

Sadness and Amusement Reactivity Differentially Predict Concurrent and Prospective Functioning in Major Depressive Disorder

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Depressed individuals often fail to react to emotionally significant stimuli. The significance of this pattern of emotional dysregulation in depression is poorly understood. In the present study, depressed and nondepressed participants viewed standardized neutral, sad, fear, and amusing films; and experiential, behavioral, and physiological responses to each film were assessed. Compared with nondepressed controls, depressed participants reported sadness and amusement in a flattened, context-insensitive manner. Those depressed participants who reported the least reactivity to the sad film exhibited the greatest concurrent impairment. Prospectively, the depressed participant who exhibited the least behavioral and heart rate reactivity to the amusing film were the least likely to recover from depression. Loss of the context-appropriate modulation of emotion in depression may reflect a core feature of emotion dysregulation in this disorder.

Depression is increasingly being conceptualized as a disorder of emotion (e.g., Barlow, 1988; Gross & Muñoz, 1995). Indeed, the core symptoms of major depressive disorder (MDD)—persistent sad mood and a loss of interest or pleasure in daily activities (*Diagnostic and Statistical Manual of Mental Disorders [DSM-IV]* [4th ed.; American Psychiatric Association, 1994])—strongly implicate emotional dysfunction in depression. Recently, clinical scientists have begun to focus on gaining a more precise understanding of the ways in which emotion is disturbed, or dysregulated, in MDD (see Kring & Bachorowski, 1999, for a review).

Depression and Abnormalities in Responding to Positive Emotional Stimuli

Because one of the cardinal symptoms of depression is *anhedonia* (an inability to experience pleasure), a number of investigators have examined the reactivity of depressed persons to positive stimuli. In fact, in a number of studies depressed individuals have been found to be relatively unresponsive to experimentally presented positive stimuli. For example, compared with nondepressed controls, depressed individuals have been found to exhibit less positive expressive behavior in response to pleasant film and pleasant drink stimuli (Berenbaum & Oltmanns, 1992) and to be less behaviorally responsive to reward contingencies (Henriques & Davidson, 2000). Depressed individuals have also been shown to have an attenuated experience of positive emotion relative to nondepressed persons, evident in reports of reduced pleasure in response to slides depicting pleasant scenes (Allen, Trinder, & Brennen, 1999; Sloan, Strauss, Quirk, & Sajatovic, 1997). Based in part on this evidence, researchers have argued that deficits in response to positive, approach-related emotion cues are characteristic of depressed individuals (e.g., Henriques & Davidson, 1991) and, further, that these deficits may distinguish depression from anxiety and other forms of psychopathology (Clark & Watson, 1991; Clark, Watson, & Mineka, 1994).

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Depression and Abnormalities in Responding to Negative Emotional Stimuli

Although it may appear paradoxical given both cognitive formulations of depression (e.g., Beck, Rush, Shaw, & Emery, 1979) and the proneness of depressed persons to experience and express high levels of negative affect (Watson, Clark, & Carey, 1988), in some contexts depressed individuals have been shown to exhibit relatively low reactivity to negative stimuli. For instance, compared with nondepressed controls, depressed individuals have reported less reaction to painful stimulation in early studies using heat (Hall & Stride, 1954; Hemphill, Hall, & Crookes, 1952), pressure (Merskey, 1965), and electric shock (Davis, Buchsbaum, & Bunney, 1979; von Knorring & Espvall, 1974). These findings have been replicated in more recent studies using pressure and cold stimuli (Lautenbacher, Sernal, Schreiber, & Krieg, 1999), and heat stimuli (Dworkin, Clark, & Lipsitz, 1995; Lautenbacher et al., 1994; but see also Adler & Gattaz, 1993).

Other findings indicate that depressed persons exhibit stereotyped and inflexible responses to a variety of emotional stimuli, suggesting that nonreactivity to negative stimuli in depression stems from a broader pattern of affective flattening. For example, compared with nondepressed controls, depressed persons have been found to show less affective modulation of startle (Allen et al., 1999), less electromyographic (EMG) modulation during affective imagery (Gehricke & Shapiro, 2000; Greden, Genero, Price, Feinberg, & Levine, 1986), less facial reactivity in response to expressive facial stimuli (Wexler, Levenson, Warrenburg, & Price, 1993), less valence-related modulation of event-related brain potentials (Deldin, Keller, Gergen, & Miller, 2001), and a lack of autonomic responding to a variety of stimuli (Dawson, Schell, & Catania, 1977; but also see Lewinsohn, Lobitz, & Wilson, 1973). Indeed, the results of naturalistic studies also indicate that depressed individuals exhibit emotional stereotypy, showing little modulation of their facial affect (e.g., Andreasen, 1979; Kuhlara & Chadda, 1987) or vocal characteristics (e.g., Hargreaves, Starkweather, & Blacker, 1965).

