

Crying Threshold and Intensity in Major Depressive Disorder

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Clinical lore suggests that depression is associated with frequent and intense crying. To test these postulations empirically, a standardized cry-evoking stimulus was presented to depressed and nondepressed participants, and their likelihood of crying and the magnitude of crying-related changes in their emotion experience, behavior, and autonomic physiology were compared. Unexpectedly, crying was no more likely in depressed than in nondepressed participants. Within the nondepressed group, participants who cried exhibited increases in the report and display of sadness and had greater cardiac and electrodermal activation than did participants who did not cry. There was less evidence of this crying-related emotional activation within the depressed group. The lack of emotional activation among clinically depressed participants who cried provides a tantalizing clue concerning how emotions are dysregulated in this disorder.

Major depressive disorder (MDD) is commonly referred to as a disorder of emotion (e.g., Barlow, 1988; Gross & Muñoz, 1995). Indeed, the centrality of emotional disturbance is readily apparent from the diagnostic features of MDD. Major depression, as described in the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders (DSM-IV)*; American Psychiatric Association, 1994), is characterized by persistent sad mood or a loss of interest or pleasure in daily activities and by a number of associated symptoms, such as weight loss or gain, loss of appetite, sleep disturbance, psychomotor retardation, fatigue, and feelings of guilt. Given the range of emotional disturbance in depression, it is surprising that scientists have only recently begun to study and specify precisely how emotion is affected in MDD (see Kring & Bachorowski, 1999, for a review).

One window into the emotional functioning of depressed individuals is provided by the crying response. Crying has long been viewed as a universal response involved in the communication of distress (Darwin, 1872), and clinicians have often remarked that depressed persons cry excessively (cf. Beck, Rush, Shaw, & Emery, 1979). The *DSM-IV*, however, does not require changes in either the threshold for crying or the intensity of the crying response for a diagnosis of MDD, and the significance of these oft-repeated clinical observations is unclear. In the following sections, we review the empirical literature on crying in nondepressed and depressed individuals and then describe a study designed to

examine the impact of depression on the likelihood of crying and the intensity of the crying response.

Crying in Nondepressed Individuals

Crying is a secretomotor response defined by the shedding of tears from lacrimal structures. Crying while sad is often accompanied by grimacing and other facial expressions of sadness, by vocalizations, and by the convulsive inhalation and exhalation of air. Like the solicitation signals of other species (Maclean, 1985; Newman, 1985), crying in human infants has been conceptualized as a signal that evolved to increase the proximity between infants and their caregivers (Ainsworth, 1969; Bowlby, 1969; Hunziker & Barr, 1986). Although crying in adults is undoubtedly more complex than crying in infants, like infant crying, adult crying has been interpreted as a signal of distress (Cornelius, 1984, 1988). Indeed, theorists have argued that crying is designed to alleviate distress by motivating the self to action (Tompkins, 1963), by motivating others to engage in prosocial behaviors (Averill, 1968), or by furthering physiological homeostasis (Efran & Spangler, 1979; Frey, DeSota-Johnson, Hoffman, & McCall, 1981).

In keeping with the view that crying is an aversive, behaviorally activated response that signals distress, empirical investigations have demonstrated that crying leads to coordinated changes across systems of emotional responding. For example, individuals who display tears while viewing a sad film report more sadness and pain and exhibit more expressive behavior than do their noncrying counterparts (Gross, Fredrickson, & Levenson, 1994). Adults who cry during a sad film also have significantly greater increases in sympathetic nervous system activity than do adults who simply watch a sad film, as evidenced by increases in measures of electrodermal response (Kraemer & Hastrup, 1988), decreases in peripheral temperature, and accelerations in heart rate beyond the demands of ongoing somatic activity (Gross et al., 1994).

Crying in Depressed Individuals

Textbook descriptions of depressed patients often include increased frequency of crying as a characteristic of the disorder (e.g.,

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Beck, 1967). The self-reports of depressed individuals on questionnaire or interview items (e.g., Beck et al., 1979; Hamilton, 1960) also suggest that crying may be increased in this disorder. This makes good sense: A common conception of crying behavior in adults is that it registers distress. Depressed individuals are well known to report acute negative affect (Watson, Clark, & Carey, 1988) and high levels of personal distress (American Psychiatric Association, 1994). More specifically, the emotional state that most immediately precedes crying among nondepressed individuals is feeling sad (Wallbott & Scherer, 1988). Because sad mood is a defining feature of MDD, it seems reasonable to expect that the depressed state lowers the threshold for crying behavior and magnifies the coordinated changes in emotion that typically occur during crying. Interestingly, recent neurobiological accounts of mood disorders also predict these crying-related changes in depression, describing pathophysiology whereby the same level of eliciting stimulus evokes increasing behavioral and physiological reactivity over time (see Post & Weiss, 1998, for a review of kindling and behavioral sensitization models).

Existing research, however, does not yet provide strong evidence that depression fundamentally alters either the frequency of crying or the magnitude of the crying response. Indeed, previous investigations examining the association between crying and depressed mood in nonclinical samples have yielded largely inconclusive results. For example, whereas Frey et al. (1981) found a positive association between frequency of crying and self-ratings of depression, but only for females, Choti, Marston, Holston, and Hart (1987) reported the opposite pattern of results: Baseline reports of depression were related to reports of crying in response to a film stimulus, but only for males. Similarly, Hammen and Padesky (1977) reported that elevated scores on the Beck Depression Inventory were associated with a reported inability to cry, but only for males. In other studies, frequency of crying was found to be unrelated to self-report measures of depressed mood (e.g., Kraemer & Hastrup, 1986; Labott & Martin, 1987).

