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Low Spirits Keep Rewards Subdued: Decreases in Sensitivity to Reward and Vulnerability to Dysphoria

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Previous theories and research show clear divergences on the roles of the behavioral activation system (BAS) and the behavioral inhibition system (BIS) in depression. Across four studies, we examined the effects of a sad mood on the motivational pattern of sensitivity to reward and punishment. Psychological variables associated with such changes and implications for vulnerability to depression were also explored. For this purpose, we designed a state version of the extensively used BIS/BAS Scales (Carver & White, 1994). Using samples of undergraduate students, we found that both a natural (Study 1) and a laboratory-induced sad mood (Studies 2 and 3) generated a marked decrease in sensitivity to reward but did not alter sensitivity to punishment. Study 3a showed that participants' anxious attachment predicted larger decreases in sensitivity to reward after a sad mood induction. Study 3b extended these results by showing that sensitivity to reward, when assessed after the negative mood induction, predicted increases in dysphoria 7 weeks later. Implications of the results for research on vulnerability to depression are discussed.

Keywords: BIS/BAS; depression; mood; punishment; reward

MANY AUTHORS (e.g., Fowles, 1993; Gray, 1987; Lang, 1995) have proposed the existence of two independent motivational systems for the regulation

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of behavior. Research on these motivational systems has mainly been conducted within the framework of Gray's (1987) reinforcement sensitivity theory. Gray's original model suggested that there are two neurobiologically independent systems of appetitive and aversive motivation. The behavioral activation system (BAS) motivates behavior in response to cues for reward and absence of punishment, and it is also responsible for escape behaviors. The behavioral inhibition system (BIS), in contrast, motivates behavior in response to cues for punishment and absence of reward, controlling for the reaction to novel stimuli. Activation of the BAS is associated with behaviors of approach toward desirable outcomes, feelings of euphoria, and reward seeking. Activation of the BIS is associated with avoidance behaviors, feelings of anxiety in the presence of signs of punishment, and feelings of frustration in the absence of reward (Gray, 1987).1

Although the BIS/BAS proposal has been elaborated and investigated in a number of different respects (e.g., Gray & McNaughton, 2000), its implications for the field of psychopathology are particularly important (see Bijttebier, Beck, Claes, & Vandereycken, 2010, for a review). Gray's theory has had the most significant impact on the study of affective disorders. Regarding depression, Gray suggested that individuals exhibiting greater sensitivity to punishment would be more vulnerable to

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¹When referring to the level of activation of the BIS and BAS, different authors have used various labels, such as impulsivity and anxiety (Gray, 1987), BIS and BAS sensitivities (Carver & White, 1994), and sensitivity to reward and punishment (Torrubia, Avila, Molto, & Caseras, 2001) among others. We employed the labels "sensitivity to reward" and "sensitivity to punishment" throughout this article because we believe they are more clear and neutral than the other labels.

anxious-depressive disorders, whereas greater sensitivity to reward would be related to impulsivity disorders (Gray, 1987). Later, Gray (1991) also hypothesized that low sensitivity to reward might also be related to anhedonic depression.

Similarly, the tripartite model of anxiety and depression proposed that low sensitivity to reward and low positive affect are specific to depression (L. A. Clark & Watson, 1991) but also noted that the BIS is a negative motivational-affective system that is important both in depression and anxiety (L. A. Clark, Watson, & Mineka, 1994; see also Zinbarg & Yoon, 2008). Thus, different theories suggest that depression is associated with lower sensitivity to reward and higher sensitivity to punishment. Various studies have confirmed that depressed individuals present significantly lower levels of sensitivity to reward (Kasch, Rottenberg, Arnow, & Gotlib, 2002; Pinto-Meza et al., 2006) and higher levels of sensitivity to punishment (Johnson, Turner, & Iwata, 2003; Pinto-Meza et al., 2006).

However, other theories have argued that only sensitivity to reward is relevant to understanding depression (Depue & Iacono, 1989; Fowles, 1993) and many research findings support this proposal. For example, although several studies have shown that clinically depressed individuals present reduced reactivity to positive stimuli in general (e.g., Rottenberg, Kasch, Gross, & Gotlib, 2002; see Bylsma, Morris, & Rottenberg, 2008, for a review) and to rewards in particular (Henriques & Davidson, 2000; Henriques, Glowacki, & Davidson, 1994; McFarland & Klein, 2008), they failed to find elevated reactivity or sensitivity to negative stimuli or punishments. The same pattern of results has been observed when analyzing life goals of depressed adolescents. Dickson and MacLeod (2004) found that depression was associated with an approachmotivational deficit but not with augmented avoidance motivation.

In longitudinal studies, only sensitivity to reward and reactivity to positive stimuli, not the BIS or reactivity to negative stimuli, have been able to predict better recovery of depressed patients (Kasch et al., 2002; McFarland, Shankman, Tenke, Bruder, & Klein, 2006; Rottenberg et al., 2002). On the other hand, the only study to our knowledge that examined the ability of sensitivity to reward or anhedonia to predict increases in depressive symptoms over time failed to find significant results (D. C. Clark, Salazar-Gruesco, Grabler, & Fawcett, 1984).

Thus, sensitivity to punishment has not been found to be relevant for depression in laboratory or longitudinal studies. Laboratory studies have found a BAS deficit in depressed individuals, but longitudinal studies have yielded divergent results depending on the sample used: BAS scores predicted changes in depressive symptoms in clinical samples, but no significant results were found when a nondepressed sample was used. What could be the reason for these contradictory findings? Motivational vulnerability may remain latent when individuals are in a euthymic mood state and be activated by negative life events. Consequently, it is possible that only sensitivity to reward assessed after the mood induction, but not before, predicts increases in depressive symptoms. Indeed, some prior work supports this mooddependent hypothesis. Research on vulnerability to depression has found that a sad mood can activate vulnerability processes that remain concealed during a euthymic mood. For example, some studies have found that negative cognitive biases can predict increases of depressive symptoms over time when assessed after a sad mood induction but not when assessed during a euthymic mood (e.g., Beevers & Carver, 2003). Thus, exploring changes in sensitivity to punishment and reward after a sad mood may help to explain the role of such variables in the development of depression.