The Nature and Significance of Emotion Response Deficits in Depression

Emotions have often been conceptualized as coordinated systems (e.g., Ekman, 1992) that function to prepare an organism for adaptive response to meaningful stimuli and challenges in the environment

(Keltner & Gross, 1999; Tooby & Cosmides, 1990). The absence of appropriate emotional responding—and in particular, a reduced response to positive emotion cues—plays an important role in theoretical accounts of emotion dysregulation in depression (Clark & Watson, 1991; Clark et al., 1994; Depue & Iacono, 1989; Depue, Krauss, & Spont, 1987; Fowles, 1994; Henriques, Glowacki, & Davidson, 1994). Despite the theoretical importance of emotion response deficits in depression, however, the nature and clinical significance of these deficits continue to be poorly understood.

Emotions are commonly viewed as multicomponential responses that can be indexed by language, behavior, or physiological responding (e.g., Lang, 1978). Interestingly, deficits in the emotional responses of depressed persons have not been observed consistently across all components of responding. For example, some investigators have found that although depressed individuals display less expressive behavior than do nondepressed persons, they report normal levels of emotional experience (e.g., Berenbaum & Oltmanns, 1992; Gehricke & Shapiro, 2000). In contrast, other researchers have found that depressed individuals report less emotional experience than do normal controls, but exhibit comparable levels of expressive behavior (Sloan et al., 1997). One explanation for these discrepant findings is that depression is characterized by low coherence or agreement across different domains of emotional functioning (e.g., Brown, Schwartz, & Sweeney, 1978).

Another important point of uncertainty concerns the clinical significance of emotional unresponsiveness in depression. Indeed, few investigators have examined the relation of emotion response deficits to specific aspects of depressive illness, such as severity of symptoms, impairment in functioning, or prediction of illness outcomes. Clark, Fawcett, Salazar-Gruoso, and Fawcett (1984) found that anhedonic depressives, as assessed by self-report, were more likely to remit at 7-month follow-up than were nonanhedonic depressives. Moos and Cronkite (1999), however, reported the opposite pattern of results: The presence of self-reported anhedonia increased the risk that depression would run a more chronic course over a 10-year period. Finally, Bonnano and Keltner (1997) found that the occurrence of positive-emotion expressive behavior during an interview predicted good subsequent adjustment among recently bereaved individuals, a group that is typically characterized by elevated levels of depressive symptoms. Though re-

sponses to emotional stimuli were not assessed systematically in these studies, the findings point to the merit of exploring emotional reactivity as a potentially important factor in explaining clinical and psychosocial outcomes in depression.

The Present Study

In the present study, we used a multimethod-standardized laboratory procedure to examine emotional reactivity among clinically depressed participants and nondepressed, nonpsychiatric controls. Participants' experiential, behavioral, and autonomic responses to previously validated sad, fear, amusing, and neutral film clips were assessed. In addition to sad stimuli, fear and amusement films were included to examine the specificity of regulatory difficulties in depression. A neutral film was included to allow us to compare the degree of reactivity, or change in response, that participants exhibited to each target emotion film from a neutral baseline. Moreover, to assess the implications of a lack of differential emotional reactivity for concurrent functioning in depression, we examined the relation between emotional reactivity deficits and depression severity, depression-index episode length, and global psychosocial functioning. Finally, to examine the relation of emotional reactivity to clinical outcome in depression, we conducted structured interviews with all depressed participants 6 months after their initial assessment (Time 2) and determined whether each had recovered from his or her depressive episode.

In this study, we examined the following three main predictions:

Hypothesis 1. At Time 1, depressed persons would exhibit less reactivity to emotion film stimuli than normal controls, as indexed by smaller changes on measures of experiential, behavioral, and autonomic functioning.

Hypothesis 2. Building on the formulation that a flattened profile of emotional responding reflects a core aspect of emotional dysregulation in depression, we predicted that those variables on which depressed persons are found to exhibit less emotional reactivity to films would be associated with poorer concurrent functioning at Time 1.

Hypothesis 3. On the basis of the premise that the capacity to emit contextually appropriate emotional responses has adaptive significance, we predicted that the depressed individuals who exhibited the least reactivity to experimentally presented emotional stimuli at Time 1 would be those who are the least likely to recover from depression 6 months later at Time 2.

Method

Participants

Participants were 72 unipolar depressed persons (73% female) and 33 nondepressed controls (70% female) who were fluent in English and were between 18 and 60 years of age. Approximately half the depressed participants were recruited from two outpatient psychiatry clinics in a university teaching hospital, and half were self-referred from the community. Clinical participants had no reported lifetime history of brain injury or primary psychotic ideation, no current diagnoses of panic disorder or social phobia, and showed no behavioral indications of impaired mental status or mental retardation. Clinical participants were also excluded from the sample if they were alcohol or substance dependent or if they showed signs of substance or alcohol abuse within the past 6 months. Thirty-one of the depressed participants were receiving pharmacotherapy.