It is possible that a more robust association between depression and crying would be found if diagnosably depressed individuals were studied or if behavioral measures of crying were used. Unfortunately, prior studies of crying in clinically depressed samples have been limited to describing demographic and clinical features associated with this behavior. For example, on the basis of ward observations, Davis, Lambert, and Ajans (1969) concluded that crying was more common among neurotic than among psychotic depressives. Although some investigators have found depressed females to be more likely than depressed males to report increased crying (e.g., Carter, Joyce, Mulder, Luty, & McKenzie, 2000; Vredenburg, Krames, & Flett, 1986; Zetin, Sklansky, & Cramer, 1984), there is evidence to suggest that this gender difference may be less reliable in depressed samples than it is among nondepressed controls (e.g., Choti et al., 1987; Davis et al., 1969; Kraemer & Hastrup, 1986; Williams, 1982). Importantly, none of these prior investigations have applied a theoretical framework regarding the known functions of crying to depression or have performed a controlled assessment of crying threshold. It is clear, therefore, that more research is required to understand the impact of depression on both the frequency of crying and the intensity of the crying response.

The Present Study

The present study was designed to examine the crying response in depression. More specifically, we tested three interrelated hypotheses.

Hypothesis 1: Compared with nondepressed controls, depressed participants will be more likely to cry in response to a sad stimulus. To evaluate this hypothesis, we videotaped clinically depressed and nondepressed participants while they viewed an emotionally provocative, cry-eliciting film clip. Having participants view this sad film clip provided an ethical, controlled, and replicable means for us to assess crying threshold in depression. Previous work with comparable stimuli indicated that only about 20% of an unselected sample would be expected to meet our strict behavioral criterion for crying (Gross et al., 1994); consequently, we enrolled a large sample of participants ($N = 104$) into this study. This behavioral criterion of observable tears also provided us with an objective measure of crying, thereby allowing us to circumvent the biases in reporting and memory that characterize depressed individuals (e.g., Gotlib & Neubauer, 2000). We predicted that depressed participants would be more likely to exhibit tears during the sad film than would nondepressed participants. To test this hypothesis, we used the chi-square statistic to compare the proportion of depressed and nondepressed participants who cried in response to the film.

Hypothesis 2: Depression will enhance the crying response. Prior work with nondepressed individuals indicates that crying leads to increases in the report and expression of sadness and in physiological arousal (Gross et al., 1994). We predicted that depression would further enhance this crying response. To evaluate this hypothesis, we first divided each diagnostic group into criers and noncriers (on the basis of the presence or absence of observable tears). Then, within each diagnostic group, we compared criers and noncriers on measures that indexed the intensity of emotional responding under neutral and sad conditions. We predicted that differences between criers and noncriers in their reports and displays of sadness and in their sympathetic physiology would be greater within the sample of depressed participants than would be the case within the nondepressed sample.

In conducting our analyses to test this hypothesis, the primary between-subjects variables were depression status (depressed, nondepressed) and cry status (crier, noncrier), and the within-subject variable was film condition (neutral, sad). Gender was also included as a between-subjects factor. Finally, medication use (medicated, unmedicated) was included in the analyses as a covariate.¹

¹ Because 31 of the 71 depressed participants were receiving pharmacotherapy, we conducted a number of analyses to examine possible medication effects. First, we compared depressed participants who were taking psychotropic medication with those who were not taking medication on their baseline physiology. A multivariate analysis of variance revealed that medication use was not related to baseline levels of sympathetic nervous system variables, $F(1, 66) < 1$. Because hypotheses concerned reactivity to a sad film, it was important to examine the impact of medication on levels of experiential, behavioral, and physiological reactivity. There were no significant differences between depressed participants on medication and depressed participants not on medication with respect to changes in self-reported and observer-rated sadness, both $t(70) < 1$, nor was there a significant effect of medication on physiological responses to the sad film,

If the effects of crying in response to a sad stimulus were enhanced in depressed individuals, we would obtain significant interactions of cry status, film condition, and diagnostic group for each emotion response domain. We could then test our hypothesis by decomposing this interaction and examining the relative magnitude of differences as a function of film condition between criers and noncriers within each diagnostic group. We conducted separate analyses on self-reported sadness, positive affect, and negative affect, observer-rated sadness behavior, and six measures of autonomic functioning.

Hypothesis 3: Crying among depressed participants will be associated with greater psychosocial impairment. To evaluate this hypothesis, we examined whether crying within the sample of depressed individuals was related to severity of depression, length of depressive episode, and scores on a measure of global functioning. If alterations in the crying response reflected the nature and extent of emotion dysregulation, crying behavior among depressed individuals would be related to their level of psychosocial impairment. More specifically, we predicted that among depressed participants, crying during the cry-eliciting film would be positively associated with depression severity and length and negatively associated with global functioning.

To test this hypothesis, we conducted *t* tests comparing crying and noncrying depressed individuals with respect to their current self-reported depression severity and global assessment of functioning. Because of the positive skew in length of depressive episode, we used a chi-square test to examine the association between episode length and crying. For this analysis, we used a cutoff of 6 months (the median length for depressive episodes observed in prior work; Keller et al., 1992) to subdivide the depressed sample into those with shorter and those with longer episode duration.

Method

Participants

Seventy-one depressed persons (66% women and 34% men) and 33 nondepressed psychiatrically healthy controls (70% women and 30% men) participated in the present study. Participants were fluent English speakers between the ages of 18 and 60 years. Approximately half of the depressed participants were recruited from two outpatient psychiatry clinics in a university teaching hospital, and half responded to advertisements posted in numerous locations in the community (e.g., Internet bulletin boards, university kiosks, supermarkets). The depressed participants had no reported history of brain injury, no lifetime history of primary psychotic ideation, no current diagnoses of panic disorder or social phobia, no

$F(1, 66) = 1.21, p > .05$. Our central predictions involved statistical interactions between cry status and diagnostic status in levels of emotional reactivity. Crying among the depressed participants was not related to the number of psychotropic medications used, $t(70) < 1$, nor did depressed psychotropic medication users and nonusers differ in their likelihood of crying, $\chi^2(1, N = 71) = 1.59, = p > .05$. Because medication use was not related to baseline sympathetic parameters or to primary measures of emotional reactivity, a statistical interaction of depression status and cry status would not be confounded by medication use. Nevertheless, because previous research had found medication effects in depressed samples (e.g., Jacobsen, Haukson, & Vestergaard, 1984), we included psychotropic medication status as a covariate in our analyses.

behavioral indications of possible impaired mental status and were not mentally retarded. Depressed 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 the same locations that were used to solicit depressed participants. Potential control participants were excluded from the study on the basis of the same general and medical criteria that were used for the clinical participants. In addition, they were interviewed to exclude those with the presence of lifetime diagnoses of any Axis I disorder (see below). All participants provided written informed consent before the experimental session and were paid \$25 per hour for their participation in the study.