In summary, some researchers have suggested that depression is associated with a reduced sensitivity to reward and an increased sensitivity to punishment. However, others have hypothesized that only sensitivity to reward is associated with depression. Overall, results from longitudinal studies and from studies using methods other than self-report suggest that only sensitivity to reward is associated with depression. However, it is not entirely clear whether sensitivity to reward predicts an increase in depressive symptoms over time when a nondepressed sample is used.

Exploring the effects of the onset of a depressed mood on the motivational systems could shed light on these contradictory results. Surprisingly, the causal relationship between mood and changes in sensitivity to reward and punishment has not been explored yet. Perhaps the most important explanation for this absence of studies is the lack of selfreport measures that assess state sensitivity to punishment and reward. Therefore, we developed a state measure of sensitivity to reward and punishment based on the BIS/BAS Scales (Carver & White, 1994), the characteristics of which will be detailed below. Some indirect evidence indicates that changes in sensitivity to punishment or reward after the onset of a sad mood are plausible. For instance, it has been found that hedonic capacity, which is theoretically related to the BAS, might diminish after a negative mood (Carson & Adams, 1980). More recently, another line of research has demonstrated that stress induction may reduce reward responsiveness (Bogdan & Pizzagalli, 2006).

In the present study, we explored whether the onset of a sad mood would produce changes in sensitivity to reward, in sensitivity to punishment, or both in order to understand the inconsistencies found across theoretical and empirical evidence. According to some of the previous research, but contrary to Gray's (1987) theoretical model, we expected that only the BAS would be affected by the onset of a sad mood.

Furthermore, we also aimed to evaluate whether sensitivity to reward, sensitivity to punishment, or both are related to vulnerability to depression. To this end, we evaluated whether changes in sensitivity to reward and punishment after the onset of a sad mood were related to other variables typically associated with vulnerability to depression.

Finally, we evaluated whether BIS/BAS scores assessed both before and after the onset of a negative mood were able to predict dysphoria over time. We expected that only BIS/BAS scores assessed after the mood induction would predict increases in dysphoria.

In summary, the present investigation included a naturalistic study, two laboratory studies, and a longitudinal study to better explore the role of sensitivity to reward and punishment in vulnerability to depression.

Study I

In Study 1, we assessed state BIS/BAS scores before and after the natural emergence of a sad mood. We used a diary study (as described by Bolger, Davis, & Rafaeli, 2003) to test the hypothesis that decreases in mood would be followed by decreases in sensitivity to reward but not by increases in sensitivity to punishment. Specifically, we used an event-based design (Bolger et al., 2003). This methodology allowed us to study the target response (i.e., a sad mood) in its natural and spontaneous context. In this study, the onset of a sad mood was the event that signaled participants to complete the questionnaires. Participants first completed a set of questionnaires in the laboratory and then completed another set immediately after the natural emergence of a sad mood.

METHOD

Participants

One hundred twenty-five university students (84% women) were recruited for the study in exchange for course credit. Their mean age was 20.7 years (SD = 1.3).

Measures

State BIS/BAS Scales. The State BIS/BAS Scales (Spanish version) were designed based on the Spanish version of the Trait BIS/BAS Scales (Carver & White, 1994; Perczek, Carver, Price, & Pozo-Kaderman, 2000). Items from the original BIS/BAS Scales were slightly modified to obtain a state measure: Expressions such as "in this moment" or "right now" were included in each of the items. In the original questionnaire, seven items correspond to the BIS Scale (sensitivity to punishment) and 13 to the BAS Scale (sensitivity to reward). Carver and White (1994) found three factors in the BAS Scale: fun-seeking (four items), responsiveness to reward (five items), and drive (four items). We added several items to improve internal consistency, which was just adequate for some subscales in previous research (e.g., Carver & White, 1994). More specifically, we added one item to the BIS Scale and two items to the BAS Scale (one item to the fun-seeking subscale and one item to the drive subscale) so that all BAS subscales had five items. Thus, the new scale, called the "State BIS/BAS Scales," comprised 23 items rated on a Likert scale from 1 (completely disagree) to 7 (completely agree). The total state BIS score ranges from 8 to 56. State BAS scores range from 15 to 105. In order to explore the factor structure of the State BIS/BAS Scales, we added data from 254 individuals of the general population to the present sample (see Hervas & Vazquez, 2011, for a detailed description of the characteristics of this additional sample). Exploratory and confirmatory factor analyses vielded evidence of convergence among the State BAS subscales and, contrary to the Trait BIS/BAS scales (e.g., Ross, Millis, Bonebright, & Bailey, 2002), supported the use of an overall score of state BAS.² Thus, we only reported results regarding overall state BIS and BAS scores. In the present study (N=125), Cronbach's alpha was .87 for the State BIS and .89 for the State BAS.

