > Cont***
Worry	-0.65	.18***	Dep > Cont***	-0.66	.17***	Dep > Cont***
One signal after event (T <sub>1</sub> )						
Positive affect	0.89	.16***	Dep > Cont**	0.91	.17***	Dep > Cont*
Negative affect	-0.47	.15**	Dep > Cont***, Com > MDD*	-0.51	.15***	Dep > Cont***
Rumination	-0.46	.13***	Dep > Cont***	-0.54	.14***	Dep > Cont***
Worry	-0.57	.15***	Dep > Cont***, Com > MDD*	-0.58	.15***	
Reported approach behaviors						
Social engagement	1.23	.23***		1.24	.30***	
Productive activity	0.71	.18***		0.70	.23**	
Reported withdrawal behaviors						
Social withdrawal	-0.50	.20*	Dep > Cont**	-0.50	.21*	Dep > Cont**
Inactivity	-0.33	.14*	Dep > Cont***, Dep > GAD*	-0.42	.16*	Dep > Cont*
MDD symptoms	-0.71	.34*	Dep > Cont***	-0.79	.36*	Dep > Cont***
GAD symptoms	-0.76	.27**	Dep > Cont***	-0.65	.29*	Dep > Cont***
Two signals after event (T <sub>2</sub> )						
Positive affect	0.26	.14		0.01	.16	
Negative affect	-0.09	.16	Dep > Cont***	-0.20	.12	
Rumination	0.00	.18	Dep > Cont**, Com > MDD*	-0.14	.14	
Worry	0.07	.18	Dep > Cont*, Com > MDD*	-0.13	.16	
Reported approach behaviors						
Social engagement	0.58	.21**		-0.14	.27	
Productive activity	0.66	.25*		0.15	.25	
Reported withdrawal behaviors						
Social withdrawal	-0.03	.17	Dep > Cont***, Dep > GAD*	0.25	.25	
Inactivity	-0.43	.14**	Dep > Cont***, Dep > GAD***	-0.33	.14*	Dep > Cont*
MDD symptoms	-0.34	.26	Dep > Cont***, Dep > GAD*	-0.22	.28	Dep > Cont***, Dep > GAD**
GAD symptoms	-0.10	.24	Dep > Cont**	-0.19	.23	

Note. MDD = major depressive disorder; GAD = generalized anxiety disorder; Com = comorbid MDD and GAD; Dep = depressed (MDD and Com groups); Cont = control. T<sub>E</sub> and T<sub>1</sub> outcomes were assessed at the same sampling occasion. Models predicting level of outcome include age, sex, and time of day as covariates. Models predicting change in outcome include age, sex, time of day, and prior level of the outcome variable (at signal T<sub>0</sub>) as covariates. All contrast effects are in the same direction as the coefficient for the main effect.

\* $p < .05$ . \*\* $p < .01$ . \*\*\* $p < .001$ .

the first signal after the event, depressed participants continued to report larger reductions in inactivity and MDD symptoms two signals after a positive event than controls, both  $\gamma > -0.18$ , both  $p < .030$ . Most differences between depressed (MDD and comorbid) and GAD participants, and all differences between pure and comorbid MDD participants, declined to nonsignificance once prior levels of the outcome variable were controlled.

We performed follow-up analyses to check whether the greater changes reported by depressed participants following positive events resulted in outcome levels similar to controls. With the exception of reported approach behaviors, which did not consistently distinguish among study groups, depressed individuals reported lower levels of PA and higher levels of all

negative outcomes than controls immediately after the event and at the next signal, even after controlling for event positivity, all  $\gamma > -0.44$ , all  $p < .006$ . In other words, depressed individuals were still functioning more poorly than controls despite experiencing greater change in these outcomes following positive events.

