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Reward Fails to Alter Response Bias in Depression

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Several different models postulate that depression is associated with decreased approach-related behavior. Relatively little has been done to date to specifically investigate this issue. In the present study, a signal-detection analysis was used to examine the response biases of dysphoric and nondysphoric female undergraduates during 3 payoff conditions: neutral, reward, and punishment. As predicted, the dysphoric subjects had a smaller change in bias from the neutral to the reward condition compared with the nondysphoric group. The 2 groups did not differ during the neutral and punishment conditions. These findings are consistent with the hypothesis that the left frontal hypoactivation observed in depression reflects a deficit in approach-related behavior.

A number of diverse research traditions converge on the idea that depressives suffer from a fundamental deficit in an approach-related positive affect system. Included among these traditions are (a) the work of Depue and Iacono (1989), who proposed that depressives suffer from a deficit in what they term the behavioral facilitation system (BFS); (b) factor-analytic studies of mood that, while identifying two broad orthogonal factors of positive affect (PA) and negative affect (NA), have shown that depressives are distinguished primarily by low levels of PA (Watson, Clark, & Carey, 1988); and (c) our own (e.g., Henriques & Davidson, 1991) and others' (e.g., Baxter et al., 1989) investigations of patterns of regional anterior cortical activation, in which it has consistently been observed that depressives show decreased activation in a left anterior-based (mostly prefrontal) PA system, which we have interpreted to play a central role in approach-related positive affect (Davidson & Tomarken, 1989).

Little experimental study of the psychological consequences of low PA among depressives has been performed. Rather the deficits in PA, the BFS, and the left anterior cortical system have mostly been related to the phenomenology of depression (see, e.g., Baxter et al., 1989). For research to move forward in this area, it is critical that experimental methods of assessing responsiveness to reward and other features of this PA system be developed and applied to the study of depression. The principal goal of this study was to develop such a method and use it to examine possible deficits among dysphoric subjects in responsiveness to reward. We first discuss some of the salient findings that converge on the notion that depressives suffer from a deficit in a reward-related PA system. We then review research that has applied signal-detection methodology to the study of depression and show how response bias can reflect the operation of an approach-based system. Next, we describe our adaption of these signal-detection methods to specifically assess responsiveness to reward and punishment in dysphoric and control subjects.

Costello (1972) posited that depression results in a decrease in reinforcer effectiveness, such that the impact of rewarding stimuli is diminished. Likewise, Meehl (1975) suggested that depressed individuals do not perceive reward as reinforcing because of a low hedonic capacity. In this tradition, Depue and Iacono (1989) argued that depression arises out of an underactivation of a reward-based system—the BFS. This underarousal results in a blunted engagement with the environment (i.e., a decrease in approach-related behaviors) and is the result of decreased levels of the neurotransmitter dopamine.

One of the more consistent findings from studies of regional cerebral activation in relation to psychopathology is a decrease in left anterior function among depressed subjects (see Davidson, 1992, for a review). We also have reported these findings in our studies that have used electroencephalography to assess patterns of regional brain function. In these studies, we have consistently observed left frontal hypoactivation in both acute and remitted depressives (Henriques & Davidson, 1990, 1991). Similar findings have been found in a small sample of bipolar seasonal affective disorder patients (Allen, Iacono, Depue, & Arbisi, 1993). Decreased left frontal metabolism has been shown to be present in both unipolar and bipolar depressives and in obsessive–compulsives with depression (Baxter et al., 1989). Studies of regional cerebral blood flow have also found decreased left frontal activation among depressives (e.g., Delvenne et al., 1990), although a more global reduction in cerebral blood flow has been found by some investigators (e.g., Sackeim et al., 1990). At the same time, factor-analytic studies of mood have shown that it is a loss of PA that distinguishes depression from other NA states such as anxiety (Bouman & Louteijn, 1986; Watson, Clark, & Carey, 1988). In this context, PA is more reflective of one's "engagement with the environment" (Watson, Clark, & Carey, 1988) than happiness per se. Viewing these two sets of findings within the framework of Davidson's (e.g., 1984, 1988, 1992; Davidson & Tomarken, 1989) model of emotion, which proposes that the anterior regions of the left and right hemispheres are cortical convergence zones (Damasio, 1989) for approach- and withdrawal-related behav-