² First, we carried out a principal components analysis using an oblimin rotation. We initially obtained a four-factor solution (Eigenvalues>1) in which BIS and drive items separated cleanly, three responsiveness-to-reward items blended with the five funseeking items, and the two remaining responsiveness-to-reward items comprised the fourth factor. However, the two-factor solution showed a perfect fit with the BIS and BAS subscales, accounting for 51.5% of the overall variance. More important, we evaluated whether the State BAS subscales represent appropriate second-order factors of the BAS construct (see Ross et al., 2002, for a similar strategy with the Trait BIS/BAS Scales). If so, treating the State BAS subscales as correlated factors should result in a better model fit than when treating them as independent factors (see Schumacker & Lomax, 1996). When the State BAS subscales were allowed to correlate, the model resulted in a good fit, $\chi^2(87) = 244.38$; CFI=.95; TLI=.94; RMSEA=.069, $\chi^2(93)=393.42$; CFI=.91; TLI=.90; RMSEA=.092. The constrained model, where subscales were specified as unrelated, resulted in a worse fit, $\chi^2(93) = 393.42$; CFI=.91; TLI=.90; RMSEA=.092. The difference between these two models was significant, $\chi^2(6)=149.04$, p<.001, confirming the convergence among the State BAS subscales. In addition, the State BAS Scale presented high internal consistency in this combined sample: .91.

Mood Assessment Scale. We assessed current sad and happy mood intensity by means of a set of adjectives rated on a visual scale (Sanz, 2001). Each of the two mood subscales comprised four items. The sadness subscale included the following items: sad, gloomy, low-spirited, and downhearted. The happiness subscale included the following items: happy, cheerful, animated, and hopeful. Each item was rated on an 11-point visual scale from 0 (nothing) to 10 (extremely). The range of scores of each subscale varies from 0 to 40. In the present study, Cronbach's alpha was .89 for sadness and .93 for happiness.

Procedure

Participants who gave informed consent were scheduled at the laboratory (T1). Once there, they completed the initial packet of questionnaires (the State BIS/BAS Scales and Mood Assessment Scale) and were given the same packet to fill out at home (T2) with the following instructions: "When you feel sad or low-spirited for any reason in the next 3 weeks, please complete the attached questionnaires and then take them back to the laboratory the following day." Sixty-six participants (53% of the initial sample) completed the task during the 3 weeks the study was running.

RESULTS

Preliminary Analyses

No significant gender differences were found for any of the variables studied. Participants who completed the second packet of questionnaires differed in mood from those who did not fill it out. Sad mood at T1 was significantly less intense for those who completed the questionnaires at T2 than for those who did not complete them, t(123)=2.20, p<.05.

Changes in Mood and State BIS and BAS Scores

As shown in Table 1, happiness mood scores were lower at T2 than at T1 and this difference was statistically significant, t(65)=16.27, p<.001. Sad mood was significantly higher at T2 than at T1, t(65)=-14.30, p<.001. In addition, sensitivity to reward was lower after the onset of a sad mood compared to baseline assessments, t(65)=11.08, p<.001, whereas sensitivity to punishment revealed no significant changes, t(65)=.65, *ns*.

It was expected that most participants would experience a reduced level of sensitivity to reward to a similar extent. However, the correlation between BAS scores at T1 and T2 was not statistically significant (r=.18, ns). Thus, since the overall mean BAS score decreased, the absence of correlation between T1 and T2 BAS scores suggests that participants' decreases in sensitivity to reward

Table 1

Mean and Standard Deviation of the Variables Included in Study 1 (Data From Participants Who Also Completed Questionnaires at T2 in Parentheses)

	Ν	Μ	SD
Happy Mood			
T1	125 (66)	24.0 (24.3)	8.6 (8.2)
T2	66	6.2	5.8
Sad Mood			
T1	125 (66)	12.4 (11.3)	6.2 (7.3)
T2	66	27.4	5.7
State BAS			
T1	125 (66)	78.6 (79.0)	13.8 (14.9)
T2	66	49.2	18.9
State BIS			
T1	125 (66)	42.9 (43.8)	9.0 (8.7)
T2	66	43.1	8.6

Note. BIS = Behavioral Inhibition System; BAS = Behavioral Activation System; T1 = Time 1; T2 = Time 2.

were not uniform. In fact, the mean change in BAS score was -29.82 and residual change scores from pre- to postinduction on the BAS Scale ranged from 10 to -82. In contrast, the correlation between BIS scores at T1 and T2 was significant (r=.49, p < .001).

Finally, we explored associations between changes in mood and changes in BAS scores using residualized change scores. Individuals who demonstrated decreases in BAS experienced decreases in positive mood (r=.50, p<.001) and increases in sad mood (r=-.34, p<.01).

DISCUSSION

In this study, we used a diary method to explore the extent to which the onset of a sad mood may change BIS/BAS scores from baseline. We found that BAS scores in the global sample decreased significantly. This result indicates that a sad mood involves a motivational alteration.

Our results suggest that vulnerability to suffer decreases in sensitivity to reward is not equally distributed across participants. Moreover, certain participants experienced remarkable drops in BAS scores after the onset of the sad mood. Also consistent with our hypothesis, changes in mood significantly correlated with changes in BAS scores. In contrast, as no global changes in BIS scores were found, we may conclude that the onset of a sad mood does not affect BIS scores. This finding is consistent with laboratory research suggesting that sensitivity to punishment is not altered in depression (e.g., Henriques et al., 1994; Rottenberg et al., 2002).

Limitations to this study include the lack of a control condition (e.g., neutral events) as this would

have helped to determine the specificity of the findings. Also, participants who completed the study differed in mood from those who did not, and this fact could compromise the validity of the results. Some possible explanations are that participants who initially felt sadder were less motivated to complete the task, less likely to experience sad episodes, or less likely to perceive sad moments as sad. In any case, we cannot ensure that results from participants who completed the study would have matched those who did not. Thus, it becomes very relevant to explore whether the same pattern of results will emerge in a laboratory setting using a sad mood induction under controlled conditions.

