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Mood Regulation Skill and the Symptoms of Endogenous and Hopelessness Depression in Spanish High School Students

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To assess the construct validity of the endogenous subtype of depression, 2 studies examined the relation of mood regulation skill to the symptoms of endogenous depression versus hopelessness depression in adolescent samples. It was hypothesized that the relationship between mood regulation skill and endogenous depression would be weaker than the relationship between mood regulation skill and hopelessness depression, because endogenous depression may be less influenced by personality variables, such as mood regulation skill. In spite of the high degree of correlation between the symptoms of hopelessness depression and endogenous depression, the relationship between mood regulation skill and hopelessness depression was stronger than the relationship between mood regulation skill and endogenous depression, as predicted. This is consistent with proposed definition of endogenous depression.

KEY WORDS: mood regulation; endogenous depression; hopelessness depression.

Past work supports the validity of a distinguishable symptom cluster termed "endogenous depression" (Lewinsohn, Zeiss, Zeiss, & Haller, 1977). Symptoms include loss of interest in food and sex, psychomotor retardation, and disturbances in sleep patterns. Endogenous depression has been defined as a state of depression for which the primary precipitating cause is linked to somatic or biological factors, as opposed to situational factors occurring in the environment of the individual. Endogenous depression can be conceptualized a syndrome that includes the symptoms of the melancholic features specifier of depression described in the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision (DSM-IV-TR;* American Psychiatric Association, 2000). Its essential feature is "lack of reactivity" to pleasurable stimuli (p. 419). Individuals with melancholic features are "less likely to have a clear precipitant to the episode" and are more likely to exhibit other biological symptoms (p. 419).

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Regarding the validity of the distinction between endogenous and other symptoms of depression, the endogenous cluster is shown to have a different prognosis and course from other types of depression (Leary, 1996). In addition, those classified as having endogenous depression demonstrate a different response to antidepressant drug treatment (Peselow, Robins, Sanfilipo, & Block, 1992) and to sleep deprivation treatment (Wirz-Justice & Van de Hoofdakker, 1999). Parker, Hadzi-Pavlovic, Wilhelm, and Hickie (1994) identified various biological factors that differentiated endogenous and nonendogenous depressions, including slower reaction times and higher rates of cortisol suppression failure. A relatively clear finding is that symptoms of endogenous depression are characterized by a distinctive feature, or specifically, lack of reactivity to the environment. This lack of reactivity was emphasized by Klein (1974) in his classification of endogenous depression. The symptomatology was characterized by a lack of interest and participation in the environment, exhibited in the decreased ability to enjoy sex, food, and recreational activities. Lewinsohn et al. (1977) examined this distinction further in a factor analytic study of depressive symptoms within three samples of depressed patients. Endogeneity and reactivity were found to exist as

separate and independent dimensions. Diminished reactivity to environmental changes, decreased interest in life, and psychomotor retardation were some of the depressive symptoms loading consistently on the endogenous factor. According to their findings, a precipitating life event was less likely to occur in temporal proximity to the expression of depressive symptoms.

In contrast to endogenous depression, hopelessness depression is a subtype of depression explicitly proposed to result, at least in part, from reaction to an environmental stressor. According to the hopelessness theory of depression formulated by Abramson, Metalsky, and Alloy (1989), hopelessness depression is defined as a type of depression for which the distal contributory cause is negative attributional style combined with a negative life event. The diathesis of negative attributional style is the tendency to ascribe negative life events to causes that are stable and global. The symptoms of this subtype of depression include apathy, diminished energy, lack of initiation of voluntary responses, and pervasive feelings of hopelessness concerning life situations.

If lack of reactivity to one's environment is a cardinal feature of endogenous depression, it was reasoned that mood regulation factors should have a weaker association with endogenous depression than with other types of depression that are associated with reactivity to one's environment. The present study examines one aspect of personality, mood regulation skill, and assesses its relationship to symptoms of endogenous and hopelessness depression. If endogenous depression symptoms are characterized by lack of reactivity, and if the distinction between endogenous and other forms of depression is valid, then mood regulation skill should have a weaker relationship with endogenous depression than with hopelessness depression.