Clinical Assessments

All depressed participants met *DSM-IV* criteria for MDD, as measured by the Structured Clinical Interview for *DSM-IV* Axis I Disorders (SCID-I; First, Gibbons, Spitzer, & Williams, 1995). The SCID-I interviews were conducted by trained advanced graduate students and a post-doctoral fellow. An independent trained rater who was unaware of group membership evaluated 15 randomly selected audiotapes of SCID-I interviews conducted with depressed and nondepressed participants and with nonparticipants who met diagnostic criteria for disorders other than depression (e.g., panic disorder). For each interview, the rater evaluated whether the participant met *DSM-IV* diagnostic criteria for MDD. In all 15 cases, ratings matched the diagnosis made by the original interviewer ($\kappa = 1.00$).

Participants also completed the Beck Depression Inventory (BDI; Beck et al., 1979) and were rated by the interviewers on the Global Assessment of Functioning scale (GAF; American Psychiatric Association, 1994). Finally, for each depressed participant, the SCID-I interviewer determined the length of the current depressive episode.

Psychophysiology Assessments

Film stimuli. Film stimuli were presented on a 20-inch (50.8-cm) television monitor at a viewing distance of 1.75 m. Film selection was based on criteria recommended by Gross and Levenson (1995). All participants viewed two films. A neutral film was used to provide a baseline against which we could compare the effects of the cry-inducing film. (See Piferi, Kline, Younger, and Lawler, 2000, for a discussion of the advantages of neutral film baseline procedures.) The neutral film was piloted to verify that it elicits little emotional behavior or report of emotion. This film lasted 60 s and depicted coastal landscape scenery. The sad film was chosen to evoke themes of loss and abandonment that are salient to depressed individuals (Beck, 1967). This film has been used previously and is known to elicit strong reports of sadness (e.g., Gross, Sutton, & Ketelaar, 1998, see Gross & Levenson, 1995, for published validation data). This film was 170 s in length. The film depicted a social scene in which several individuals grieve in the immediate aftermath of a death. The most poignant figure in the film is a young boy who learns of the death of his father, reacting first with disbelief, then trying to awaken his dead father and, as the scene develops, becoming increasingly distraught and inconsolable.

Self-report measures. An emotion inventory taken from Gross et al. (1998) was used to measure emotion experience at baseline and in response to the sad film. Each item on the emotion inventory was rated on a 9-point scale, ranging from 0 (*not at all*) to 8 (*extremely*). The inventory included sadness, which was the emotion targeted by the cry-inducing film. The inventory also included terms designed to assess the broader dimensions of positive affect and negative affect (Watson & Tellegen, 1985). A broad sampling of affective experience was desirable because depression has been associated with both increased negative affect and decreased positive

affect. The Positive Affect scale included six terms: *elated, enthusiastic, amused, happy, lively, and interested* (α at baseline = .85). The Negative Affect scale included six terms: *annoyed, anxious, distressed, hostile, jittery, and nervous* (α at baseline = .85).

Behavioral measures. A remote-controlled camera positioned behind darkened glass unobtrusively recorded the participants' facial expressive behaviors. Recording took place in low ambient light. To assess behavioral reactivity, sadness and crying behaviors were coded using 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 before beginning work on this project. All raters were unaware of the diagnostic status of participants, the nature of the experimental manipulation, and the study hypotheses. Behavioral coding training required coders to learn prototypes of emotional behaviors (Ekman & Friesen, 1975) as well as to recognize specific molecular behaviors indicative of each prototype. Whereas sadness and crying behaviors are both manifestations of sadness and would therefore tend to be correlated, scores for sadness also reflect a variety of molecular behaviors that do not depend on the presence of crying. Sadness behavior was rated on a 0–6 scale, with each value representing an aggregate of intensity, duration, and frequency of response. For example, a participant would receive a score of 1 on this scale if she or he exhibited a relatively brief downturn of the mouth or a slight upturning of the inner eyebrow with closed body posture and the head moved forward. Participants received a sadness score that reflected their maximal responding to the neutral film and the sad film. To ensure that each film score represented comparable time periods, the longer sad film was divided into two rating periods that were averaged. All segments of behavior were rated by at least two coders. The average interrater agreement for observer-rated sadness was acceptable ($\alpha = .71$). Crying was coded when the participant displayed visible tears in at least one eye, not due to yawning. Discrepancies in crying coding were resolved through consensus discussion after sadness ratings had been completed. Finally, participants' latency to cry was measured by recording the time from the onset of the sad film stimulus to the first appearance of visible tears.

Physiological measures. An SA Instruments 12-channel bioamplifier was used to record physiological responses. Data were acquired using a Pentium PC that used a Data Translation 3001 PCI 12-bit 16-channel analog-to-digital converter. During the experimental session, physiological channels were sampled continuously at 400 Hz. Physiological data were reduced off-line using custom laboratory software written in MATLAB (Wilhelm, Grossman, & Roth, 1999). 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 and derived parameter. Artifactual epochs were edited manually for each channel. A 60-Hz digital notch filter was used to attenuate ambient electronic noise. Period averages were calculated separately for the 60-s neutral film and the 170-s sad film.