The nondepressed, nonpsychiatric controls were recruited from the community through advertisements posted in numerous locations (e.g., Internet bulletin boards, university kiosks, supermarkets). Potential control participants were excluded from the study based on the same general and medical criteria that were used for the clinical participants. In addition, they were interviewed with the Structured Clinical Interview for *DSM-IV* Axis I (SCID-I; First, Gibbons, Spitzer, & Williams, 1995) to exclude those with the presence of lifetime diagnoses of any Axis I disorder. All participants provided written informed consent prior to the experimental session, and were paid \$25 per hour for their participation in the study.

Clinical Assessments

All depressed participants met *DSM-IV* criteria (American Psychiatric Association, 1994) for MDD using the SCID-I. SCID-I interviewers had previous experience with administering structured clinical interviews and were trained specifically to administer the SCID-I interview prior to beginning work on this study. An independent, trained rater who was unaware of group membership evaluated 15 randomly selected audiotapes of SCID interviews with depressed and nondepressed participants, and with nonparticipants who met diagnostic criteria for disorders other than depression (e.g., panic disorder), and for each determined whether the participant met *DSM-IV* diagnostic criteria for MDD. In all 15 cases, ratings matched the diagnosis made by the original interviewer ($\kappa = 1.00$).

Six months following participants' entry to the study (i.e., Time 2), they were administered a modified version of the SCID-I. We used guidelines recommended by the National Institute of Mental Health Collaborative Program on the Psychobiology of Depression (e.g., Winokur, Coryell, Keller, Endicott, & Akiskal, 1993) to define recovery from depression. Depressed participants were considered to be recovered if they reported at Time 2 that essentially no signs of depressive illness were present during each of the past 8 weeks (e.g., no more than two symptoms experienced to more than a mild degree). We adopted this stringent definition of recovery because of the significant functional impairment associated with residual depressive symptoms (Judd, Paulus, Wells, & Rapaport, 1996). Therefore, participants who exhibited subsyndromal or syndromal depression were considered to be nonrecovered.

Additional Clinical Measures

Depression severity. At the time of the SCID-I interview, participants completed the Hamilton Depression Inventory (HDI; Kobak & Reynolds, 2000; Reynolds & Kobak, 1995). The HDI is a 23-item self-report version of the Hamilton Rating Scale for Depression (Hamilton, 1960) that has been shown to correlate highly with the clinical interview and has demonstrated high reliability and validity (Kobak & Reynolds, 2000; Reynolds & Kobak, 1995). The internal consistency of the HDI was .94 for the present sample.

Length of current depressive episode. The SCID-I interviewer determined the onset of the current depressive episode for all individuals diagnosed with current depression.

Global functioning. The Global Assessment of Functioning Scale (GAF; Axis V, *DSM-IV*; American Psychiatric Association, 1994) was used to assess global functioning. This is a single rating scale used to evaluate an individual's overall level of psychological, social and occupational functioning. Ratings are made based on information obtained during the SCID-I interview. Values on the scale range from 1 (*lowest level of functioning*) to 100 (*highest level of functioning*), and are divided into ten 10-point intervals. Each interval is anchored with detailed, behaviorally oriented descriptors of functioning. Validation studies conducted with both inpatients and outpatients have indicated that the *DSM-IV* GAF and its predecessors correlate highly with other previously validated measures of overall severity of illness and changes in severity (e.g., Mental Status Examination

Record; Endicott, Spitzer, & Fleiss, 1975), as well as with therapists' and relatives' ratings of patient functioning (Endicott, Spitzer, Fleiss, & Cohen, 1976). The GAF has also been found to have good interrater reliability (Endicott et al., 1976). For reliability purposes in the present study, an independent, SCID-I trained rater who was unaware of group membership listened to the audiotaped SCID interviews of 12 randomly selected participants and made GAF ratings. Interrater reliability for the GAF was high ($r = .89$).

Stimuli

Films are dynamic visual stimuli that have been shown to elicit emotions ethically and reliably with relatively low demand characteristics. Film selection was based on criteria recommended by Gross and Levenson (1995). The neutral film lasted 180 s and depicted coastal landscape scenery. The fear film was 140 s and depicted heavy turbulence in the cabin of a commercial airline. The sad film was 170 s and depicted a boy who was distraught at the death of his father. The amusing film lasted 120 s and depicted antic, slapstick-type comedy.

Equipment

An SA Instruments 12-channel bioamplifier was used to record physiological responses. Signals were sampled at 400 Hz. Data were acquired using a Pentium PC that used a Data Translation 3001 PCI 12-bit 16-channel analog to digital converter. Data were reduced offline with custom laboratory software. During the psychophysiology assessment, film stimuli were presented on a 20 in. (51 cm) television monitor at a viewing distance of 1.75 m. The participant room was equipped with a remotely controlled video camera that unobtrusively recorded participants' facial behaviors. Recording took place in low ambient light.