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ior, we have suggested that the observed left frontal hypoactivation reflects a decrease in approach-related behaviors (e.g., Henriques & Davidson, 1990, 1991). This deficit manifests itself clinically in the symptoms of decreased interest, loss of pleasure, decreased responsiveness to reward, and loss of appetite. This interpretation is also consistent with the finding of decreased levels of PA in "normal" individuals who are characterized by extreme left-sided frontal inactivation (Tomarken, Davidson, Wheeler, & Doss, 1992).

Although these models (i.e., Costello, 1972; Davidson & Tomarken, 1989; Depue & Iacono, 1989; Meehl, 1975) all suggest a deficit in the approach-related functioning of depressives, the question is whether these subjects manifest these deficits behaviorally. The assessment of depressed and nondepressed subjects on behavioral tasks is complicated by the fact that the use of a single parameter of performance (e.g., accuracy) to compare subjects does not take into account possible differences in their decision criterion. For instance, 2 subjects may have very different response styles underlying similar levels of accuracy, with 1 subject having a large number of false alarms and the other a large number of misses. A signal-detection analysis allows the decomposition of a subject's performance into sensitivity and response bias by examining the percentage of both hits and false alarms. Sensitivity is a measure of the subject's ability to discriminate between a target and nontarget stimulus. Response bias reflects a subject's willingness to define an ambiguous stimulus as a target. Changing payoffs for hits and false alarms produces changes in response bias (e.g., Healy & Kubovy, 1978). Depressed subjects might be expected to require a greater degree of certainty before they respond, reflecting a deficit in approach-related functioning. On this view, depressives would have low levels of hits and false alarms, and as such they would be characterized as having a conservative response bias. Subjects with a more liberal bias would be more likely to respond "target" and would have higher levels of both hits and false alarms. In this scenario, the two groups would not differ in their ability to detect the stimuli, as measured by sensitivity (i.e., the ratio of hits to false alarms), only in their willingness to respond to perceived targets.

The first studies to examine response bias in depression were those that investigated memory loss in depressed elderly. Miller and Lewis (1977) found that depressed subjects and controls did not differ from one another on sensitivity; there were, however, significant differences in response bias. This was a result of depressives using a much more conservative response bias. The authors suggested that the depressives' more cautious response style could be reflective of lowered motivation. Other studies of elderly depressives (e.g., Larner, 1977; Niederehe & Camp, 1985) and younger depressives (Corwin, Peselow, Feenan, Rotrosen, & Fieve, 1990; Dunbar & Lishman, 1984; Herskovic, Kietzman, & Sutton, 1986; Johnson, 1988; Malone & Hemsley, 1977) have found similar results. One study found differences in sensitivity but failed to find differences in response bias (Watts, Morris, & MacLeod, 1987). This study used a verbal memory task under two different conditions such that half of the subjects repeated the target words aloud as they were presented and the other subjects were silent during target presentation. Although these authors did not provide the values for response bias, an examination of group means suggests that depressed subjects were more conservative than controls during the silent condition.