Study 2

In this study, we attempted to replicate the findings of Study 1, partially modifying the methodology. This time, participants were assessed in the laboratory and then underwent a sad mood induction. Both mood and sensitivity to reward and punishment were assessed before and after the mood induction. We also explored the relation between level of dysphoria, and BIS/BAS levels. According to previous research (Kasch et al., 2002), we hypothesized that dysphoria would be cross-sectionally related to BAS scores but not BIS scores. More specifically, the level of dysphoria would be negatively associated with BAS scores.

METHOD

Participants

Seventy-two university students (76% women) were recruited for the study in exchange for course credit. Their mean age was 21.6 years (SD = 2.0).

Measures

State BIS/BAS Scales. In this study, Cronbach's alpha was .86 for the State BIS and .90 for the State BAS.

Mood Assessment Scale. We assessed mood intensity in the present moment with the same visual scale as in Study 1. In this sample, Cronbach's alpha was .88 for happiness and .86 for sadness.

Beck Depression Inventory-II (BDI-II). We used the Spanish version of the BDI-II (Beck, Steer, & Brown, 1996), which has very good psychometric properties, high internal consistency (.89), and good criterion validity, obtaining a sensitivity of 93% and a specificity of 84% for its optimal cut-score (Sanz, Navarro, & Vazquez, 2003). This scale contains 21 items. The total score ranges from 0 to 63. In this study, Cronbach's alpha was .90.

Procedure

Participants who gave informed consent were recruited from several psychology courses and were scheduled at the laboratory in groups of approximately 10 individuals. There, they were situated in front of a computer separated at least 7 feet from each other and were given an initial packet of questionnaires (i.e., the State BIS/BAS Scales, Mood Assessment Scale, and BDI-II). Then, a sad mood was induced through a 9-minute guided-imagery procedure combined with sad music (Prokofiev's "Russia Under the Mongolian Yoke" played at half speed; see L. A. Clark & Teasdale, 1985). Participants were asked to listen through a pair of earphones to a voice that guided them, step by step, to imagine that they were experiencing the following situation: Their current or imagined partner broke up with them with vague excuses. After the mood induction, participants completed the State BIS/BAS Scales and the Mood Assessment Scale again. Finally, participants were given a positive mood induction before leaving the laboratory.

RESULTS

Table 2 shows the means and standard deviations of the main variables at baseline. Gender differences were only found with respect to baseline BIS scores: Women (M=44.28, SD=6.78) scored significantly higher on the BIS Scale than men (M=39.88, SD=6.05; t(69)=2.39, p<.05). However, a two-way repeated measures ANOVA with the factors gender (male, female) and BIS scores (pre, post) did not find a significant interaction between sex and changes in BIS scores.

Manipulation Check

As Table 2 shows, positive mood scores significantly decreased from pre- to postmood induction, t(71)=11.2; p<.001. Sad mood significantly

Table 2

Mean and Standard Deviation of the Variables Included in Study 2

	Ν	М	SD
Depressive Symptoms	71	8.86	7.00
Happy Mood			
T1	72	23.10	5.97
T2	72	12.98	7.91
Sad Mood			
T1	72	8.28	7.35
T2	72	20.10	9.44
State BAS			
T1	71	83.14	13.08
T2	72	63.72	21.50
State BIS			
T1	71	43.23	6.84
T2	72	42.24	9.05

Note, BIS = Behavioral Inhibition System; BAS = Behavioral Activation System; T1 = Time 1; T2 = Time 2.

increased from pre- to postmood induction, t(71) = -10.4; p < .001. Thus, the sad mood induction was successful.

Changes in State BIS and BAS Scores

As the data in Table 2 show, sensitivity to reward decreased significantly after the sad mood induction, t(70)=7.26, p<.001, and again, sensitivity to punishment did not yield significant changes, t(70)=1.11, *ns*.

The correlation between BAS scores at T1 and T2 was statistically significant (r=.25, p<.05). With regard to the BIS, the correlation between BIS scores at T1 and T2 was also significant (r=.50, p<.001). Residual change scores from pre- to postinduction on the BAS Scale ranged from 29 to -73. Again, some participants experienced very large decreases in sensitivity to reward after the onset of the mood induction.

At T1, we found a significant correlation between BDI-II scores and BAS (r=-.53, p<.001) but not BIS scores (r=.06, ns). No significant correlations were found between BDI-II scores and changes in BIS (r=-.22, ns) or BAS scores (r=.13, ns).

Finally, we explored associations between changes in mood and changes in BAS scores using residualized change scores. Individuals who demonstrated decreases in BAS scores experienced decreases in positive mood (r=.61, p<.001) and increases in sad mood (r=-.50, p<.001).

DISCUSSION

Findings from Study 1 were replicated using a sad mood induction in the laboratory. The sad mood induction produced notable decreases in sensitivity to reward but again failed to modify sensitivity to punishment. Similar to previous studies (e.g., Henriques & Davidson, 2000), we found that the level of dysphoria was associated with low sensitivity to reward but not with high sensitivity to punishment.

It could be argued that these results might have been influenced by a demand effect so that responses of participants are more an artifact of the experimental procedure than real motivational changes. However, we did not find changes in BIS scores. In fact, demand effects after a sad mood induction should influence BIS scores more strongly than BAS scores as a result of the negative content of BIS items. Thus, because we only found changes in BAS scores, our results tend to discount an alternative explanation of the influence of a demand effect.

The baseline level of dysphoria did not predict drops in sensitivity to reward, which was still congruent with our hypothesis. We hypothesized that changes in sadness (i.e., dysphoria) would predict changes in BAS scores; that is, we expected that mood and BAS scores would covary. There was no reason to expect that baseline dysphoria would predict motivational changes.