Measures

Self-report assessment of emotion. At the start of the experiment and after each film, participants rated their levels of sadness, fear, and amusement on 9-point scales ranging from 0 (*not at all*) to 8 (*extremely*). Other terms (e.g., embarrassment) were included to reduce demand characteristics.

Behavior. A remotely controlled camera positioned behind darkened glass unobtrusively made a videotape recording of participants' facial expressive behaviors. To assess levels of sadness, fear, and amusement reactivity, expressive behavior was coded with the Emotional Behavior Coding System (Gross & Levenson, 1993). This system requires coders to

view and rate videotapes in real time. Six undergraduate research assistants read the training manual for this coding system and participated in 10 weeks of training prior to beginning work on this project. This training required coders to learn full prototypes of each behavior (Ekman & Friesen, 1975) as well as to recognize a number of specific molecular behaviors indicative of the prototype. Sadness, amusement, and fear were rated for each film period on 7-point scales, with values representing an aggregate of intensity, duration, and frequency of response (e.g., on the 0–6 scale, a 1 for sadness was to be coded when participant exhibited for less than 5 s a slight downturn of the mouth, or a slight upturning of the inner eyebrow with closed body posture and the head moved forward). Videotapes were coded by at least two raters who were unaware of the diagnostic status of participants, the nature of the experimental manipulation, and the study hypotheses. The average interrater agreement was acceptable for sadness and amusement: $\alpha = .66$ for sadness ratings to the sad film; $\alpha = .85$, and for amusement ratings to the amusing film. Fear-behavior reliability was low, likely because of the low base rates of fear behavior elicited by the films ($\alpha = .44$) for fear ratings to the fear film.

Physiology. The cardiovascular and electrodermal systems are known to be important organ systems involved in emotional responding. From these two systems, we sampled heart rate and skin conductance response rate. Previous work has indicated that depressed individuals exhibit blunted responding on these measures in emotional contexts (e.g., Albus, Mueller-Spahn, Ackenheil, & Engel, 1987; Dawson et al., 1977). A measure of somatic activity was also included both to afford an index of gross motor activity and to provide a means to control for the effects of motor activity on the other physiological measures:

Heart rate. Beckman miniature electrodes were placed in a bipolar configuration on opposite sides of the participant's chest. The interbeat interval was calculated as the interval (in ms) between successive R waves in the electrocardiogram (ECG) and converted to instantaneous heart rate.

Skin conductance response rate. A constant-voltage device passed a small voltage between Beckman regular electrodes attached to the palmar surface of the proximal phalanges of the first and second fingers of the nondominant hand. Skin conductance fluctuations were detected as changes in skin conductance level from a zero-slope baseline exceeding $0.2 \mu\text{S}$.

Somatic activity. A piezoelectric sensor attached to the leg of the participant's chair provided a sensitive index of overall body movement.

Physiological data were reduced offline. Data reduction software interfaced with recorded binary data files to extract segments of raw data and then performed waveform transformation, feature detection, and graphic display for each channel. Finally, all segments were examined for artifacts and edited. Period averages were calculated for each film epoch.

Procedure

Participants were greeted and then positioned in a comfortable chair facing a video monitor in a quiet, well-furnished laboratory room. Following an orientation period and the attachment of physiological sensors, participants completed an emotion questionnaire and viewed the neutral film. The two negative films were then shown in counterbalanced order, separated by a short arithmetic task to minimize carryover. The amusing film was shown last. All films were preceded by instructions to watch each film carefully and followed by an emotion questionnaire. Finally, participants were disconnected from monitoring devices, paid, and reminded that they would be contacted for follow-up interviews in 6 months.

Results

As is evident from Table 1, the nondepressed, non-psychiatric participants were similar to the depressed participants with respect to gender composition, age, and level of education (all $ps > .1$). As expected, the depressed participants obtained higher scores on the HDI and lower scores on the GAF than did their non-depressed counterparts (both $ps < .001$).

Table 1
Demographic and Clinical Characteristics of the Sample

Variable	Group	
	Depressed ($n = 72$)	Nondepressed ($n = 33$)
% female	66.7	69.7
<i>M</i> (and <i>SD</i>) for:		
Age	33.4 (10.5)	32.3 (11.7)
Education level	6.6 ^a (1.5)	6.5 ^a (1.4)
GAF score	53.7 (8.4)	86.4 (4.1)*
HDI score	28.1 (7.0)	4.4 (3.7)*

Note. GAF = Global Assessment of Functioning; HDI = Hamilton Depression Inventory.

^a Education was assessed on an 8-point scale, with higher numbers representing more education—a score of 6.6 reflects some college education.

* $p < .001$.