Heart rate, skin conductance level and response, finger temperature, respiratory rate, and somatic activity were measured. Three criteria guided selection of these measures: (a) samples from major organ systems known to be important in emotional responding (*cardiac*: heart rate; *vascular*: finger temperature; *electrodermal*: skin conductance level and response; *somatic*: motor activity; *respiratory*: respiratory rate); (b) indexes of the intensity of responding in a sad context (e.g., heart rate acceleration, Schwartz, Weinberger, & Singer, 1981; electrodermal activation and increases in respiratory rate, Levenson, Ekman, Heider, & Friesen, 1992); (3) sensitivity to the effects of crying (all measures, Gross et al., 1994; heart rate acceleration and electrodermal activation, Kraemer & Hastrup, 1988).

The following six measures were obtained:

(a) **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 milliseconds) between successive

R waves in the electrocardiogram (ECG) and was converted to instantaneous heart rate after editing of R–R interval outliers due to movement artifacts or ectopic myocardial activity.

(b) **Skin conductance level:** A constant-voltage device passed 0.5 V between Beckman regular electrodes (using an electrolyte of sodium chloride in Unibase) attached to the palmar surface of the proximal phalanges of the first and second fingers of the nondominant hand. The mean skin conductance level was analyzed after movement and electrode contact artifacts appearing as irregular spikes had been edited out using a special procedure (Wilhelm & Roth, 1996).

(c) **Skin conductance response:** Skin conductance fluctuations were detected as changes in skin conductance level from a zero-slope baseline exceeding 0.2 μ S.

(d) **Finger temperature:** A thermistor attached to the palmar surface of the distal phalange of the fourth finger recorded temperature in degrees Fahrenheit.

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

(f) **Respiratory rate:** Two bands (Respirace) were placed around the upper thorax and abdomen and connected to an inductive plethysmography system (Ambulatory Monitoring, Ardsley, NY).

Procedure

Participants were greeted and were positioned in a comfortable chair facing a video monitor in a quiet, well-furnished laboratory room. After procedures for connecting physiological monitoring devices were explained and the monitors were attached, participants performed a number of experimental tasks that were not relevant to this report. They then were shown neutral and sad films. Participants' expressive behavior and physiology were assessed during the neutral and sad films to permit a continuous assessment of changes that accompanied crying. Because pilot data indicated that assessing self-reported emotional experience between films disrupted film viewing, participants completed their baseline emotion questionnaires immediately before they viewed the neutral film. The sad film was presented directly after the neutral film, and participants' self-reported responses to the sad film were assessed immediately after they viewed the sad film. Participants were simply instructed to watch the films carefully. Finally, to avoid possible carryover effects of the sad film, the neutral film was always shown before the sad film.

Results

Demographic and Clinical Characteristics

As is evident from the data presented in Table 1, the depressed and nondepressed participants did not differ with respect to age, gender composition, and level of education (all $ps > .05$). As expected, the two groups of participants did differ significantly with respect to severity of depressive symptomatology and level of global functioning (both $ps < .001$).

Manipulation Check

We first sought to determine, for all participants, whether the sad film affected the report and display of emotions relevant to crying. As expected on the basis of extensive pretesting, participants reported higher levels of the target emotion of sadness after viewing the sad film ($M = 4.16$, $SD = 2.66$) than they reported on their prefilm emotion inventory ($M = 2.24$, $SD = 2.26$), $t(103) = 6.71$, $p < .001$. The sad film also produced behavior consistent with a sad emotional state: Participants displayed greater sadness behavior while viewing the sad film ($M = 1.31$,

Table 1
Sample Characteristics

Group	n	Age		% Women	Education		BDI		GAF		
		M	SD		M	SD	M	SD	M	SD	
Depressed	71	33.4	10.5	66.2%	33.8%	6.5	1.5	23.1	7.4	87.2	4.2
Nondepressed	33	32.3	11.7	69.7%	30.3%	6.6	1.4	2.6	3.0	53.7	8.4

Note. Education = 1–8 interval scale, a score of 6.5 was equivalent to part college; BDI = Beck Depression Inventory score; GAF = Global Assessment of Functioning score. BDI and GAF groups differ significantly ($p < .001$).

$SD = 1.21$) than they did while viewing the neutral film ($M = 0.19$, $SD = 0.61$), $t(103) = 9.72$, $p < .001$. Finally, in keeping with Gross et al.'s (1994) report of crying frequency, 23 of the 104 participants (23.0%) in this study exhibited observable crying in response to the sad film.

Hypothesis 1: Likelihood of Crying

Contrary to expectation, depressed and nondepressed participants did not differ significantly from each other in their likelihood of crying in response to the sad film (23.6% vs. 18.2%, respectively), $\chi^2(1, N = 104) < 1$. Moreover, depressed and nondepressed criers were not distinguished by either their latency of tear onset ($M = 116.2$ s vs. $M = 110.5$ s, respectively), $t(21) < 1$, or the length of their crying episodes ($M = 54.7$ s vs. $M = 59.5$ s, respectively), $t(21) < 1$. The lack of diagnostic group differences in tearful behavior did not support the hypothesis that major depression is characterized by a lowered threshold for crying.

As suggested by previous research (see Gross et al., 1994), the likelihood of crying did differ as a function of gender: Women were more likely to cry than were men (31% vs. 3%, respectively), $\chi^2(1, N = 104) = 10.50$, $p < .001$. Although this pattern was apparent in both diagnostic groups, our cell sizes did not permit us to examine whether the magnitude of this gender difference varied by diagnosis. In the depressed group, 17 of the 48 women cried (35%) compared with 0 of the 23 men (0%), $\chi^2(1, N = 71) = 11.13$, $p < .001$; in the nondepressed control group, 5 of the 24 women cried (21%) compared with 1 of the 9 men (11%), $\chi^2(1, N = 33) < 1$.