Our results show evidence that a sad mood can affect basic motivational processes. However, a sad mood hardly produced similar decreases in sensitivity to reward across participants. What are the processes that could explain the acute drops experienced by some participants? We speculate that the magnitude of these differences may represent a marker of vulnerability to depression. In consequence, factors that have previously been demonstrated to predict depression over time may also be responsible for decreases in sensitivity to reward. To examine this possibility, Study 3a explored whether consolidated personality variables related to vulnerability to depression could predict decreases in sensitivity to reward.

Study 3a

In this study, we employed a slightly different mood induction paradigm and attempted to replicate findings from Studies 1 and 2. More important, several personality variables were assessed to explore whether such variables predicted changes in sensitivity to reward.

Previous research has shown that low sensitivity to reward is associated with a deficit in mood regulation (Hervas, Hernangomez, & Vazquez, 2006). In addition, the onset of symptoms of anhedonia is associated with a lack of interest in daily activities (Germans & Kring, 2000). Moreover, another investigation showed that a lack of positive experiences mediated the relation between low sensitivity to reward and symptoms of anhedonia (Beevers & Meyer, 2002).

Similarly, several vulnerability factors for depression have been related to mood dysregulation. For example, attachment style has been associated with difficulties in mood regulation. Pereg and Mikulincer (2004) showed that anxious attachment was associated to a mood regulation deficit after the onset of a sad mood. Likewise, low self-esteem has been shown to be related to mood regulation deficits (e.g., Smith & Petty, 1995). Importantly, research has demonstrated that individuals with low self-esteem present mood regulation problems because of their lack of motivation to repair moods (Heimpel, Wood, Marshall, & Brown, 2002). Furthermore, self-esteem variability has been found to be associated with mood variability and to predict dysphoria in response to negative life events (e.g., Roberts & Gotlib, 1997). Thus, if low sensitivity to reward is associated with mood dysregulation, it is plausible that vulnerability factors such as anxious attachment, self-esteem, or self-esteem variability may predict drops in BAS scores after the onset of a sad

mood. Aside from trait self-esteem, which does not consistently predict the development of depression (Roberts & Monroe, 1999), the rest of the variables have been found to have a potential role as vulnerability factors to depression. More specifically, there is empirical evidence that self-esteem variability (e.g., Roberts & Gotlib, 1997) and attachment style (e.g., Roberts, Gotlib, & Kassel, 1996) are significant predictors of dysphoria over time.

In sum, we hypothesized that high self-esteem variability, low self-esteem, and anxious attachment would be positively associated with decreases in BAS scores after a sad mood induction. If these factors predict changes in BAS scores, this would provide preliminary evidence for the potential role of motivational changes in the processes of vulnerability to depression.

METHOD

Participants

A total of 55 students were initially assessed of whom 49 adequately completed the two sessions; therefore, only their data were included (84% women). Participants were recruited for the study in exchange for course credit. Their mean age was 20.6 years (SD = 1.36).

Measures

Similarly to Studies 1–3, we used the State BIS/BAS Scales, the Mood Assessment Scale, and the BDI-II. Cronbach's alphas are presented in Table 3.

Rosenberg Self-Esteem Questionnaire (RSQ). The RSQ scale (Rosenberg, 1965) is a 10-item scale for assessing global self-esteem. Each item is rated on a Likert scale ranging from 1 (*strongly disagree*) to 4 (*strongly agree*). Overall scores can range from 10 to 40 with higher scores indicating higher selfesteem. We used the Spanish version of the RSQ (Zubizarreta et al., 1994), which has very good psychometric properties, high internal consistency (.92), and high test-retest reliability (.85). Conver-

Table 3 Descriptive Statistics of the Variables Included in Study 3 gent and discriminant validity for the Spanish RSQ has proven to be satisfactory (Zubizarreta et al., 1994).

Experiences in Close Relationships Inventory (*ECRI*). he ECRI attachment scale (Brennan, Clark, & Shaver, 1998) comprises 36 items with a Likert scale ranging from 1 (*strongly disagree*) to 7 (*strongly agree*) and two subscales: anxious style and avoidant style. The scores of each subscale range from 18 to 126 points with higher scores corresponding to higher intensity of the dysfunctional attachment style. For this research, we used the Spanish version of the ECRI (Hervas, 2006), which presents good internal consistency for anxiety (.88) and avoidance (.93) as well as 1-year test–retest reliability (.79 for anxiety; .74 for avoidance).

Self-esteem variability. To assess self-esteem variability, we employed a strategy similar to that used in previous research (e.g., Roberts & Gotlib, 1997). In order to calculate the degree of variability in self-esteem, we assessed participants' self-esteem for 10 consecutive days using the RSQ. We used the standard deviation of these measures as an index of self-esteem variability. In the Procedure section, we describe in more detail the approach for assessing this variable.

Procedure

Participants who gave informed consent were scheduled in groups of 12 to 15 people for the laboratory session (T1) where they completed the State BIS/BAS Scales, Mood Assessment Scale, BDI-II, RSQ, and ECRI. One week later, participants returned for a second appointment. In the laboratory, they were seated in front of a computer and wore headphones. Each participant was at least 7 feet from the next. They completed the State BIS/BAS Scales and underwent a sad mood induction. Thus, state BIS and BAS assessments were separated by 1 week to explore whether changes in BIS/BAS scores would be

	Ν	М	SD	Cronbach's α	1	2	3	4	5	6
1. Depressive Symptoms	49	8.36	6.04	.77						
2. Anxious Attachment	49	71.18	16.37	.79	.26					
3. Avoidant Attachment	49	44.17	14.23	.74	.23	.04				
4. Trait Self-Esteem	49	34.61	4.6	.83	56***	24	45**			
5. Self-Esteem Variability	47	5.99	5.8	-	.09	.24	.03	23		
6. State BAS Pre	49	83.34	12.9	.90	29	.08	20	.48**	.11	
7. State BIS Pre	49	41.73	7.4	.82	.11	.43**	06	28	.12	12

Note. BIS = Behavioral Inhibition System; BAS = Behavioral Activation System. p < .05, ** p < .01, *** p < .001.

facilitated when the practice effect due to repeated administration was minimized.