Depression-Associated Differences in Emotional Reactivity

Hypothesis 1 predicted that the depressed individuals would show blunted emotional reactivity relative to the nondepressed controls. To test this hypothesis, we conducted a repeated measures multivariate analysis of variance (MANOVA) for each domain of emotional response (experience, expressive behavior, and autonomic functioning). The primary between-subjects variable was depression (depressed, nondepressed), and the within-subjects variables were film condition (neutral, sad, fear, amusing) and emotion (sadness, fear, and amusement).¹ More specifically, to examine experiential reactivity, a repeated measures MANOVA was conducted on reported sadness, fear, and amusement. To examine behavioral reactivity, a similar repeated measures MANOVA was conducted on observer-rated sadness, fear, and amusement behavior. Finally, to examine physiological reactivity, a repeated measures MANOVA was conducted on the three physiological measures. In analyses where Greenhouse–Geisser correction was appropriate (e.g., repeated measures analysis of variance [ANOVA]), we report adjusted p values.

Emotion experience. The MANOVA conducted on emotion experience yielded significant main effects for film condition, $F(3, 99) = 23.89, p < .001$, and emotion, $F(2, 100) = 12.95, p < .01$, as well as a significant interaction of film condition and emotion, $F(3, 99) = 46.64, p < .001$. These effects were qualified, however, by a significant higher order interaction of film condition, emotion, and depression status, $F(6, 99) = 2.24, p < .05$. To identify the source of this interaction, we conducted two-way (Film Condition \times Depression Status) repeated measures ANOVAs separately for each emotion. The two-way interactions were significant for sadness, $F(3, 100) = 3.44, p < .05$, and amusement, $F(3, 100) = 5.50, p < .01$, but not for fear, $F(3, 100) = 1.57, p > .1$. Mean self-reports of sadness and amusement for each film condition and diagnostic group are indicated in Table 2.

For sadness, follow-up one-way ANOVAs indicated that the depressed participants reported more sadness than did the normal controls during the neutral film, $F(1, 103) = 25.11, p < .001$, and the amusing film, $F(1, 103) = 8.25, p < .01$, but not during the sad film, $F(1, 103) = 1.19, p > .1$, or the fear film, $F(1, 103) = 2.05, p > .1$. In other words, depressed persons reported elevated levels of sadness in contexts in which the report of sadness would ordinarily be low

Table 2
Means (and Standard Deviations) of Participant Self-Reports of Sadness and Amusement by Film Condition and Diagnostic Group

Condition and group	Sadness	Amusement
Neutral film		
Depressed	2.34 (2.28)*	1.46 (1.56)
Nondepressed	0.25 (0.62)	1.81 (1.65)
Sad film		
Depressed	4.37 (2.71)	1.00 (1.56)
Nondepressed	3.69 (2.49)	0.53 (1.05)
Fear film		
Depressed	3.00 (2.53)	1.62 (1.98)
Nondepressed	2.28 (1.90)	0.97 (1.51)
Amusing film		
Depressed	0.80 (1.54)*	3.41 (2.38)*
Nondepressed	0.03 (0.18)	4.53 (2.33)

* $p < .05$.

(i.e., to the neutral and amusing films), but not in negative emotional contexts. Change scores, created by subtracting participants' reports of sadness to the sad film from their responses to the neutral film, confirmed that the depressed participants ($M = 2.03, SD = 2.68$) exhibited a smaller differential response to the sad film than did the nondepressed participants ($M = 3.45, SD = 2.45$), $t(102) = 3.79, p < .001$. For amusement, follow-up one-way ANOVAs revealed that the depressed participants reported less amusement than did the controls to the amusing film stimulus, $F(1, 100) = 4.91, p < .05$, but did not differ in reports of amusement for any of the other films (all $ps > .1$). Change-score analyses for amusement produced conceptually similar, but nonsignificant, results (depressed: $M = 2.00, SD = 2.55$; nondepressed: $M = 2.72, SD = 2.20$), $t(103) = 1.38, p > .05$. For both sadness and amusement, therefore, consistent with

¹ Preliminary analyses included film order, psychotropic medication status, and gender as between-subjects factors. Analyses conducted within the depressed group indicated that medicated patients did not differ from unmedicated patients in their experiential, behavioral, or autonomic reactivity (all $ps > .1$). Preliminary analyses of each of the three domains of emotional functioning using the entire sample indicated that there were no significant effects for film order (all $ps > .1$). Parallel analyses using gender as a factor indicated that gender did not interact with depression status for any of the three domains of emotional functioning (all $ps > .1$). Consequently, we eliminated these variables from subsequent analyses to increase statistical power.

Hypothesis 1, depressed individuals showed less of a differential response to emotionally significant stimuli than did normal controls.

Behavior. The repeated measures MANOVA conducted on the behavioral variables yielded no significant main effects or interactions involving diagnostic group. Significant main effects were obtained for film condition, $F(3, 99) = 33.41, p < .001$, and emotion, $F(2, 100) = 70.29, p < .001$; both of these effects were qualified, however, by a significant interaction of film condition and emotion, $F(6, 96) = 25.92, p < .001$. Follow-up examination of this interaction indicated that, consistent with the experimental manipulation, participants exhibited more sadness during the sad film than during the other three conditions, exhibited more fear to the fear film than they did to the other three conditions, and exhibited more amusement to the amusing film than they did to other three conditions (all $ps < .05$).