Hypothesis 2: Intensity of the Crying Response

Hypothesis 2 predicted that the experiential, behavioral, and physiological consequences of crying would be enhanced by depression. As we described earlier, in this hypothesis, we predicted three-way interactions of diagnostic group, cry status, and film condition for our measures of emotion experience, expressive behavior, and physiological responding.

Emotion experience. For self-reported sadness, we obtained the predicted three-way interaction of diagnostic group, cry status, and film condition, $F(7, 97) = 3.92$, $p = .05$. Means and standard deviations of self-reported sadness broken down by diagnostic group, cry status, and film condition are presented graphically in Figure 1a. To determine whether depression was associated with an enhanced crying response, two-way analyses of covariance (ANCOVAs; Cry Status \times Film Condition, with medication use as

a covariate) were conducted on self-reported sadness separately for two diagnostic groups. The Cry Status \times Film Condition interaction was not significant for the depressed participants, $F(1, 67) < 1$. The fact that the initial levels of sadness reported by the depressed criers ($M = 4.00$, $SD = 2.24$) were marginally higher than those reported by the depressed noncriers ($M = 2.85$, $SD = 2.10$), $t(69) = 1.93$, $p < .06$, may have contributed to the nonsignificant nature of this interaction. In contrast, the interaction of cry status and film condition was significant for the nondepressed participants, $F(1, 29) = 8.24$, $p < .01$. Follow-up analyses indicated that whereas nondepressed criers and noncriers reported comparable levels of sadness at baseline, $t(31) < 1$, the nondepressed criers reported greater sadness in response to the sad film than did the nondepressed noncriers, $t(31) = 3.91$, $p < .01$. Finally, a change score analysis directly comparing depressed criers' and nondepressed criers' increase in sadness reported to the sad film yielded comparable results: Depressed criers reported significantly smaller increases in self-reported sadness to the sad film ($M = 1.53$, $SD = 2.92$) than did nondepressed criers ($M = 5.83$, $SD = 0.75$), $t(21) = 3.52$, $p < .005$. These findings for self-reported sadness, therefore, are inconsistent with the hypothesis that depression enhances the crying response.

The ANCOVA conducted on self-reported levels of negative affect yielded only a significant main effect for diagnostic group: Not unexpectedly, depressed individuals ($M = 12.11$, $SD = 7.71$) reported more negative affect than did nondepressed individuals ($M = 6.24$, $SD = 4.74$) across both cry status and film condition, $F(1, 96) = 11.33$, $p < .001$. Finally, the ANCOVA conducted on levels of positive affect yielded a main effect for film condition, $F(1, 96) = 64.98$, $p < .001$, which was subsumed by a significant interaction of diagnostic group and film condition, $F(1, 96) = 9.86$, $p < .005$. Follow-up t tests indicated that whereas at baseline, nondepressed participants reported more positive affect ($M = 19.61$, $SD = 9.88$) than did depressed participants ($M = 12.41$, $SD = 6.91$), $t(100) = 4.26$, $p < .001$, the two diagnostic groups did not differ in their report of positive affect in response to the sad film (nondepressed: $M = 5.23$, $SD = 3.89$; depressed: $M = 6.19$, $SD = 5.31$), $t(100) < 1$. In keeping with this pattern of findings, the magnitude of the change in positive affect from baseline to the sad film was greater for the nondepressed than for the depressed participants, $t(100) = 4.57$, $p < .001$.

Expressive behavior. As predicted, the ANCOVA conducted on observer-rated sadness yielded a three-way interaction of cry status, film condition, and diagnostic group, $F(1, 98) = 4.77$, $p < .05$. Means and standard deviations of observer-rated sadness,