The sad mood induction was carried out with music while asking participants to imagine three negative scenes (being sick in bed with the flu, visiting a dying friend at the hospital, meeting a friend who tells you about recent serious adversities that happened to him or her) for a total duration of 9 minutes (3 minutes per scene). The piece of music was the same one used in Study 2. Before the imagery task, participants received instructions aimed at facilitating their imagination to be as realistic and as effective as possible.

Immediately after the mood induction (T2), participants were asked to complete the State BIS/BAS Scales and Mood Assessment Scale. Finally, they underwent a positive mood induction (i.e., a 10-minute funny video) in order to ensure that the induced sad mood did not persist when leaving the session. During the following 10 days, they completed a daily measure of self-esteem in order to assess self-esteem variability. Each day, participants were required to hand in their questionnaire at a preestablished location in the university center. If they were absent from the center on any day, they could send their questionnaire in by e-mail. Questionnaires that were not handed in on time were not included in the analyses. Only the data of participants who completed the self-esteem measures correctly at least seven times were included (47 participants).

RESULTS

Table 3 shows the means and standard deviations of the variables at baseline. No significant gender differences were found in any of the variables under study.

Manipulation Check

The sad mood induction significantly reduced happy mood, t(48)=8.97, p<.001, from pre- (M=25.43, SD=6.75) to postinduction (M=15.96, SD=8.69). Likewise, the induction significantly increased sad mood, t(48)=-7.17, p<.001, from pre- (M=6.75, SD=6.81) to postinduction (M=16.53, SD=9.50).

Changes in State BIS/BAS Scores

We explored changes in sensitivity to reward and punishment. Sensitivity to reward decreased significantly after the sad mood induction, t(48)=4.35, p<.001, from T1 (M=83.34, SD=12.91) to T2 (M=71.57, SD=16.48) and again, sensitivity to punishment did not show significant changes, t(48)=-1.43, *ns*, from T1 (M=41.73, SD=7.39) to T2 (M=42.92, SD=7.55). Residual change scores from pre- to postinduction on the BAS Scale ranged from 17 to -67.

As in Study 1, the correlation between BAS scores at T1 and T2 was very low and not statistically significant (r=.19, ns). With regard to sensitivity to punishment, the correlation between BIS scores at T1 and T2 was high and significant (r=.70, p<.001).

Predictors of Changes in BAS Scores

Since changes in BIS and BAS scores were calculated by subtracting the scores that were collected upon completing the initial questionnaires from the postinduction scores, mood changes were calculated using the same time intervals (see Table 4).

Anxious attachment exhibited a significant correlation with decreases in BAS scores (r=-.31, p<.05) and remained significant after controlling for both happy and sad mood changes (pr=-.33, p<.05). No significant correlations were found between anxious attachment and changes in BIS scores.

Self-esteem variability showed a marginally significant correlation with increases in BIS scores from T1 to T2 (r=.19, p<.10) and with decreases in BAS scores from T1 to T2 (r=-.19, p<.10).

DISCUSSION

First, the results obtained in Studies 1 and 2 regarding changes in sensitivity to reward and punishment were replicated. Moreover, this study revealed that a variable linked to vulnerability to depression was associated with this motivational change. More specifically, we found that anxious attachment was associated with decreases in sensitivity to reward, even when controlling for the effect of mood change. These findings suggest that changes in sensitivity to reward may actually be related to vulnerability processes leading to depression. That is, the results suggest that motivational changes could be the mediation link between several vulnerability factors and future depression.

Table 4

Correlations Between Predictors and Changes in BIS and BAS Scores With and Without Controlling for Changes in Mood

		-	-		
			Controlling for Changes in Positive and Negative Mood		
	BAS Change	BIS Change	BAS Change	BIS Change	
Anxious Attachment	31*	.02	33*	.03	
Avoidant Attachment	.04	24	06	20	
Trait Self-Esteem	22	.19	04	.15	
Self-esteem Variability	19	.19	29 **	.17	

Note. BIS = Behavioral Inhibition System; BAS = Behavioral Activation System.

**p*<.05, ** *p*<.10.

Interestingly, the variable that predicted decreases in sensitivity to reward (i.e., anxious attachment) and the variable that showed a trend in the same direction (i.e., self-esteem variability) have something in common: They are both theoretically and empirically linked to fragile self-esteem (Roberts & Monroe, 1999). It has been proposed that individuals with anxious attachment are characterized by having a negative self-view (Bartholomew & Horowitz, 1991). Thus, these results imply that motivational changes after the onset of a sad mood may be closely linked to decreases in self-esteem.

Trait self-esteem did not significantly predict changes in BAS scores. However, self-esteem highly and positively correlated with baseline BAS scores. Thus, our results are consistent with previous research showing that individuals with low selfesteem present low tendency to repair sad moods. This is probably due to the fact that these individuals present low sensitivity to reward even in the absence of a sad mood. In addition, it is also possible that a floor effect has limited our ability to detect significant changes in BAS scores in these individuals.

This study further corroborates the construct validity of the State BIS/BAS Scales. Anxious attachment positively correlated with baseline BIS scores, which replicates previous research (Meyer, Olivier, & Roth, 2004). Moreover, anxious attachment predicted changes in state BAS scores after the sad mood induction as hypothesized.

This latter result tends to discount some alternative explanations about the validity of our results. For example, because we used self-report to assess sensitivity to reward and punishment through the State BIS/BAS Scales in Studies 1–3, it could be argued that decreases in BAS scores after a sad mood could be a result of changes in self-perception or willingness to introspect accurately without representing actual motivational changes. If this were true, however, anxious attachment would not be expected to predict such artifactual changes.