Although most of these studies demonstrate a more conservative response bias on the part of depressed subjects, none to date have examined depressed subjects' response to reward contingencies. The hypothesis that depressed subjects are deficient in approach-related behaviors specifically argues that depressed subjects should be less responsive to reward, and in a signal-detection paradigm, depressed subjects, unlike nondepressed subjects, should not change their response bias relative to a neutral condition when rewarded for hits. The present experiment was designed to test this hypothesis. Dysphoric college students were selected on the basis of extreme and stable Beck Depression Inventory (BDI) scores. These subjects are presumed to be at increased risk for depression, and we have previously shown that these subjects, like clinically depressed subjects, have less left frontal activation than normal controls (Davidson, Chapman, & Chapman, 1987; Schaffer, Davidson, & Saron, 1983). Subjects were evaluated on a verbal memory task under three monetary conditions: neutral, reward, and punishment. Payoff contingencies were structured so as to demonstrate a differential deficit in the behavior of dysphoric subjects: In both the reward and punishment conditions, a more liberal response style (i.e., responding target) maximized the subject's earnings. Thus, any differences in the response biases of dysphoric subjects between conditions could not be attributed to differences in the type of responding that maximized payoffs. Furthermore, there were no penalties for false alarms. In a mixed incentive condition (i.e., in which behavior is both rewarded and punished), a subject's response bias reflects the subject's responsiveness to reward relative to punishment. To examine responsiveness to reward uncontaminated by responsiveness to punishment, it is necessary that behavior in single (i.e., reward or punishment) and not mixed (i.e., reward and punishment) incentive conditions be examined. If dysphoria is associated with deficits in responsiveness to reward, then, irrespective of differences in their ability to discriminate target stimuli, dysphoric subjects should not alter their response criterion when their behavior is being rewarded in comparison to a condition in which no incentives are present.

On the basis of the neuropsychological, neurochemical, and psychometric literatures just briefly reviewed, we predicted that the primary difference between depressives and controls is in their reactivity to reward. None of these theories predicts differences between groups in their responsiveness to punishment. Thus, in the present experiment, we did not expect dysphoric and nondysphoric subjects to differ in their responses to punishment: Both groups should become more liberal in their response bias when their behavior is being punished in comparison to a neutral condition. In view of the findings cited previously here from experiments using signal-detection methods in the absence of incentives, we also expected that the dysphoric subjects would have a more conservative bias during the neutral condition. Subjects' response times were also examined because other investigators (e.g., Schwartz et al., 1989) have reported that depressed subjects are slower than normal controls.

Method

Subjects

Thirty-five dysphoric and 22 nondysphoric female subjects were recruited from the introductory psychology pool at the University of Wis-

Table 1
Subject Characteristics by Group

Measure	Dysphoric		Control	
	M	*SD*	*M*	*SD*
Age (years)	19.09	1.28	19.25	1.16
BDI—Time 1	22.96	5.71	0.55	0.51*
BDI—Time 2	18.52	5.78	2.25	1.80*
CRS	19.96	6.92	3.25	2.20*
PANAS—Positive	21.22	5.28	37.25	6.21*
PANAS—Negative	28.17	7.23	16.20	3.30*

Note. For dysphoric $n = 23$; for control $n = 20$. BDI = Beck Depression Inventory; CRS = Carroll Rating Scale for Depression; PANAS = Positive and Negative Affect Schedule.
* Controls versus dysphoric subjects, $p < .0001$.

consin—Madison. Subjects were selected on the basis of their scores on the BDI (Beck, Ward, Mendelson, & Erbaugh, 1961). Nondysphoric control subjects were required to have a BDI score of less than 3, and dysphoric subjects were required to have a BDI score of 16 or greater. At the time of testing, approximately 8 weeks later, controls were required to have a BDI score of less than 6 and dysphoric subjects were required to have a BDI score of 12 or greater.[1] This resulted in a final group of 23 dysphoric and 20 nondysphoric subjects. Because we wished to have the present sample comparable to our electrophysiological studies (e.g., Henriques & Davidson, 1990, 1991) in which right-handedness is an important criterion, all subjects were right-handed, as assessed using the Chapman Handedness Inventory (Chapman & Chapman, 1987). All subjects received course credit for their participation. Relevant subject variables are listed in Table 1.