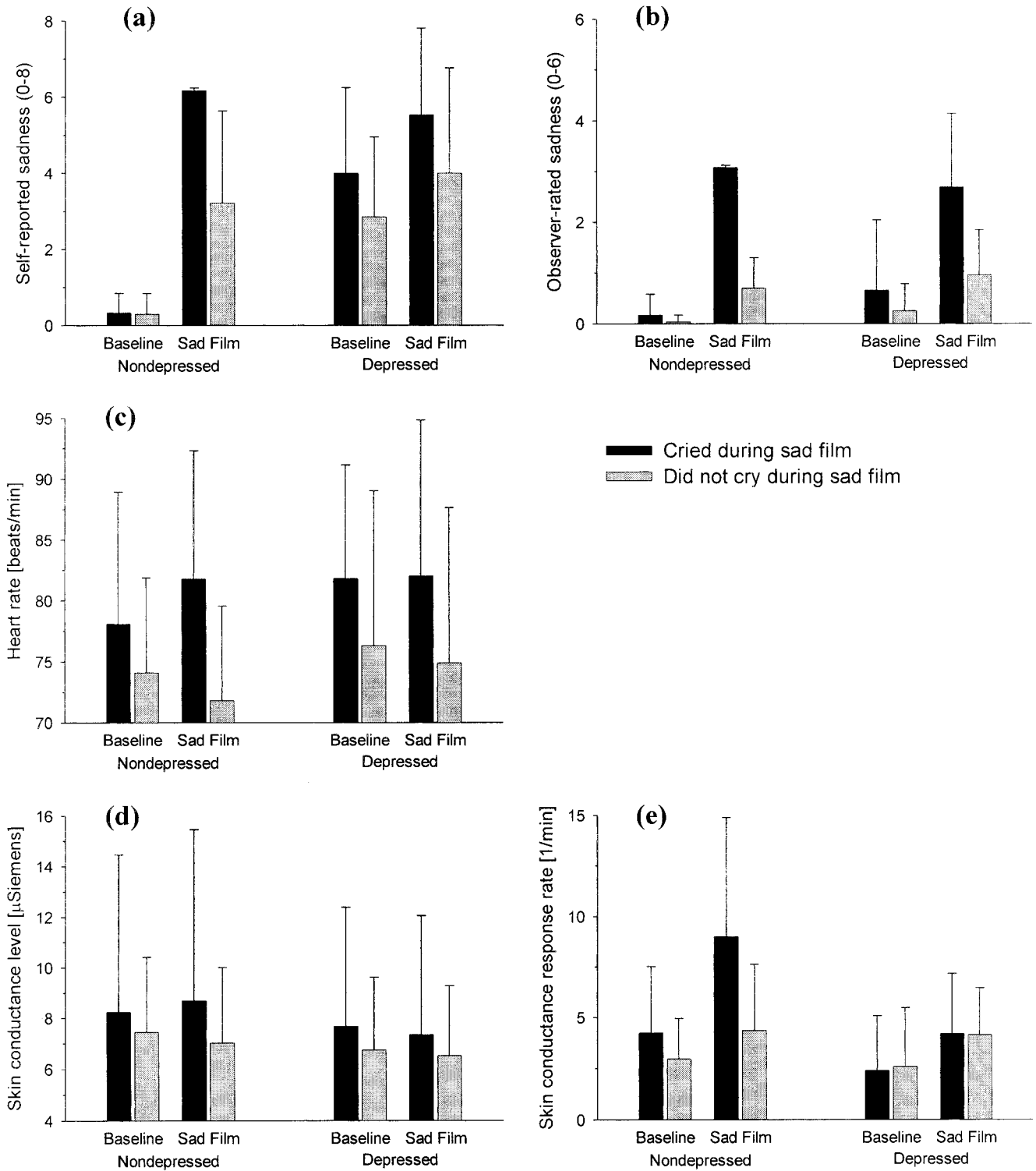


Figure 1. (a) Mean Self-Reported Sadness, (b) mean Observer-Rated Sadness, (c) mean Heart Rate (beats/min), (d) mean Skin Conductance Level [μ S], (e) mean Skin Conductance Response Rate (response count/min) \times Cry Status, Film Condition, and Diagnostic Status. Error bars represent standard deviations. In Panel 1a, *baseline* refers to responses before the neutral film; in Panels 1b–1e, *baseline* refers to responses during the neutral film.

broken down by cry status, film condition, and diagnostic group, are presented graphically in Figure 1b. To determine whether depression was associated with an enhanced crying response, two-way ANCOVAs (Cry Status \times Film Condition, with medication use as a covariate) were conducted on observer-rated sadness separately for each diagnostic group. The interaction of cry status and film condition was significant in both diagnostic groups: nondepressed participants, $F(1, 29) = 47.06, p < .001$; depressed participants $F(1, 68) = 15.88, p < .01$. Follow-up analyses suggested that these two-way interactions were similar in nature in the two groups but different in magnitude. Specifically, follow-up t tests revealed that in the nondepressed group, criers were rated as sadder than noncriers during the sad film, $t(31) = 8.64, p < .001$, but not during the neutral film, $t(31) = 1.40, p > .15$. In the depressed group, criers were rated as sadder than noncriers during the sad film, $t(70) = 6.15, p < .001$, and marginally so during the neutral film, $t(70) = 1.70, p = .08$. Contrary to prediction, then, whether tears were present in response to a sad film had less impact on the sadness behavior of depressed participants than it did for nondepressed participants. Finally, a change score analysis directly comparing depressed criers' with nondepressed criers' increases in observable sadness to the sad film yielded conceptually similar, but statistically nonsignificant, results (depressed criers: $M = 2.04, SD = 1.17$; nondepressed criers: $M = 2.92, SD = 1.02$), $t(21) = 1.62, p > .05$. It is notable that, despite higher levels of observable sadness among depressed criers during the neutral film, there is not evidence of a ceiling effect. Indeed, an inspection of Figure 1b indicates that as expected, the neutral film elicited low levels of observable sadness across all groups (i.e., all four cry groups < 1 on the 0–6 scale).

Autonomic physiology. To examine the consequences of crying on autonomic physiology in depressed and nondepressed participants, a repeated measures multivariate analysis of covariance (MANCOVA) was conducted on the physiological measures. This analysis also yielded the predicted three-way interaction of cry status, film condition, and diagnostic group, $F(1, 95) = 8.07, p < .01$. To determine whether depression was associated with an enhanced crying response, separate two-way MANCOVAs (Cry Status \times Film Condition, with medication use as a covariate) were conducted on the six autonomic parameters for the two diagnostic groups. The interaction of cry status and film condition was not significant for the depressed participants, $F(1, 65) < 1$, but was significant for the nondepressed participants, $F(1, 29) = 11.30, p < .005$.

To determine which variables contributed to this significant interaction for the nondepressed participants, we conducted separate two-way ANCOVAs (Cry Status \times Film Condition) on each of the six physiological measures for this group of participants. These analyses yielded significant interactions of cry status and film condition for heart rate, $F(1, 29) = 16.47, p < .01$, skin conductance level, $F(1, 29) = 5.64, p < .05$, and skin conductance response rate, $F(1, 29) = 7.01, p = .01$. Means and standard deviations of these three variables broken down by cry status, film condition, and diagnostic group are presented graphically in Figures 1c, 1d, and 1e, respectively. Follow-up analyses indicated that nondepressed participants who cried during the sad film had relatively greater cardiac and electrodermal responses than did nondepressed participants who did not cry. Specifically, while exhib-

iting comparable heart rate and skin conductance response rates during the neutral film (all $ps > .05$), during the sad film nondepressed criers had higher heart rates, $t(31) = 2.87, p < .01$, and a greater number of skin conductance responses, $t(31) = 2.70, p < .01$, than did nondepressed noncriers. The two groups of nondepressed participants did not differ in skin conductance level for either the neutral or the sad film, both $ts(31) < .15$. Analyses of change scores conducted to locate the source of the interaction for skin conductance level indicated that nondepressed noncriers exhibited significant decreases in skin conductance level from the neutral to the sad film, $t(26) = 2.73, p < .05$, whereas nondepressed criers did not change their skin conductance level between the two films, $t(5) = 1.43, p > .05$. Thus, whereas nondepressed criers exhibited greater sympathetic activation during the sad film than did nondepressed noncriers, among depressed participants, the presence or absence of observable tears did not have significant physiological consequences. Inspection of the relevant means in Table 2 suggests that ceiling or initial level effects on physiological measures assessed during the neutral film are an unlikely explanation for this pattern of results.