It also can be argued that changes in BAS scores could simply be an epiphenomenon of the mood alteration generated by the mood induction. That is, vulnerability factors could be increasing sad mood and thereby causing changes in BAS scores. However, because the correlation between anxious attachment and changes in BAS scores remained significant even after controlling for pre–post mood changes, this possibility can be discarded. Our results suggest that the vulnerability factors are directly responsible for changes in sensitivity to reward.

Moreover, this study shows a novel association between a psychological vulnerability factor (i.e., anxious attachment) and motivational changes.

Study 3b

We followed up with participants from Study 3a for 7 weeks. The main aim was to examine the capacity of state BIS/BAS scores to predict increases in dysphoria over time. Based on the results from Studies 1–3a, we expected sensitivity to reward to play a significant role in vulnerability to depression. Previous research has also found that sensitivity to reward is able to predict clinical outcomes longitudinally (e.g., Rottenberg et al., 2002).

Previous longitudinal research found that anhedonia does not predict depression over time (D. C. Clark et al., 1984). As such, we could not expect our measure of sensitivity to reward assessed before mood induction to be able to predict dysphoria over time. Thus, it was hypothesized that only state BAS scores after the sad mood induction would be able to predict increases in dysphoria 7 weeks later. By contrast, we did not expect state BIS scores to predict dysphoria over time because trait BIS scores failed to predict recovery in previous longitudinal studies with depressed samples (e.g., Rottenberg et al., 2002).

METHOD

Participants, Measures, and Procedure

A total of 43 students out of the 49 students in Study 3a were reassessed 7 weeks later. Participants were contacted individually and they filled out the BDI-II again in the laboratory.

RESULTS

We conducted a series of regression analyses in order to evaluate significant predictors of changes in dysphoria over time. In all the regression analyses conducted, we used BDI-II scores at T2 as the criterion and introduced the initial BDI-II scores (i.e., T1) at the first step. This first step explained a large proportion of variance ($\beta = .68$; p < .001). Then, we introduced the variables included in Study 3a one at a time in separate analyses (i.e., trait self-esteem, self-esteem variability, anxious attachment, preand postinduction BIS/BAS scores, and residualized change in BIS and BAS). Only two variables were able to predict changes in dysphoria: state BAS scores when tested after the sad mood induction ($\beta = -.26$; p=.01) and residualized change in BAS ($\beta=-.26$; p = .01). Because none of the vulnerability variables, including anxious attachment, was able to predict dysphoria over time, mediational analyses were not conducted.

DISCUSSION

Study 3b yielded results consistent with the previous three studies. We evaluated potential predictors of

increases in dysphoria over time. Again, the results demonstrate that sensitivity to reward played an important role in depression vulnerability, whereas sensitivity to punishment failed to play a significant role. The pattern of results was particularly interesting: We found that both BAS scores after the sad mood induction and the magnitude of drops in BAS scores predicted increases in dysphoria 7 weeks later.

General Discussion

Various long-established motivational theories of depression posit that both sensitivity to reward and sensitivity to punishment are relevant for understanding the onset and development of depressive disorders (e.g., L. A. Clark et al., 1994; Gray, 1987). Conversely, empirical evidence from several sources converges in showing that only sensitivity to reward is altered in depression (e.g., Henriques et al., 1994) and moreover, that only this factor has a role in the maintenance of depression (e.g., Kasch et al., 2002).

MOOD, BIS, AND DYSPHORIA

Studies 1–3 consistently showed that the onset of a sad mood-natural or induced-did not affect sensitivity to punishment but considerably reduced sensitivity to reward. The tripartite theory of anxiety and depression posits that the high negative affect typically observed in depression may reflect the role of high sensitivity to punishment in the development of depressive disorders (L. A. Clark et al., 1994). However, negative affectivity of depressed patients may not necessarily be a consequence of enhanced reactivity to punishment. First, low sensitivity to reward might thwart effective mood regulation, which could indirectly maintain high levels of negative affect (Hervas et al., 2006). Second, depressed individuals' persistent negative mood can be derived from a complex emotional experience (Hervas & Vazquez, 2011) or be a result of daily minor hassles (Hammen, 2005). And finally, it may also arise from negative appraisals of oneself, the world, or the future (Beck, Rush, Shaw, & Emery, 1979). In summary, a number of factors can contribute to maintaining high levels of negative affect even in individuals with average sensitivity to punishment.

In several studies, scores on the BIS Scale were higher in depressed patients than in healthy controls (e.g., Kasch et al., 2002). Yet, comorbid anxiety may be artificially increasing BIS scores in these participants. Although two studies controlled for comorbidity with other anxiety disorders, they failed to control for anxiety symptoms (Johnson et al., 2003; Kasch et al., 2002). In a third study, when anxiety symptoms were controlled, differences in sensitivity to punishment between depressed individuals and controls became nonsignificant (Pinto-Meza et al., 2006).

To summarize, we failed to find increases in BIS scores after a sad mood induction. Likewise, BIS scores did not predict increases of dysphoria over time. However, our results do not deny the potential influence of sensitivity to punishment on depression onset. For example, it might have turned out to be a significant predictor of depression in a larger sample. Nevertheless, we believe such an influence of the BIS to be less likely because previous research has consistently shown that BIS scores are not able to predict changes in depression severity (Kasch et al., 2002; McFarland et al., 2006).