Procedure

Subjects were given an overview of the experiment, asked to sign a consent form, and oriented to the computer used in this study. Both the task and questionnaires were presented on computer with the aid of the Micro Experimental Laboratory (Psychology Software Tools, Inc., 1988). Subjects were seated 50 to 56 cm from the computer screen. The questionnaires consisted of the BDI (Beck et al., 1961), the trait form of the Positive and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988), and the Carroll Rating Scale for Depression (CRS; Carroll, Feinberg, Smouse, Rawson, & Greden, 1981). The questionnaires were presented first, followed by a practice block of trials that familiarized the subject with the task. The structure of the practice block was similar to the neutral block (described next). However, it contained fewer trials, and the verbal stimuli used in the practice trials were not used in the experimental trials.

The verbal recognition task contained three blocks, which varied in payoff contingencies: neutral, reward, and punishment. In the neutral condition, accuracy feedback was provided visually without monetary incentive. In the reward condition, subjects earned $.10 each time they correctly identified a target word. In the punishment condition, subjects were initially credited $2.50, but $.10 was deducted each time they failed to identify a target word. Subjects were informed at the beginning of the experiment that they would be able to keep all money earned during the course of the experiment. The neutral condition was always presented first, and the reward and punishment conditions followed, counterbalanced across subjects.

Each block was divided into three parts: presentation of target words, color distractor task, and discrimination trials. In the first part, 24 target words were presented consecutively for 400 ms each, with an interstimulus interval (ISI) of 200 ms. All words were in lowercase letters, 1.0 to 1.5 cm in height. The target words were presented in the same order for all subjects. Subjects were informed of the payoff contingencies for the reward and punishment conditions before the presentation of the target words and again before the discrimination trials in each respective block. Immediately after the presentation of the target words, subjects completed the color distractor task. This task was included to increase the difficulty of the verbal recognition task by preventing subjects from rehearsing the target words. The color discrimination task consisted of 20 trials, designed to vary in difficulty, in which subjects were asked to determine whether two consecutively displayed circles had been identical in color. The circles had a diameter of 1.8 cm, were displayed for 100 ms, and had an ISI of 2,000 ms. Subjects received visual feedback on their accuracy after each trial. On completion of the color discrimination task, the subjects were immediately presented with the 24 target and 24 distractor words in a randomized order. In each trial, words were presented one at a time and remained on the screen until the subject pushed one of two keys indicating whether the word was a target or distractor. Subjects received immediate visual feedback of "correct response" or "incorrect response." In the reward and punishment conditions, accuracy feedback was followed by the display of the amount of money the subjects had earned thus far in the block. After the experiment, subjects were paid and debriefed.

Materials

The three word lists were composed of 48 words each: 24 targets and 24 distractors. All words were chosen from Toglia and Battig (1978) and were five to six letters in length. Words were selected so that targets and distractors were matched on imagery, familiarity, and pleasantness across lists. Pilot work with groups of 15 to 20 subjects ensured comparable levels of difficulty across lists. The lists were randomized across the three payoff conditions.

Results

Self-Report Measures

The two groups did not differ in age. As expected, dysphoric subjects reported more depression on both the BDI, $t(26.8) = -12.81$, $p < .0001$, and the CRS, $t(27) = -10.96$, $p < .0001$, at the time of testing. On the PANAS, dysphoric subjects reported less PA, $t(41) = 9.15$, $p < .0001$, and more NA, $t(31.7) = -7.13$, $p < .0001$, compared with control subjects (see Table 1).

Dependent Measures

The nonparametric measures for sensitivity (A′) and response bias (B″) were computed for each subject based on their hit rate (HR) and false alarm rate (FAR) using the following formulas from Grier (1971). These measures do not assume that the target and distractor distributions are normally distributed, and pilot testing revealed that the distributions of A′ and B″ were less skewed than their parametric equivalents. When the HR is greater than or equal to the FAR, $A' = .5 + [(HR - FAR)(1 + HR - FAR)]/[4HR(1 - FAR)]$, and $B'' = [HR(1 - HR) - FAR(1 - FAR)]/[HR(1 - HR) + FAR(1 - FAR)]$. When HR is less than FAR, $A' = .5 + [(FAR - HR)(1 + FAR - HR)]/[4FAR(1 - HR)]$, and $B'' = [FAR(1 - FAR) - HR(1 - HR)]/$

[1] We have previously used these Time 1 and Time 2 BDI cutpoints (Davidson et al., 1987; Schaffer et al., 1983). Although BDI scores of 6 and 12 are cutpoints for no depression and mild depression, we used a more stringent cutpoint at Time 1 to increase the likelihood that dysphoric subjects would still be in a dysphoric mood state at Time 2.