Finally, analyses comparing the change scores of depressed and nondepressed criers on each of these three autonomic variables from the neutral to the sad film yielded a convergent pattern of results. Depressed criers exhibited significantly smaller increases in skin conductance level ($M = -0.34, SD = 0.74$) and skin conductance response rate ($M = 1.82, SD = 2.25$) to the sad film than did nondepressed criers (level: $M = 0.44, SD = 0.76$; rate: $M = 4.76, SD = 3.44$), $t(21) = 2.29, p < .05$, and $t(21) = 2.37, p < .05$, respectively. Depressed criers also had nonsignificantly smaller increases in heart rate to the sad film ($M = 0.20, SD = 6.73$) than did nondepressed criers ($M = 3.69, SD = 2.18$), $t(20) = 1.22, p > .05$. These results, therefore, do not support the hypothesis that the crying response is enhanced by depression.

Hypothesis 3: Clinical Significance of Crying

Before assessing the clinical significance of crying in depression, we examined the associations between crying and demographic variables. Although depressed criers did not differ significantly from depressed noncriers in age or level of education, both $ts(70) < 1$, there was a strong gender difference in crying (as noted previously, depressed men did not cry). Because of this gender difference, we restricted subsequent analyses examining the clinical significance of crying in depression to female depressed participants.

We predicted that the presence of crying behavior would be related to more impaired psychosocial functioning among depressed individuals, as measured by self-reported severity of depression, length of the current depressive episode, and global assessment of functioning scores. The results of the analyses indicated that depressed participants who cried did not differ from depressed participants who did not cry, with respect to either self-reported depression severity, as measured by the BDI, or global functioning, as measured by the GAF, both $ts(46) < 1, p > .05$. In terms of episode length, a significantly smaller proportion of depressed women with longer depressive episodes cried (7 of 28; 25.0%) than was the case among depressed women with

Table 2
Means (and Standard Deviations) of Autonomic Nervous System Measures by Cry Status, Film Condition, and Diagnostic Status

Group	HR		SCL		SCR		ACT		TMP		RR	
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
Depressed criers												
Neutral film	81.81	9.34	7.68	4.71	2.39	2.70	0.89	0.88	89.59	6.12	13.86	5.36
Sad film	82.01	12.82	7.35	4.70	4.21	2.96	1.28	2.88	88.90	6.69	15.05	2.66
Depressed noncriers												
Neutral film	76.31	12.71	6.76	2.86	2.58	2.90	1.30	2.73	89.22	5.97	14.89	3.87
Sad film	74.90	12.74	6.53	2.74	4.14	2.32	0.91	1.02	88.75	5.97	17.46	2.58
Nondepressed criers												
Neutral film	78.09	10.84	8.25	6.21	4.24	3.29	0.86	0.48	94.12	1.77	13.75	2.58
Sad film	81.78	10.54	8.70	6.76	9.00	5.88	0.99	0.45	94.02	1.92	15.32	2.66
Nondepressed noncriers												
Neutral film	74.11	7.76	7.45	2.96	2.96	2.00	1.20	1.70	90.28	5.34	13.57	2.77
Sad film	71.82	7.03	7.03	2.98	4.36	3.27	0.80	1.12	89.66	5.57	16.80	3.31

Note. HR = heart rate (beats/min); SCL = skin conductance level (μ S); SCR = skin conductance response count (count/min); ACT = somatic activity (A/D units); TMP = finger temperature ($^{\circ}$ F); RR = respiratory rate (breaths/min). There was a significant three-way interaction between HR, SCL, and SCR ($p < .05$).

shorter episodes (10 of 19; 52.6%), $\chi^2(1, N = 47) = 3.74, p < .05$, a pattern opposite to that predicted by Hypothesis 3.

Discussion

In this study, we evaluated how clinical depression affects the crying response. On the basis of clinical reports, we expected that depressed participants would exhibit tearful behavior more readily than would nondepressed persons and that depression would enhance the crying response. We also predicted that the presence of tearful crying among the depressed participants would be associated with longer and more severe depressive episodes and with greater psychosocial impairment. None of these predictions were supported. Depressed participants were no more likely to cry than were their nondepressed counterparts. Moreover, among depressed individuals, the presence or absence of crying had relatively little impact on reports or displays of sadness, or on levels of sympathetic arousal. Finally, crying among depressed participants was not related to severity of depression and was in fact associated with shorter, not longer, depressive episodes.

Why Didn't Depression Influence Crying Threshold?

Our finding that there was no difference in crying threshold between depressed and nondepressed participants was particularly surprising. We expected that the depressed participants would begin the experiment in a sad, distressed state and would therefore be predisposed to cry relatively easily. In fact, our finding of no difference in crying threshold between the depressed and nondepressed participants was all the more surprising given that the film we used to elicit sadness depicted themes of relationship loss, death, and abandonment—themes that are strongly salient to currently depressed persons (Beck, 1983). Despite all of these considerations, no differences in crying likelihood emerged. How might our findings be reconciled with the observations in clinical contexts of notable crying among depressed individuals?

One possible reconciliation of this discrepancy is that excessive crying in depression is observable primarily in social situations. It

is well established, for example, that depressed persons are prone to disclose and dramatize their negative experiences to others (e.g., Coyne, 1976). Indeed, out of this concern, we chose to examine the threshold for crying behavior outside of the influence of an interpersonal context. Perhaps we would have observed increased tearfulness in depressed persons had we used an interactional paradigm rather than testing each participant alone. The results of the present study raise the possibility that variations in social context may be an important determinant of crying in depression, and future research might benefit from a systematic examination of this issue.