Physiology. The repeated measures MANOVA conducted on heart rate, skin conductance response rate, and somatic activity yielded no significant main effects or interactions (all $ps > .05$).

Relation of Differential Emotional Reactivity to Concurrent Functioning in Depression

Hypothesis 2 predicted that group differences in emotional reactivity between depressed and nondepressed individuals would be related to psychosocial functioning in MDD. To address this question, we first computed sad and amusement reactivity scores by subtracting the participants' response to the neutral film from their response to the target film. We then conducted regression analyses on our three measures of psychosocial functioning (depression severity, depression episode length, and GAF scores) using sadness and amusement reactivity as predictor variables.

Sadness reactivity accounted for significant variance in depression severity ($\beta = -.75, p < .05$), depression episode length ($\beta = 2.05, p < .05$), and GAF scores ($\beta = .89, p < .02$). That is, within the sample of depressed individuals lower sadness reactivity scores were associated with worse psychosocial functioning across the three measures. Because our change-score reactivity metric was a joint function of responses to the sad and the neutral films, however, it was important to ascertain whether one of these two raw film scores predominantly accounted for the variance in observed psychosocial functioning. To examine this question, we conducted regressions in which we first separately entered the neutral and the sad film raw scores to predict each measure of concurrent psy-

chosocial functioning. Neutral film scores predicted depression severity when entered individually ($\beta = 1.59, p < .001$), and when sadness film scores were added to the model ($\beta = 1.75, p < .001$). By contrast, sad film scores did not predict depression severity either when entered separately or together with the neutral film scores (all $ps > .1$). Thus, neutral film scores, in large part, accounted for the observed inverse association between sadness reactivity and depression severity. This was not the case, however, for the regressions performed with the other two measures. In fact, neither sadness film scores nor neutral film scores predicted levels of depression episode length or global functioning (all $ps > .1$). In general, therefore, and underscoring our present emphasis on emotional reactivity, the failure of depressed persons to *modulate* sadness reports across contexts predicted impaired functioning more consistently than did reports of sadness in either context taken separately.

Finally, we wanted to assess whether these results might be explained more simply as a function of participants' tonic levels of sadness experience (i.e., sadness reports taken before any films were shown). Indeed, one could easily surmise that the amount of sadness reported by depressed persons at the outset of the experiment might be inversely related to both subsequent sadness reactivity and to levels of psychosocial functioning, accounting for our obtained results. Although this is plausible, it was not found to be the case. Initial sadness reports among depressed participants were unrelated to sadness reactivity scores ($r = .03, p > .05$). Furthermore, sadness reactivity continued to predict psychosocial functioning even when initial levels of reported sadness were added into the regression analysis (depression severity: $\beta = -.75$; GAF score, $\beta = .88$; depression episode length: $\beta = -2.06$, all $ps < .05$). Thus, also consistent with Hypothesis 2, a lower degree of sadness reactivity among depressed individuals was associated with poorer functioning across measures, even when tonic levels of reported sadness were taken into account.

We performed parallel regression analyses using reported amusement reactivity as a predictor variable. These analyses indicated that self-reported amusement reactivity did not account for significant variance in any of the three measures of concurrent functioning (all $ps > .05$).

Relation of Differential Emotional Reactivity to Prospective Functioning in Depression

SCID interview data at Time 2 were available for 57 of the 72 (79%) depressed individuals. Of the 15

participants who did not participate in the Time 2 assessment, 1 declined participation because of health problems, 5 had moved, 6 were lost to contact, and 3 had been contacted but could not be scheduled for interviews. Attrition analyses indicated that the depressed study completers did not differ from the non-completers at Time 1 with respect to gender composition, treatment status, psychotropic medication use, age, education level, GAF scores, self-reported depression severity, or length of current episode (all $ps > .15$). Of the 57 depressed individuals assessed at Time 2, 11 were completely recovered from depression (19.3%) and 46 were not recovered (80.7%). The characteristics of these two groups are presented in Table 3.

According to Hypothesis 3, flattened emotional reactivity to the films presented at Time 1 would predict nonrecovery from depression at Time 2. To examine this question, we again computed reactivity scores separately for each emotion film and each measure of emotional response. Experiential reactivity was computed by subtracting the participants' reported response on the target emotion in the neutral film from their response on the target to each relevant emotion film (e.g., fear reported to the fear film minus fear reported to the neutral film). Similarly, behavioral reactivity was computed by subtracting the participants' behavior on the target emotion during the neutral film from their behavioral response on the target during the relevant emotion film (e.g., observable sadness during the sadness film minus sadness displayed during the neutral film). Finally, physiological reactivity was calculated by subtracting heart rate and skin conductance responding during the neutral film from responses to each emotion film.