Table 2
Hits and False Alarms During Each Condition

	Neutral		Reward		Punishment	
Group	*M*	*SD*	*M*	*SD*	*M*	*SD*
Controls						
Hits	20.4	1.98	21.40	2.26	20.30	2.34
False alarms	6.8	4.01	9.55	4.08	9.85	4.11
Dysphorics						
Hits	20.17	2.37	19.70	3.04	21.17	1.75
False alarms	6.13	3.29	8.78	3.42	9.30	3.83

Note. For dysphorics, $n = 23$; for controls, $n = 20$.

[FAR(1 − FAR) + HR(1 − HR)]. These formulas produce errors in the computation of B″ when the HR or FAR equals 0.00 or 1.00 (e.g., when the HR equals 1.00, B″ equals −1.00 irrespective of any variations in the FAR). Thus, the following transformations were used before the computation of A′ and B″: HR = (number of hits + 0.5)/(number of targets + 1) and FAR = (number of false alarms + 0.5)/(number of distractors + 1) (Snodgrass & Corwin, 1988). Group means for HR and FAR are listed in Table 2.

Response Bias

A two-way repeated measures multivariate analysis of variance (MANOVA; SAS Institute Inc., 1989) was computed on subjects' response bias[2] using group (dysphoric/nondysphoric) and condition (neutral/reward/punishment) as variables. There was a significant main effect for condition, $F(2, 40) = 5.90$, $p < .006$. This was a result of subjects having a more liberal response bias in the reward and punishment conditions in comparison to the neutral condition. There was also a significant Group × Condition interaction, $F(2, 40) = 4.58$, $p < .02$.[3] This was a result of dysphoric subjects having a more conservative response bias during the reward condition and a more liberal response bias during the punishment condition compared with control subjects. The difference between dysphoric and nondysphoric subjects during the reward condition was significant, $t(41) = -2.03$, $p < .05$. The differences between groups during the neutral, $t(41) = -.20$, $p = .84$, and punishment, $t(41) = 1.51$, $p = .14$, conditions were not significant (see Figure 1). To investigate the possibility that differences in sensitivity were related to differences in response bias, we computed residuals wherein subjects' A′ values were regressed from their B″ values in each condition. The nature of the Group × Condition interaction was unchanged, $F(2, 40) = 3.29$, $p < .05$.[4]

Further examination of the response bias data revealed that for dysphoric subjects B″ did not differ between the neutral and reward conditions, $F(1, 22) = 1.18$, $p = .29$, whereas B″ did differ between the reward and punishment conditions, $F(1, 22) = 6.76$, $p < .02$, and between the neutral and punishment conditions, $F(1, 22) = 11.14$, $p < .004$. The reverse was true for the controls: The difference between neutral and reward was significant, $F(1, 19) = 8.40$, $p < .01$, whereas the differences between reward and punishment and between neutral and punishment were not, $F(1, 19) = 3.26$, $p = .09$, and $F(1, 19) = 1.93$, $p = .18$, respectively.

Sensitivity

A two-way repeated measures MANOVA was computed using A′ as the dependent measure. The within-subjects variable was condition (neutral/reward/punishment) and the between-subjects variable was group (dysphoric/nondysphoric). There was a main effect for condition, $F(2, 40) = 6.18$, $p < .005$. This was a result of all subjects being more accurate during the neutral condition ($M = .85$, $SD = .062$) compared with the reward ($M = .81$, $SD = .072$) and punishment ($M = .81$, $SD = .069$) conditions. The Group × Condition interaction was not significant, $F(2, 40) = 2.91$, $p = .07$. An examination of means within each condition did not reveal any significant group differences (all $ps > .05$; Table 3).