A second possibility is that there are, in fact, changes in the threshold for crying in depression but that these changes occur only in a subgroup of depressed individuals. Indeed, although no evidence of a lowered crying threshold was obtained in group level analyses, internal analyses within the sample of depressed persons suggested that high levels of sadness may nevertheless facilitate crying in depression. At a trend level, depressed persons who cried in response to a sad film reported more sadness and looked sadder before viewing the sad film than did their noncrying counterparts. Thus, although generally elevated sadness does not appear to facilitate crying in depression, extremely high levels of sadness may do so (Wallbott & Scherer, 1988).

Gender Differences in Crying

We also found that women were more likely than men to cry among both depressed and nondepressed participants. These strong gender differences in observable tearfulness were found in the absence of other gender differences in emotional responding to the sad film. Similar results were obtained by Delp and Sackheim (1987), who measured lacrimal flow directly while participants engaged in sad imagery. These investigators found that although women reported levels of dysphoric affect comparable to those of men, they demonstrated greater increases in lacrimal flow. Although there may be a biological basis for gender differences in psychogenic crying, note that developmental research has gener-

ally found either no sex differences or more frequent and spontaneous crying behavior in boys relative to girls (e.g., Maccoby & Feldman, 1972). A more comprehensive examination of the developmental origins of gender differences in crying would help to clarify this issue.

It is important to underscore here that strong gender differences in crying behavior were observed in both diagnostic groups. Although it would clearly be desirable in future work to ensure that crying is elicited in both males and females to examine the consequences of crying in both genders, it is noteworthy that gender differences in crying were preserved in the depressed state. This pattern is consistent with previous research indicating that compared with depressed females, depressed males are characterized by attenuated outward expression of sad emotion (e.g., Hammen & Padesky, 1977; Vredenburg et al., 1986). In the current study, however, gender differences were observed only with respect to tear presence and not on a measure of overall sadness behavior. The present findings clearly highlight the need for future research to examine more explicitly which aspects of sadness behavior distinguish depressed males from depressed females and to elucidate more systematically the nature of gender differences in the clinical presentation of depression.

Depression, Crying Intensity, and Psychosocial Functioning

As expected on the basis of previous research (e.g., Gross et al., 1994; Kraemer & Hastrup, 1988), normal (i.e., nondepressed) participants who cried in response to a sad film exhibited increases in the report and expression of sadness and showed greater physiological arousal than did nondepressed participants who did not cry. Contrary to our expectations, however, these differences in emotional responding between criers and noncriers were less pronounced among diagnosably depressed individuals than they were among the nondepressed participants. Indeed, the presence or absence of a tearful response to a sad film had relatively little impact on depressed participants' report of sadness, displays of sadness, or their levels of sympathetic arousal. Data concerning the impact of the duration of the current depressive episode on observable crying behavior also suggested that being depressed may serve to blunt the crying response: Those individuals who had been depressed the longest were the least likely to cry.

How might we account for depression-related attenuation of the crying response? One possibility is that these findings concerning the crying response reflect statistical or methodological artifacts that resulted from differences between our participant groups in their initial levels of sadness (e.g., Berntson, Uchino, & Cacioppo, 1994). More specifically, it is possible that we obtained a significant three-way interaction in our analysis of self-reported sadness because depressed criers may have been near ceiling before the sad film, thereby truncating the possible range of change on self-reported sadness as a function of crying. It is worth noting, however, that the other two domains of emotional responding yielded convergent results while being less vulnerable to initial-levels explanations. In fact, group differences in initial levels are an unlikely explanation for the three-way interactions obtained with both the behavioral and the autonomic data. There was no evidence that depressed criers were near ceiling on any of these measures during the neutral film, nor was there evidence of an

association between initial levels and subsequent change in either diagnostic group. (See Myrtek and Foerster, 1986, for a critique of the concept of initial level dependency.) Finally, these statistical considerations cannot explain why longer episodes of depression were associated with a reduced likelihood of crying, because this analysis was conducted entirely within the group of depressed participants.

A more substantive explanation for why the crying response might become less likely and less powerful over time among depressed individuals (rather than more likely and more powerful, as we had hypothesized) involves the prolonged self-regulatory difficulties that typically occur in depression. Depressed individuals, virtually by definition, have a persistent inability to regulate their negative affect (Gross & Muñoz, 1995). Indeed, not only does depression often run a chronic course (e.g., Boland & Keller, in press; Mueller et al., 1996) but there is also evidence to indicate that most depressed individuals experience unrelieved negative affect before the onset of diagnosable episodes of the disorder (Watson & Walker, 1996). Equally important, depression has a corrosive effect on socially supportive relationships (Joiner, in press). Thus, these features of depression suggest that depressed individuals may be discouraged from mounting vigorous crying episodes because of a reduced probability that crying episodes will secure effective help from others or will lead to affect remediation. Clearly, documenting these implications of the present data will be a high priority for future research.

The attenuated crying response in depression also may reflect a broader deficit with respect to emotional functioning in depression. Typically, emotional responses show some degree of coordination, enabling an organism to rapidly mobilize an appropriate response to meaningful stimuli in the environment (Keltner & Gross, 1999; Plutchik, 1980). Indeed, this rapid mobilization was apparent in the well-orchestrated changes in experience, behavior, and physiology that accompanied crying in the nondepressed participants. In contrast, tearfulness was not associated with dramatic changes across systems of emotional responding among depressed persons. In this respect, tearful behavior among depressed persons appeared to be largely disconnected from its usual sequelae. Indeed, the present results are consistent with previous findings indicating low coherence or agreement across domains of emotional responding in depressed persons (Allen, Trinder, & Brennen, 1999; Brown, Schwartz, & Sweeney, 1978). In extending this work and moving toward a broader account of emotional dysregulation in MDD, it will be critical that other emotional responses receive the scrutiny we applied to crying. The findings of the present investigation should serve notice that the integration of basic affective science with research on psychopathology may bear important, and surprising, insights regarding the ways in which emotion-related processes are affected by various forms of disorder.

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