Mood regulation is defined as "all the extrinsic and intrinsic processes responsible for monitoring, evaluating, and modifying emotional reactions, especially their intensive and temporal features, to accomplish one's goals" (Thompson, 1994, p. 27). The capacity to engage in such processes can be defined as the "ability to identify, understand, express, and respond effectively to the full range of human emotions" (Rudd, Joiner, & Rajab, 2001, p. 37). Mood regulation is a broad topic and has been assessed in a variety of ways. A study by Garnefski, Kraaij, and Spinhoven (2001) examined the role of mood regulation in the relationship between negative life events, depression, and anxiety. The authors developed a questionnaire that assessed specific forms of coping that utilized cognitive strategies used by individuals following the experience of a negative life event. There was a negative relationship between adaptive forms of mood regulation and symptoms of anxiety and depression. Some studies have assessed the expectancies concerning mood regulation and their effect on symptoms of depression (Catanzaro, 1993; Catanzaro, Wasch, Kirsch, & Mearns, 2000). Other research has demonstrated that ability to regulate mood efficiently is associated with lower levels of symptom reporting (Salovey, Woolery, Stroud, & Epel, 2002), lower illness reports, and reduced frequency of visits to the doctor's office (Goldman, Kraemer, & Salovey, 1996; Salovey, 2001).

There is merit in examining the relationship between this personality variable and endogenous and hopelessness depression. According to Boyce, Gordon, Wilhelm, and Hickie (1990), among all the classifications of depression, "the differential relevance of personality factors has emerged most clearly" in this differentiation between endogenous and nonendogenous depression (Boyce et al., 1990, p. 1476). Past work has assessed other personality differences between those who suffer from endogenous depression and those whose depression is of the nonendogenous subtype. The study by Boyce et al. (1990) examined characteristics of personality using the Eysenck Personality Inventory (Eysenck & Eysenck, 1964), The Interpersonal Dependency Inventory (Hirschfeld, Klerman, & Gough, 1977), a locus of control measure (Craig, Franklin, & Andrews, 1984), and the Interpersonal Sensitivity Measure (Boyce, Hadzi-Pavlovic, & Parker, 1989). Those with non-endogenous depression displayed a more vulnerable personality style as measured by these scales. Zimmerman, Coryell, Pfohl, and Stangl (1986) found that those with endogenous depression had lower rates of personality disorders that were preexisting, again consistent with the view that personality factors may be more related to non-endogenous than to endogenous symptoms of depression.

There is empirical evidence demonstrating that both endogenous and hopelessness depression exist in adolescents, and their latent structures and symptomatology are similar to those experienced in adults. Ambrosini, Bennett, Cleland, and Haslam (2002) cited evidence that endogenous depression is a valid subtype among adolescents and consists of symptoms that include psychomotor retardation, anhedonia, and lack of reactivity. In addition, taxometric analyses confirmed that endogenous depression is most accurately and best described as a valid taxonic construct among adolescents. Voelz, Walker, Pettit, Joiner, and Wagner (2003) presented empirical evidence of the symptoms of hopelessness depression among psychiatric child and adolescent inpatients. The current study predicts that the association between mood regulation skill and endogenous depression should be lower in magnitude than the relationship between mood regulation skill and

hopelessness depression in two samples of adolescents. Participants completed items of the Beck Depression Inventory reflecting symptoms of endogenous and hopelessness depression, as well as a questionnaire assessing mood regulation skill.

STUDY 1

As an initial test of the hypothesis that the correlation of mood regulation skill with hopelessness depression symptoms would be higher than that with endogenous depression symptoms, zero-order and partial correlations between the three constructs were examined in a sample of high school students.

Method

Participants

Two hundred and fifty adolescents (120 boys, 130 girls; aged 14 to 19; $M = 14.7$, $SD = 0.63$) of a high school in Malaga, Spain voluntarily completed questionnaires in class during their weekly tutorial. The adolescents were told that they would be completing questionnaires that would be used to assess their emotions and feelings. The procedures for obtaining informed consent from the participants set forth by the school were followed.