Reaction Times

Median reaction times were computed for each subject for correct and incorrect trials in each payoff condition. These data were then used as the dependent variable in a three-way MANOVA with group (dysphoric/nondysphoric) as the between-subjects variable and accuracy (correct/incorrect) and condition (neutral/reward/punishment) as the within-subjects variables. There was a main effect for accuracy, $F(1, 41) = 42.53$, $p < .0001$, which was a result of all subjects responding faster on correct trials ($M = 899.89$ ms, $SD = 165.97$) compared with trials in which their responses were incorrect ($M = 1111.82$ ms, $SD = 309.93$). There were no significant main effects nor were there interactions with the variables group or condition.

Discussion

Although studies have found that depressed subjects have lower levels of self-reinforcement than nondepressed subjects (e.g., Gotlib, 1982; Rokke & Kozak, 1989), our results are unique in that they demonstrate behavioral differences between dysphoric and nondysphoric subjects in responding to reward. Our findings are at odds with the results of Layne, Gross, and Buckley (1980), who found that depressed subjects, although rating the loss of money as being more aversive than did controls, did not differ from controls in rating the reward value of money. Our dysphoric subjects were more sensitive to the loss of money than were controls, and they were less responsive to reward. This highlights the need to go beyond self-report and examine actual performance in response to varying incentives.

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[2] On the basis of pilot testing, the first two target words in each list were not used in the computation of A′ and B″ to eliminate primary effects.

[3] Snodgrass and Corwin (1988) examined the appropriateness of four different measures of bias and sensitivity. They found a lack of independence between A′ and B″, and these investigators urged the adoption of a two-high-threshold model. Using this measure of bias, the Group × Condition interaction is unchanged, $F(2, 40) = 4.18$, $p = .02$.

[4] Examination of the amount of money subjects earned during the experiment also revealed a significant Group × Condition interaction, $F(1, 41) = 9.58$, $p < .005$. This was a result of control subjects earning more than dysphoric subjects during the reward condition (control $M = \$2.14$, dysphoric $M = \$1.97$) and earning less during the punishment condition (control $M = \$2.12$, dysphoric $M = \$2.21$).