MOOD, BAS, AND DYSPHORIA

Across Studies 1-3a, we found that decreases in sensitivity to reward after the onset of a negative mood were far from uniform as some were surprisingly large. Could these substantial drops have implications for vulnerability to depression? Our results from Studies 3a and 3b suggest that they do. We found that a depression-related factor, anxious attachment style, was directly associated with the magnitude of decreases in BAS scores. Interestingly, some authors have proposed that people with anxious attachment have a negative self-view (Bartholomew & Horowitz, 1991). Recent research has also suggested that individuals with anxious attachment are uncertain about their own self-concept, resulting in a more vulnerable selfesteem (Wu, 2009). Thus, taking into account previous research and our own pattern of results, we hypothesize that drops in self-esteem may promote or exacerbate decreases in sensitivity to reward. Future research should explore this intriguing possibility.

BAS AND VULNERABILITY TO DEPRESSION

Our results provide support for the rather unexplored idea that sensitivity to reward could be a vulnerability factor to depression (see Carver, Johnston, & Joormann, 2008, for an exception). We found that sensitivity to reward assessed after the mood induction predicted increases in dysphoria 7 weeks later. Studies 1–3a demonstrated that the magnitude of changes in BAS scores presents a great variability. As we noted above, some participants reacted to the onset of a sad mood with very large drops in BAS scores and Study 3b confirmed that the magnitude of decreased BAS scores is relevant for depression vulnerability.

As previously discussed, sensitivity to reward assessed before an induced sad mood did not predict depressive symptoms over time in previous research (e.g., D. C. Clark et al., 1984). In other words, motivational vulnerability to depression might become relevant only when depressed mood appears. This result is consistent with some studies that use a mood-priming methodology in which researchers found that cognitive vulnerability factors in previously depressed individuals became active only after a negative mood induction (e.g., Beevers & Carver, 2003). Thus, as occurs with other cognitive factors, it is possible that sensitivity to reward may predict depression over time only when it is evaluated after a sad mood induction. Conversely, evaluating increases in sensitivity to reward after a positive mood induction could be useful in predicting future mania episodes (see Urosevic, Abramson, Harmon-Jones, & Alloy, 2008).

Previous research suggests some explanations for why a large drop in sensitivity to reward could favor the onset of a depressive episode. A very low level of sensitivity to reward can generate states of anhedonia, which could hinder the activation of adaptive coping and emotional regulation strategies. In fact, low sensitivity to reward and anhedonia are associated with a lack of interest in daily activities (Germans & Kring, 2000) and with a lack of positive experiences (Beevers & Meyer, 2002). Lack of interest in positive experiences might foster mood dysregulation, which, in turn, would perpetuate dysphoria.

THE STATE BIS/BAS SCALES

This research also offers an initial validation of the State BIS/BAS Scales. Overall, this new measure seems to be reliable and valid in light of the results obtained. The pattern of associations with depressionrelated variables provides evidence for its construct validity. The State BAS Scale has shown good predictive validity and sensitivity to change. Although sensitivity to punishment did not change after the sad mood onset in Studies 1–3, this does not necessarily reflect poor sensitivity to change. For example, it is conceivable that sensitivity to punishment can change after the induction of anxiety or uncertainty (e.g., Gray, 1987). Moreover, other mood alterations may significantly affect BIS scores. Some studies demonstrate that state BIS scores—using the same scale we used here-decreased after the onset of a positive mood (Hervas & Sanchez-Lopez, 2012). Regarding their structural validity, it is important to note that an exploratory factor analysis did not initially yield a clear-cut structure. However, we found evidence of convergence among the three BAS subscales, which is important because it supports the validity of a two-factor structure (i.e., BIS and BAS).

For future research, it would be valuable to explore the convergence between this self-report measure and BIS/BAS-related behaviors. It is important to note that for some widely used state scales, such as state self-esteem, there has been very scarce reported evidence about their behavior correlates (Heatherton & Polivy, 1991). Moreover, because the original BIS/BAS Scales successfully predicted behavioral outcomes (e.g., Carver & White, 1994) and the new scales were based on them, we may expect a similar performance. Nevertheless, we think that it would be important to further validate or even refine the State BIS/BAS Scales.

LIMITATIONS

The generalizability of these results is conditioned by some limitations. First, the sample used in these studies was not representative of the general population. Future research should replicate these results with more representative samples. Furthermore, in Studies 3a and 3b, some results might have been significant with a larger sample (i.e., more statistical power). For example, in Study 3a, selfesteem variability was marginally significant as a predictor of changes in BAS scores. It would have probably been significant in a larger sample. Likewise, some vulnerability factors included might have been significant predictors of dysphoria over time in a more powerful analysis. However, this fact does not invalidate the main hypothesis and conclusions drawn in this research (i.e., the mooddependent role of the BAS as a vulnerability factor for depression). We believe that sample size would have been a limitation if our aim had been to accurately evaluate which variables are related to changes in BIS and BAS scores, and which are not. Future research will determine the precise variables that are related to depression through a BAS-drop mechanism, but such a complex aim exceeds the scope of the present research.

As a second limitation, findings from this research may not necessarily be generalizable to clinical disorders. Future research should examine whether low sensitivity to reward after a sad mood induction predicts the onset of a clinical depressive disorder. Although there is substantial evidence on the dimensional nature of depression (Flett, Vredenburg, & Krames, 1997; Prisciandaro & Roberts, 2009), it is crucial to evaluate whether sensitivity to reward may account for more severe outcomes. Third, it would have been interesting to include a control group (i.e., neutral mood) in Studies 1-3a in order to compare changes in BIS and BAS scores after the onset of a negative mood with the changes produced over time. Finally, the present studies rely solely on self-report measures and some of the shared variance reported among measures could be partially due to the common method.

Conclusion

Cognitive, personality, and interpersonal theories have sought to explain vulnerability to depression, but the role of motivational factors, especially in relation to other well-validated theories, has been largely neglected. We hope this research encourages future investigations on whether and how motivation intertwines with other vulnerability processes increasing the probability of suffering clinical depressive episodes.

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