Measures

*Trait Meta-Mood Scale (*TMMS; Salovel, Mayer, Goldman, Turvey, & Palfai, 1995*).* To evaluate mood regulation skill, adolescents completed the Trait Meta-Mood Scale (Salovey et al., 1995). The TMMS is a 48-item Likert-type scale. This scale addresses different aspects of intrapersonal emotional intelligence. In this study, given the emphasis on mood regulation, the focus was placed on the *Repair* subscale, which assesses the tendency of the participant to regulate unpleasant feelings (12 items, e.g. "No matter how badly I feel, I try to think about pleasant things"; "When I become upset I remind myself of all the pleasures in life"). Participants were required to rate the extent to which they agreed with each item on 5-point scale ranging from *strongly disagree* (1) to *strongly agree* (5). Previous studies have shown this scale to be reliable and satisfactory ($\alpha = .82$). This scale has been validated for use among adolescents and has demonstrated good psychometric properties, particularly discriminant validity (Moriarty, Stough, Tidmarsh, Eger, & Dennison, 2001). For this study, a Spanish adaptation was used ($\alpha = .76$; Fernández-Berrocal et al., 1998). Mood regulation skill was assessed by the score obtained in the *Repair* section of the questionnaire.

*Beck Depression Inventory (*BDI; Beck, Rush, Shaw, & Emery, 1979; Beck & Steer, 1987*).* The BDI is a 21-item self-report inventory. Each item is rated on a 0 to 3 scale; inventory scores thus may range from 0 to 63. The BDI is a reliable and well-validated measure of depressive symptomatology (Beck, Steer, & Garbin, 1988). In addition, the BDI has been demonstrated to be a valid screening tool for adolescent depression, with adequate psychometric properties for use in such a sample (Bennett et al., 1997). A Spanish version was used that has demonstrated good internal consistency, reliability, and validity in clinical samples ($\alpha = .82$, test–retest reliability between .65 and .72; Vazquez & Sanz, 1991).

Consistent with Haslam and Beck (1994), classical descriptions as well as *DSM-III-R* criteria were used in selecting the following five BDI items to tap the endogenous cluster: Items 4 (loss of satisfaction), 21 (loss of interest in sex), 16 (sleep disturbance), 18 (loss of appetite), and 19 (loss of weight). Scores on these five items were summed to obtain an endogenous cluster score. Haslam and Beck (1994) found that this cluster comprised a taxon. The lack of reactivity characteristic of endogenous depression is consistent with the BDI items used in this study to capture the endogenous symptom cluster. To assess the hopelessness depression syndrome, core hopelessness symptoms were emphasized, including Items 2 (hopelessness), 13 (difficulty making decisions), 15 (trouble getting started; work difficulty), 16 (sleep disturbance), and 17 (tiredness). Item 15 (trouble getting started; work difficulty) was added to the list of hopelessness symptoms, because it assesses a central aspect of hopelessness depression—retarded initiation of voluntary responses. Scores on these five items were summed to obtain a hopelessness cluster score. These five items are the same set that Joiner et al. (2001) used to empirically demonstrate that the hopelessness depression cluster is a distinct and discernible syndrome of depressive symptomatology. Structural equation modeling analyses were used on the aforementioned BDI items to obtain these results. These results were demonstrated among relatively large clinical and nonclinical samples. It is notable that Item 16 (sleep disturbance) is included on both the hopelessness and endogenous depression clusters, in keeping with the approach of Haslam and Beck (1994), Joiner (2001), as well as with seminal descriptions of each cluster. This overlap will only work against the hypothesis that mood regulation skill will relate more to one cluster than the other.

Table I. Intercorrelations, Partial Correlations, and Descriptive Statistics for All Variables

	1	2	3
1. Repair (mood regulation skill)	3.17 (0.52)	−.37*	−.24*
2. BDI hopelessness	−.37*	2.06 (2.01)	.52*
3. BDI endogenous	−.24*	.52*	2.38 (2.14)
4. BDI hopelessness controlling BDI endogenous	−.30*	—	
5. BDI endogenous controlling BDI hopelessness	−.05 ($p = .43$)	—	—

Note. $N = 250$. Means, with standard deviations in parentheses, are listed on the diagonal. BDI = Beck Depression Inventory. 95% Confidence Intervals are as follows for the following zero-order correlations: $r = -.37 = (-.26, -.47)$; $r = -.24 = (-.12, -.65)$; $r = .52 = (.42, .61)$.
*$p < .0001$.