#### MDD AND GAD SEVERITY AS PREDICTORS OF POSITIVE EVENT RESPONDING

To further investigate the relative contribution of MDD and GAD to positive event responding, we repeated the analyses using MDD and GAD severity as predictors. As described above, the models controlled for the level of the outcome variable at the signal prior to the event so that we



could examine change in outcomes following positive events. We included as predictors only MDD severity, only GAD severity (see Table 3, left columns), and then both MDD and GAD severity entered simultaneously so that they controlled for one another (see Table 3, right columns). When examined separately, MDD and GAD severity predicted change in outcomes following positive events to similar degrees. When controlling for one another, however, MDD severity continued to predict greater changes in negative affect, rumination, and worry immediately after the event; rumination, inactivity, and symptoms of MDD and GAD at the next signal; and symptoms of MDD two signals after the event, all  $\gamma > 0.19$ ,

all  $p < .024$ . By contrast, GAD severity continued to predict only greater changes in rumination immediately after the event ( $\gamma = -0.05$ ,  $p = .033$ ). Surprisingly, GAD severity predicted fewer changes in social engagement two signals after the event on its own and when controlling for MDD severity (both  $\gamma > -0.08$ , both  $p < .008$ ) despite not significantly predicting this outcome one signal after the event (both  $\gamma = -0.01$ , both  $p > .768$ ).

#### OTHER CLINICAL PREDICTORS OF CHANGE IN AFFECT FOLLOWING POSITIVE EVENTS

Finally, we used clinical features aside from MDD and GAD severity to predict change in PA and NA immediately following the positive event ( $T_E$ ). We

Table 3  
Occurrence of Positive Events Predicting Change in Outcomes

Outcome	MDD severity only		GAD severity only		Controlling for the severity of the other disorder			
	$\gamma$	SE	$\gamma$	SE	MDD severity		GAD severity	
	$\gamma$	SE	$\gamma$	SE	$\gamma$	SE	$\gamma$	SE
Immediately after event ( $T_E$ )								
Positive affect	0.05	.02*	0.08	.03**	<0.00	.03	0.08	.04
Negative affect	-0.12	.01***	-0.10	.02***	-0.10	.02***	-0.03	.02
Rumination	-0.11	.02***	-0.11	.02***	-0.08	.02***	-0.05	.02*
Worry	-0.09	.02***	-0.09	.02***	-0.07	.03**	-0.04	.03
One signal after event ( $T_1$ )								
Positive affect	0.03	.02	0.04	.02*	<0.00	.02	0.04	.03
Negative affect	-0.06	.01***	-0.05	.02**	-0.05	.02	-0.02	.03
Rumination	-0.05	.01***	-0.04	.02*	-0.05	.02*	-0.01	.02
Worry	-0.03	.02	-0.03	.02	-0.02	.03	-0.02	.03
Approach behaviors								
Social engagement	-0.01	.03	-0.01	.03	<0.00	.04	-0.01	.04
Productive activity	<0.00	.03	-0.01	.03	0.02	.03	-0.02	.04
Withdrawal behaviors								
Social withdrawal	-0.06	.02**	-0.09	.02***	-0.03	.03	-0.07	.03
Inactivity	-0.05	.02*	-0.03	.02	-0.06	.02*	<0.00	.02
MDD symptoms	-0.18	.04***	-0.10	.04*	-0.19	.06**	0.02	.06
GAD symptoms	-0.12	.03***	-0.08	.04*	-0.12	.04**	-0.01	.05
Two signals after event ( $T_2$ )								
Positive affect	-0.01	.01	<0.00	.02	-0.01	.02	0.01	.02
Negative affect	-0.02	.01	-0.01	.01	-0.02	.02	0.01	.02
Rumination	-0.02	.01	-0.02	.01	-0.02	.02	-0.01	.02
Worry	-0.03	.02	-0.02	.02	-0.04	.02	0.01	.02
Approach behaviors								
Social engagement	-0.04	.03	-0.08	.03**	0.02	.03	-0.10	.03**
Productive activity	0.02	.03	-0.01	.03	0.04	.03	-0.04	.04
Withdrawal behaviors								
Social withdrawal	-0.02	.03	0.01	.03	-0.04	.04	0.03	.04
Inactivity	-0.04	.02*	-0.02	.02	-0.04	.02	<0.00	.02
MDD symptoms	-0.11	.04**	-0.04	.04	-0.14	.05**	0.05	.04
GAD symptoms	-0.04	.03	-0.04	.03	-0.02	.03	-0.03	.03

Note. MDD = major depressive disorder; GAD = generalized anxiety disorder. TE and T1 outcomes were assessed at the same sampling occasion. All models include age, sex, time of day, and prior level of the outcome variable (at signal T0) as covariates. The two left columns include only MDD or GAD severity as predictors. The two right columns include both MDD and GAD severity as predictors in the same model, thereby controlling for the other disorder.