Each of these reactivity measures was entered separately into a multinomial logistic regression using Time 2 diagnostic status as the dependent variable. As can be seen in Table 4, amusement-heart rate reactiv-

Table 3
Characteristics of Depressed Participants at Time 2 by Time 2 Recovery Status

Variable	Recovered ($n = 11$)	Nonrecovered ($n = 46$)
% female	72.7	66.0
<i>M</i> and (<i>SD</i>) for:		
Age	33.7 (11.2)	34.4 (10.9)
Education level	6.3 (1.5)	6.6 (1.4)
HDI score	10.8 (4.5)	24.5 (9.0)*

Note. HDI = Hamilton Depression Inventory.
* $p < .001$.

Table 4
Time 1 Emotional Reactivity Scores as Predictors of 6-Month Illness Outcome in Depression

Reactivity score	χ^2	p
Sadness experience	0.13	.72
Sadness behavior	0.00	.99
Sadness skin conductance responses	0.02	.89
Sadness heart rate responses	0.34	.56
Fear experience	0.96	.33
Fear behavior	0.75	.39
Fear skin conductance responses	0.87	.35
Fear heart rate responses	1.43	.23
Amusement experience	1.16	.28
Amusement behavior	5.58	.02*
Amusement skin conductance responses	2.07	.15
Amusement heart rate responses	7.51	.006*

Note. For all χ^2 values, all degrees of freedom equal 1.
* $p < .05$.

ity and amusement behavioral reactivity to the amusement film at Time 1 predicted diagnostic status at Time 2. Specifically, depressed individuals who subsequently recovered showed larger amusement-heart rate reactivity ($M = 1.72$, $SD = 5.07$) than did depressed persons who did not recover ($M = -1.89$, $SD = 3.35$), $F(1, 55) = 8.36$, $p < .01$. Similarly, at Time 1 depressed participants who subsequently recovered displayed greater amusement behavioral reactivity ($M = 2.36$, $SD = 2.37$) than did depressed persons who did not recover ($M = 0.98$, $SD = 1.30$), $F(1, 55) = 7.13$, $p = .01$. Thus, consistent with Hypothesis 3, nonrecovery from depression at Time 2 was associated with lesser behavioral and physiological reactivity to the amusing film at Time 1.

When these two variables were entered simultaneously into a multinomial logistic regression, amusement-heart rate reactivity remained a significant predictor of Time 2 status, $\chi^2(1, N = 57) = 5.08$, $p < .05$, whereas amusement behavioral reactivity became marginally significant, $\chi^2(1, N = 57) = 3.42$, $p < .07$, suggesting that heart rate reactivity was a more robust predictor of Time 2 status than was the level of amusement behavioral reactivity. Indeed, heart rate reactivity to the amusing film continued to significantly predict Time 2 status even when Time 1 depression severity, depression episode length, and GAF scores were entered into regression analyses, $\chi^2(1, N = 50) = 7.99$, $p < .01$.

Discussion

Although there is increasing agreement that depression is a disorder of emotion, much remains to be

learned about precisely how emotion is dysregulated in depression. Because the failure to respond appropriately to emotionally significant stimuli in the environment has received extensive treatment by previous theorists as a potentially important aspect of emotion dysregulation in depression, we designed a comprehensive study that included both a cross-sectional laboratory assessment of emotional reactivity (including both between- and within-subjects analyses) and a prospective examination of recovery. We believed that this combined approach would strengthen our conclusions about the nature and significance of emotional reactivity in depression.

Consistent with our predictions that depressed individuals would demonstrate deficits in responding to emotional stimuli relative to nondepressed control participants, depressed persons were found to be characterized by aberrations in their reporting of sadness and amusement. More specifically, whereas depressed participants reported more sadness to a neutral, or innocuous, stimulus than did normal controls, they also reported a smaller increase in sadness than did controls in response to an objectively sad stimulus. Similarly, depressed participants also tended to report a smaller increase in amusement than did nondepressed controls when an amusing film was presented. Although this finding of lesser self-reported reactivity to positive stimuli among depressed persons is consistent with a number of previous findings (e.g., Allen et al., 1999; Sloan et al., 1997), reactivity to evocative sad stimuli has seldom been assessed in depression. That depressed individuals exhibited less reactivity than did nondepressed controls in both sad and amusing contexts lends support to the view that the self-reports of both negative and positive emotions by depressed persons are not sensitive to context—a view of depression, interestingly, that has also been formulated in recent neuropsychological theorizing (e.g., hippocampal deficits; Davidson, Jackson, & Kalin, 2000).