Data Analytic Approach

The zero-order intercorrelation matrix was computed. Next, the correlation between mood regulation skill (Repair) and BDI hopelessness depression was compared to the correlation between mood regulation skill and BDI endogenous depression, using Steiger's technique for comparing correlated correlation coefficients (Steiger, 1980).[4] Finally, each correlation between mood regulation skill and a symptom cluster was computed, controlling for the other symptom cluster, consistent with the recommendations of Kendall and Ingram (1989) for constructing stringent specificity tests (cf. Joiner, Katz, & Lew, 1997 for a recent application).

Results and Discussion

Table I presents the intercorrelations, means, and standard deviations for all variables. Various features of Table I deserve emphasis. First, the zero-order correlation of mood regulation skill with BDI endogenous symptoms, which was statistically significant, was lower than the correlation of mood regulation skill with BDI hopelessness symptoms, which also was statistically significant. Second, as would be expected, the symptom clusters of endogenous and hopelessness depression were highly intercorrelated, a fact that makes for a stringent test of the present prediction that mood regulation will be more correlated to one cluster of depression symptoms than to another. The correlation between the hopelessness depression cluster and mood regulation skill, controlling for the endogenous cluster, was $r = -.3$, and was significant at the .01 level. In contrast, the correlation between the endogenous depression cluster and mood regulation skill, controlling for the hopelessness cluster, was $r = -.05$, and was not significant at the .01 level.

As noted, the correlation between mood regulation skill and endogenous depression symptoms was lower than the correlation between mood regulation skill and hopelessness depression. However, was the difference between these correlations *significant?* This question was answered by using a formula that tests the significance of the difference between dependent correlations (i.e., those that come from the same population; as cited by Cohen & Cohen, 1983; Steiger, 1980). The correlation between mood regulation skill and endogenous depression, $r = -.24$; 95% CI (−.12, −.65) was compared to the correlation between mood regulation skill and hopelessness depression, $r = -.37$, 95% CI (−.26, −.47). The difference between the two correlations was significant at the .01 level (two-tailed; $t = 5.61$, $df = 247$).

In spite of the high intercorrelation between the two depression symptom clusters, $r = 0.52$, 95% CI (.42, .61), the difference between their respective correlations with mood regulation skill was significant at the .01 level. The pattern of findings is consistent with the hypothesis that mood regulation skill is less associated with endogenous depression symptoms as compared to hopelessness depression symptoms.

Despite the predicted and significant difference between these correlations, the coefficient alphas for both clusters were low (i.e., ∼.50). As a result, the goal of the next study was to replicate the results of Study 1 and use BDI endogenous and hopelessness clusters with a higher degree of internal consistency reliability. The replication was conducted in a study with a larger sample.

STUDY 2

The goal of Study 2 was to replicate the findings of Study 1 on the interrelations of mood regulation skill, endogenous, and hopelessness depression symptoms, using

[4]In Study 2, the depression symptom clusters had a somewhat higher degree of internal consistency and reliability. The new symptom clusters for Study 2 were reapplied to Study 1, and the data were reanalyzed with the new symptom clusters. Results were generally similar.

a larger sample and similar methodology. The BDI hopelessness and endogenous clusters were changed from the first study to obtain clusters with higher reliabilities.

Participants

Three hundred and ninety-six adolescents (210 boys, 186 girls; aged 13 to 20; $M = 15.8$ $SD = 1.41$) of two different public high schools in Malaga, Spain completed the tests voluntarily, in class during their weekly tutorial. The adolescents were told that they would be completing questionnaires that would be used to assess their emotions and feelings. The procedures for obtaining informed consent from the participants set forth by the school were followed.

Measures

Trait Meta-Mood Scale (TMMS; Salovey et al., 1995*)*

As described in Study 1, adolescents completed the Trait Meta-Mood Scale (Salovey et al., 1995). Mood regulation skill was assessed by the score obtained on the *Repair* section of the questionnaire.