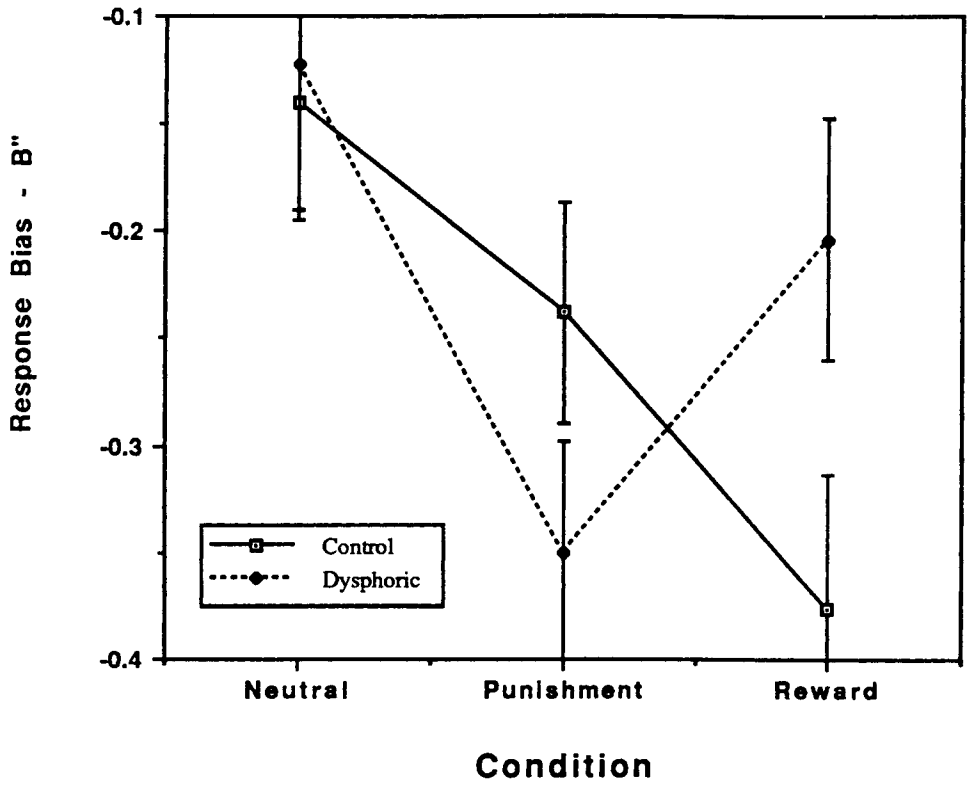

Figure 1. Mean response biases and standard errors of dysphoric (n = 23) and control (n = 20) subjects during differing payoff conditions. Lower numbers on this index denote a more liberal response bias.

The dysphoric subjects' relative insensitivity to reward is consistent with the model put forth early in this article. In that model, the pattern of left frontal hypoactivation observed in depressed subjects is interpreted as reflecting a deficit in approach-related behavior. As predicted, dysphoric subjects had a significantly more conservative response bias in response to reward than did the control subjects. This indifference to reward cues was not the result of a general behavioral deficit in these subjects. During the punishment condition, compared to both the neutral and reward conditions, the dysphoric subjects responded "target" more frequently as evidenced by the greater number of both hits and false alarms and in fact had a more liberal response bias than did nondysphoric subjects. This more liberal response bias, although not statistically significant, is in line with earlier findings of increased sensitivity to aversive stimuli in depression (e.g., Lewinsohn, Lobitz, & Wilson, 1973). Our failure to find significant differences during punishment may well reflect less than adequate power, and it may be that, with a larger sample size or a stronger punishment condition (e.g., shock), significant group differences may have emerged.

Table 3
Sensitivity During Each Payoff Condition

	Neutral		Reward		Punishment	
Group	*M*	*SD*	*M*	*SD*	*M*	*SD*
Controls	.85	.077	.83	.070	.79	.076
Dysphorics	.86	.047	.80	.073	.83	.058

Note. For dysphorics, n = 23; for controls, n = 20.

Depressed subjects have previously reported experiencing decreased levels of reinforcement in their everyday life (e.g., Lewinsohn & Graf, 1973). Buchwald (1977) found that depressed subjects underestimated the frequency of positive reinforcement received, but they did not differ from nondepressed subjects in the amount of reinforcement actually received. Studies of mood and memory have found that depression impairs both the amount of pleasant events recalled as well as the speed with which those memories are retrieved (e.g., Lloyd & Lishman, 1975; Teasdale & Fogarty, 1979). These findings can be understood within the context of our results. If depression results in the decreased salience of reward, then events associated with reward or PA would be less likely to be remembered and thus less likely to be reported.

Contrary to the studies cited earlier (e.g., Miller & Lewis, 1977), we did not find that dysphoric subjects differed from controls during the neutral condition. It is possible that this discrepancy is the result of our providing subjects with accuracy feedback during all conditions. None of the other studies of response bias in depression mention providing subjects with feedback on accuracy. It seems possible that this visual feedback on performance was incentive or, more likely, punishment enough to elicit a more liberal response bias from our dysphoric subjects during the neutral condition. It is also possible that the fact that our subjects were not clinically depressed accounted for this lack of a group difference during the neutral condition.