In contrast to their self-reports, however, the behavioral and physiological responding of depressed persons to emotion films was comparable to that of nondepressed controls. Although this mixed result tempers our conclusions about the degree to which depressed individuals exhibit generalized emotional response deficits, a disjunctive pattern in which results varied by emotional response domain was not altogether unexpected. Disjunctive relations among emotion response components in depression have been observed previously (e.g., Brown et al., 1978). In exploratory analyses of our own data, we found sug-

gestive, though not statistically reliable evidence that responses to sad and amusing films by the depressed participants were on average less coherent than the responses by controls (average correlations between reactivity measures; sadness: depressed = .17; nondepressed = .37; amusement: depressed = .22; nondepressed = .28). Although a detailed treatment of this issue is beyond the scope of this article, we believe that additional work using advanced methods for assessing emotion response coherence (i.e., use of continuous measurement; Rosenberg & Ekman, 1994) and improved theory for interpreting this pattern of response is clearly warranted.

Our prediction that the failure of depressed individuals to exhibit differential emotional reactivity would be related concurrently to difficulties in their psychosocial functioning was also supported. The depressed individuals who exhibited the least discrimination between neutral and sad stimuli in their self-reports were characterized concurrently by the poorest psychosocial functioning. These results, though predicted from our theoretical perspective, are not altogether intuitive. Why should lower levels of emotional reactivity be associated with greater impairment in depression? Our explanation for the association we have observed between nonresponding to emotionally significant environmental stimuli and poorer functioning follows a functionalist approach, which, at base, regards emotions as responses that facilitate swift and efficient adaptation to changing environmental demands (Keltner & Gross, 1999). Indeed, one implication of the data presented here is that the adaptive significance of emotional responses is retained during episodes of psychopathology. More specifically, the present findings suggest that, even during an episode of depression in which affected individuals report problems with persistent sadness, depressed persons who retain a capacity to be further saddened by sad stimuli function better than depressed individuals who do not discriminate in their responses between sad and neutral stimuli.

Our prospective analyses offered further evidence of a relation between emotional nonresponding and worse psychosocial adaptation. Consistent with predictions, emotional reactivity at Time 1 predicted the diagnostic status of depressed individuals at Time 2. More specifically, lower behavioral and physiological reactivity to an amusing film predicted nonrecovery from depression. It is notable that a measure of heart rate reactivity obtained when participants were engaged with a pleasurable stimulus yielded our strongest results. Fowles and others have argued that

heart rate reflects activity in the behavioral approach system and is sensitive to signals of impending reward (anticipatory pleasure) and to actual reward (consummatory pleasure; e.g., Fowles, Fisher, & Tranel, 1982). It is therefore particularly significant that heart rate responses to an amusing film predicted subsequent recovery from depression, whereas heart rate responses to negative emotion films did not. Moreover, it is also noteworthy that heart rate during the amusing film was a better predictor of recovery than were concurrent behavioral or experiential responses to the film.

These findings also speak to the importance of incorporating physiological indicators into the measurement of anhedonia. This construct is typically defined by a lack of experienced pleasure; consequently, investigators rely heavily on patients' self-reports to assess anhedonia (Willner, 1993). The present results suggest, however, that incentive responding in depression is not unitary. As further evidence that experiential, behavioral, and physiological indicators of anhedonia may tap distinct aspects of the phenomenon, it is noteworthy that anhedonic responsivity by depressed participants to an amusing film was evident at Time 1 only in their self-reports, and not in their behavior or psychophysiology; yet, it was anhedonic behavior and heart rate responses at Time 1, and not self-reports, that predicted subsequent recovery from depression. Thus, a multisystem assessment of anhedonia appears to provide information that might be missed through a less comprehensive measurement of emotional functioning.

To our knowledge, this is the first demonstration that emotional reactivity is related to psychosocial impairment or to the course of MDD. Nevertheless, it is important to acknowledge two specific limitations of this study. First, although our sample of depressed individuals at Time 1 was sizable and our rate of attrition was relatively low, the rate of full recovery in this sample was also relatively low and may have limited our power to detect small effects in the prediction of depression recovery. Second, although films are highly standardized and reliable elicitors of emotion, they represent only one type of possible stimulus—external and visual. In replicating these findings, it is critical to devote attention to the effects of other stimulus dimensions on emotional responding in depression (e.g., vicarious vs. actual, internally vs. externally generated).

In conclusion, diagnosed depressed participants in this study showed lower context-appropriate emotional responding than did nondepressed persons.

Less differential response to standardized emotional stimuli in depression was associated with both worse concurrent and worse prospective functioning. In addition, our results suggest that this pattern of emotional deficits is multifaceted. Cross-sectionally, depressed individuals exhibited response deficits in the experiential domain for both sadness and amusement. The correlates of these cross-sectional findings represented an important pattern of emotion specificity: Within the group of depressed participants, less sadness reactivity was associated with greater concurrent psychosocial impairment. Finally, consistent with the importance of considering positive emotion when understanding emotional dysregulation in depression, reactivity to the amusing film was the strongest predictor of prospective functioning in this disorder. Taken together, these results suggest that the inability to mount emotional responses to significant environment stimuli in depression may reflect a core aspect of emotion dysregulation in this disorder. Assessment of multiple emotions and multiple emotion response systems is important for future progress in this area of research.

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