\* $p < .05$ . \*\* $p < .01$ . \*\*\* $p < .001$ .

focused on affective outcomes to hold down the number of analyses while connecting our study to prior research on anhedonia, in which short-term affective responses are the most common outcomes (Bylsma et al., 2008; Treadway & Zald, 2011). Confirming our previous findings, measures of clinical severity were associated with greater brightening. Individuals with a larger number of current (but not past) comorbid disorders showed greater changes in positive affect ( $\gamma = 0.55$ ,  $p = .029$ ) and negative affect ( $\gamma = -0.78$ ,  $p < .001$ ) following positive events. Individuals with a family history of psychopathology and past (but not current) mental health treatment also reported larger changes in negative affect following positive events (both  $\gamma > -.19$ , both  $p < .048$ ). By contrast, indicators of MDD and GAD course, including current episode duration, age of onset, recurrence, and lifetime persistence, were not related to positive event responding (all  $\gamma < .35$ , all  $p > .087$ ).

### Discussion

The present study used EMA to examine reactions to positive events in the daily lives of individuals with MDD, GAD, comorbid MDD-GAD, and no psychopathology. Our results indicate that the “mood brightening” effect observed in previous studies is better understood as a broader brightening effect that is evident across multiple domains of functioning. Depressed individuals exhibited larger changes in affect, cognition, reported withdrawal (but not approach) behavior, and symptoms than controls at the time of the positive event and at the signal following the event. In a particularly powerful demonstration of the brightening effect, we found evidence for a dose-response relationship between indicators of depression severity and the magnitude of the brightening effect. Although reactivity to positive events did not consistently distinguish depressed individuals from those with GAD, MDD severity was a stronger predictor of brightening than GAD severity. These findings shed light on the breadth of the brightening effect and provide the first evidence for its relative specificity to depression vis-à-vis anxiety.

#### FIRST AIM: SCOPE OF THE BRIGHTENING EFFECT

Our results extend the findings of three previous EMA studies that observed greater emotional reactivity to daily positive events in individuals with MDD compared to controls (Bylsma et al., 2011; Peeters et al., 2003; Thompson et al., 2012). The consistency of these results across samples, measures, and research groups underlines the need to explain the brightening effect and its divergence from laboratory findings of diminished reactivity to

positive stimuli. One possibility is that brightening represents a measurement artifact rather than a substantive difference between groups. For example, greater variability in negative outcomes like symptom severity allows more room for change in those outcomes among depressed than nondepressed individuals. However, our finding that brightening extends to positive affect, a variable without range restriction in any group, indicates that range restriction does not fully account for brightening. Our finding that brightening is greater for individuals with more severe MDD also implies that this effect is not due solely to range restriction in controls. Similar conclusions are suggested by past evidence for brightening in individuals with subclinical MDD, including those with minor depression (Bylsma et al., 2011) and elevated depression symptoms (Nezlek & Gable, 2001; Takano et al., 2013), whose outcome distributions differ less markedly from controls. Although other measurement artifacts could account for brightening, several of these, including group differences in baseline levels of outcome variables (Bylsma et al., 2011; Thompson et al., 2012; the present study) or in thresholds for rating events as positive (Bylsma et al., 2011), have been tested and ruled out in previous studies.

Taken together, the available data suggest that while measurement factors may contribute to the brightening effect, substantive explanations should also be considered. For example, brightening may result from depressed individuals' more negative expectations for future events compared to controls (Strunk, Lopez, & DeRubeis, 2006). As research on positive contrast effects has shown, low expectations amplify reactions to positive stimuli (McNamara, Fawcett, & Houston, 2013). Our results align with affective contrast theories as well, which posit that an emotional experience (e.g., a pleasant reaction to a positive event) is heightened when it is preceded by, and therefore experienced in direct contrast to, an opposite emotional experience (e.g., depressed mood; Newman & Llera, 2011). Alternatively, features of typical laboratory studies, like the use of standardized stimuli that may be less personally relevant and emotionally salient than stimuli in EMA studies, could dampen brightening. Finally, recent research has distinguished between the anticipation of a future event and the response to an event that has already taken place, and has suggested that MDD is more strongly associated with decreased anticipation of future positive events (Shankman et al., 2014). As the present study examined reactions to positive events that had already taken place, measuring anticipation of future positive events may have yielded different results.