Although subjects could maximize their earnings by responding "target" to every probe stimulus, none of our subjects responded in such a manner (i.e., no subject responded "target" to every target and every distractor). It is likely that this was because the task was structured such that the emphasis of the experiment was on detecting target stimuli as opposed to earning money. It might be argued that we have simply demonstrated group differences in learning and shown that the reward disrupts learning in dysphoric subjects, whereas punishment disrupts learning in control subjects. However, when we controlled for differences in sensitivity using residualized bias scores, the significant Group × Condition interaction was unchanged.

A concern about the generalizability of our findings relates to our using only female subjects. Our decision to use only female subjects was based in large part on earlier work in our laboratory with this subject population (e.g., Davidson et al., 1987; Schaffer et al., 1983), and we wished to replicate these studies as closely as possible to maximize the likelihood of finding significant group differences. Furthermore, the incidence of depression is greater among women (Nolen-Hoeksema, 1990). Whether similar results will be found in men is a matter for future investigation.

Studies have shown that extreme BDI scores are indicative of increased anxiety and maladjustment as well as increased depression (e.g., Kendall, Hollon, Beck, Hammen, & Ingram, 1987), suggesting that our results may apply to a broad range of disease. The dysphoric subjects in this study reported significantly lower levels of PA on the PANAS than did our nondysphoric subjects. This is consistent with the finding that depressed and anxious subjects differ in their levels of PA, such that anxious subjects do not differ from control subjects, whereas depressed subjects have significantly lower levels on this dimension compared with both controls and anxious subjects (Watson, Clark, & Carey, 1988). As such, we believe that our results may be specific to the depression spectrum of mood disorders. Further support for the specificity of our findings to depression is the fact that all of our dysphoric subjects had scores of 10 or greater on the CRS, and Carroll et al. (1981) suggested a cutpoint of 10 for screening depressed subjects. Finally, we have found that dysphoric subjects selected on the basis of extreme and stable BDI scores have a pattern of left frontal hypoactivation similar to that seen in clinically depressed subjects (Schaffer et al., 1983). Although we do not argue that our subjects could be classified as "clinically depressed," our findings warrant further investigation with a population of clinically depressed subjects.

Another possible explanation for our findings is that dysphoric subjects' decreased responsiveness to reward and increased responsiveness to punishment reflect their lowered levels of PA and higher levels of NA relative to control subjects. Although it is possible that either the low levels of PA or the increased levels of NA alone account for the observed group differences, we believe that the differences in the reward condition may reflect the dysphoric subjects' low levels of PA, and their high levels of NA may manifest as an increased sensitivity to punishment. Within this framework, PA is reflective of an approach-based system, and NA reflects the activity of subjects' withdrawal-based system. Decreases in this approach-based system are hypothesized to manifest clinically in depression as loss of interest, pleasure, and appetite as well as a lack of reactivity to rewarding stimuli, whereas increases in withdrawal-related behavior are presumed to manifest as increases in anxiety and psychomotor agitation. Given the heterogeneity of depression, it may be that variations in symptom profiles are related to variations in responsiveness to reward and punishment. For instance, we predict that decreased responsiveness to reward would, in particular, characterize depressives with melancholic features. At the same time, anxious subjects who differ only from normal subjects in their increased levels of NA (Watson, Clark, & Carey, 1988) are expected to show a more liberal response bias during the punishment condition compared with control subjects. We do not expect group differences between anxious and nonanxious subjects in the reward condition. To further clarify relations among PA, NA, and response bias during reward and punishment, it is necessary to examine this issue in a sample of subjects with more variable PANAS scores.

The fact that our observed group differences are a function of changes in responsiveness to both reward and punishment does not invalidate our main hypothesis that the previously observed decrease in left frontal activation in depressives reflects a general decrease in approach-related behavior and a specific lack of responsiveness to reward. We predict that variations in right frontal activation are reflected in differences in responsiveness to punishment and in differences in withdrawal-related behavior and emotions such as anxiety and fear.

Our data illustrate the application of signal-detection methods in the examination of the effects of incentives on performance and may represent a simple and useful strategy for examining reactivity to reward and punishment cues in clinical populations with affective disturbances.

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