Further research is needed to adjudicate among the possible artifactual and substantive explanations for brightening. First, future EMA studies should address restricted ranges in outcome variables for nonclinical controls by expanding response scales to increase their sensitivity or including lower-difficulty items more readily endorsed by nondepressed individuals following positive events (e.g., down instead of sad). EMA studies could also test a wider variety of positive (e.g., positive thoughts) or neutral (e.g., continuing with the tasks of the day) outcomes that are less likely to have floor effects. Second, there is a need to include more personally relevant stimuli in laboratory studies to increase external validity, like interpersonally significant positive events (e.g., Forbes & Dahl, 2012). Third, mechanism-focused EMA studies could elucidate the sequence of internal and external experiences that lead to mood brightening. Expanding assessments to include event-specific thoughts (e.g., event expectations) and behaviors (e.g., sharing the positive experience with others) would be especially informative.

Finally, research is needed to explain the discordance between depressed individuals' retrospective reports of diminished pleasure on clinical assessments and their reports of heightened reactivity to positive events in momentary assessments, especially given the reliance on retrospective reports in diagnosis and treatment planning. This research would also help reconcile depressed individuals' heightened reactivity to positive daily events with the persistence of MDD. One possibility is that in experiencing fewer positive events than controls, depressed individuals profit less from these events both directly (through improvements in mood and other depression symptoms) and indirectly (through declines in maladaptive cognitions and behaviors that may later reduce symptoms). A second possibility is that the benefits of positive events are overshadowed by the impact of stressful events, which previous analyses in this dataset found to be more frequent—and followed by more adverse consequences—in depressed than nondepressed individuals (Ruscio et al., 2015). A third possibility is that reactions to positive events, although larger in peak amplitude, may be of shorter duration in depressed than nondepressed individuals due to diminished anticipatory pleasure before the event (McFarland & Klein, 2009) or a more rapid return to baseline after the event (Moses-Kolko et al., 2011). Other processes may also play a role, such as depressed individuals' tendency to recall negative material more easily than positive material (Gotlib & Joormann, 2010). Each of these explanations suggests a different path forward for refining interventions aimed at increasing the impact of positive events on individuals with depression.

Importantly, although depressed individuals showed greater change in outcomes following positive events, they continued to show more adverse levels of these outcomes relative to controls, even after adjusting for event positivity. This opens an opportunity to strengthen clinical interventions that involve positive event scheduling like behavioral activation (Hopko et al., 2003) by capitalizing on depressed individuals' responsiveness to those events. If brightening extends to nonnaturally occurring events, it would support the use of EMA in treatment (Heron & Smyth, 2010) to enhance attention to positive events occurring in real-time, and their impact on mood, thoughts, behaviors, and symptoms. Depressed individuals may also benefit from scheduling additional positive events in the immediate aftermath of a positive event to translate initial responsiveness into greater motivation to pursue additional beneficial activities.

#### SECOND AIM: SPECIFICITY OF BRIGHTENING

The current study was the first to test whether the brightening effect extends beyond depression. The preponderance of evidence supported the relative specificity of altered event responding to MDD vis-à-vis GAD. The MDD and comorbid groups, but not the GAD group, reported fewer moderately to highly positive events than the control group. There were few differences between the MDD and comorbid groups, indicating that comorbid GAD had little impact on responsiveness to positive events among depressed individuals. While GAD severity individually predicted larger post-event changes (i.e., brightening) across domains, it did not continue to do so once MDD severity was controlled. There were, however, also indications that the brightening effect is relevant to GAD as well as MDD. Depressed participants showed greater brightening than anxious participants on very few outcomes, and positive events predicted improvement in GAD as well as MDD symptoms. Inspection of mean levels of outcomes following positive events revealed that, for most variables, individuals with GAD reported more adverse outcomes than controls and less adverse outcomes than those with MDD.

Taken together, these findings imply that altered responsiveness to positive events is relatively specific to MDD, but extends in milder form to GAD. These results mirror findings showing that deficits in overall levels of positive emotions are relatively specific to depression when compared with anxiety (Watson & Naragon-Gainey, 2010) at the cross-sectional level (Khazanov & Ruscio, 2016). This similarity between the present brightening findings and previous research on positive

emotions suggests that brightening may represent an important aspect of positive emotional processing in depression. Additionally, brightening could potentially be used to distinguish MDD even from GAD, the disorder most closely related to it. Interventions targeting these processes may be especially beneficial for treating depression, although they may improve symptoms of anxiety as well. Importantly, future research should consider anxiety when examining positive emotional processes, which have been investigated primarily in relation to depression (*Workshop Proceedings, 2011*).

#### STUDY LIMITATIONS

This study had several limitations. First, many, though not all, outcome variables exhibited a restricted range in the control group, which limited the amount of change that could be observed in that group. Second, although our design greatly reduced the possibility of retrospective recall bias relative to traditional longitudinal designs, we did not eliminate this bias, as events and event reactions were rated from memory up to 180 minutes after they occurred. We chose this design, instead of one in which participants initiated event reports, out of concern that asking participants to attend to and record events as they occurred would alter event reactions (*Stone & Shiffman, 2002*) and that differing levels of sensitivity to positive events would lead to differences in event reporting. Reporting biases are a particular concern when participants are asked to initiate reports about events that cannot be defined concretely (*Reis & Gable, 2000*), such as “significant” events for which the threshold for reporting may differ by depression status.

Third,  $T_E$  and  $T_1$  ratings were made at the same assessment occasion, so event-related variables did not temporally precede the variables reflecting participants’ state at the signal. To better differentiate the two sets of ratings, we asked participants to rate their current experiences before rating the event that occurred earlier, separated  $T_1$  and  $T_E$  ratings with a series of questions about the event, and performed analyses with  $T_2$  outcomes. Although the findings for  $T_E$  and  $T_1$  outcomes were similar, they were not identical, and the two sets of outcomes were differentiable, sharing 40%–49% of their variance. That events and  $T_2$  ratings were separated by up to 4.5 hours likely diminished our ability to identify proximate sequelae of positive events, although a number of associations survived this delay. This sampling schedule provided good coverage of the day and allowed enough time between signals for significant events to occur, but

was not frequent enough to characterize the time course of responding to positive events. Fourth, participants indicated spending an average of 4–5 minutes responding to each survey. The survey length may have led to participant fatigue, although there were no indications of this in the data.

Fifth, as in most EMA studies, we relied on participants’ own ratings of event positivity. Although prior research has shown that depressed participants’ subjective judgments of event pleasantness correspond closely to those of nondepressed participants according to the judgments of blind raters (*Bylsma et al., 2011*), it remains possible that the events reported by depressed and nondepressed participants differed in systematic ways that contributed to the differential reactivity of these groups. Sixth, reported behaviors were measured with only one item, with some focusing more on behavior initiation (e.g., “distancing or isolating myself from others”) and some more on behavior maintenance (e.g., “keeping active and busy”). Seventh, due to the number of analyses, we focused on reactivity to positive events. Past studies have found that heightened affective responses do not extend to negative events (*Bylsma et al., 2011; Peeters et al., 2003; Thompson et al., 2012*), but future research should examine whether the other types of responses identified in the present study are similarly limited to positive events. Finally, the limited differences observed between pure and comorbid MDD may have been due, in part, to lower statistical power for these comparisons relative to analyses that combined all depressed participants into a single group for comparison with GAD or control participants.

Importantly, our results are consistent with previous EMA studies examining responses to positive events in depressed individuals, which each had different strengths and limitations. While these studies found heightened affective reactivity to positive events among depressed individuals relative to controls, we demonstrated that this brightening effect extends to cognitive, behavioral, and symptomatic outcomes and that it increases along with depression severity. We also showed that heightened reactivity to positive events is relatively specific to MDD compared with GAD, but is relevant to both disorders. With the brightening effect replicated and its boundaries more clearly defined, future studies can focus on investigating the causes of this effect and its theoretical and clinical implications.

#### Conflict of Interest Statement

The authors declare no conflict of interest.

## Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.beth.2018.05.008>.

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RECEIVED: January 7, 2018

ACCEPTED: May 29, 2018

AVAILABLE ONLINE: 5 June 2018