Beck Depression Inventory (BDI; Beck et al., 1979; Beck & Steer, 1987*)*

As in Study 1, five BDI items were used to tap the endogenous depression cluster. The same items were used from Study 1; however, Item 19 (loss of weight) was replaced with Item 17 (tiredness). Therefore, the BDI items used in this Study 2 for the endogenous depression cluster were Items 4 (loss of satisfaction), 21 (loss of interest in sex), 16 (sleep disturbance), 18 (loss of appetite), and 17 (tiredness). For the hopelessness cluster in Study 2, two items from the hopelessness cluster (Items 2 and 15) were kept from the original cluster. All the other items were removed, and two more items, 12 (loss of interest in others) and 8 (self-blame) were added to the hopelessness cluster. Therefore, the hopelessness cluster for Study 2 were comprised of the following items: 2 (hopelessness), 8 (self blame), 12 (loss of interest in others), and 15 (trouble getting started; work difficulty). The coefficient alphas for the respective clusters were as follows: .608 for the endogenous symptom cluster, and .573 for the hopelessness depression cluster.

Data Analytic Approach

As in Study 1, the zero-order intercorrelation matrix, as well as partial correlations, were computed for the entire sample.

Results and Discussion

Replication of Study 1 Findings

Table II presents the intercorrelations and partial correlations for the variables. The correlation between the hopelessness depression cluster and mood regulation skill, controlling for the endogenous cluster, was $r = -.29$, and was significant at the .01 level. In contrast, the correlation between the endogenous depression cluster and mood regulation skill, controlling for the hopelessness cluster, was $r = -.09$, and was nonsignificant. The correlation between the endogenous and hopelessness symptom clusters was $r = .52$, 95% CI (.42, .61).

The zero-order correlation of mood regulation skill (repair) with BDI endogenous symptoms, $r = -.29$, 95% CI ($-.20$, $-.38$) was slightly lower than the zero-order correlation of mood regulation skill with the BDI hopelessness symptoms, $r = -.40$, 95% CI ($-.31$, $-.48$).

Table II. Intercorrelations, Partial Correlations, and Descriptive Statistics for All Variables

	1	2	3
1. Repair (mood regulation skill)	3.20 (0.52)	$-.40^*$	$-.29^*$
2. BDI hopelessness	$-.40^*$	1.87 (2.04)	$.57^*$
3. BDI endogenous	$-.29^*$	$.57^*$	1.98 (2.02)
4. BDI hopelessness controlling BDI endogenous	$-.29^*$	—	—
5. BDI endogenous controlling BDI hopelessness	$-.09$ ($p = .08$)	—	—

Note. $N = 396$. Means, with standard deviations in parentheses, are listed on the diagonal. BDI = Beck Depression Inventory. 95% Confidence Intervals are as follows for the following zero-order correlations: $r = -.40 = (-.31, -.48)$; $r = -.29 = (-.20, -.38)$; $r = .57 = (.50, .63)$.
$^*p < .0001$.

Importantly, this difference between correlations achieved statistical significance at the .01 level (two-tailed; $t = 2.73$, $df = 393$) using Steiger's formula (Steiger, 1980), as described in Study 1.[4] As in Study 1, despite the high degree of correlation between the depression symptom clusters, the difference between their respective correlations with mood regulation skill was as predicted, and significant at the .01 level.

GENERAL DISCUSSION

To examine the validity of the distinction between endogenous and nonendogenous depression, these studies assessed the relationship between mood regulation skill, hopelessness, and endogenous depression. Depressive symptoms that are endogenous in nature may be affected less by mood regulation skill than symptoms of the hopelessness depression cluster, which is, at least in part, a non-endogenous symptom dimension. The current studies supported this prediction. The correlation between mood regulation skill and endogenous depression was smaller in magnitude than the correlation between mood regulation skill and hopelessness depression. In addition, the difference between these correlations achieved statistical significance in two separate samples.

This finding further supports the validity of the endogenous subtype of depression, especially in light of the high degree of symptom overlap between endogenous and hopelessness depression, which makes the differential relation of mood regulation to endogenous versus hopelessness depression a more risky prediction. Regarding this overlap, several studies have shown that prototypical hopelessness depression indicators (e.g., feelings of helplessness and powerlessness) load relatively strongly on an endogenous symptom factor (Lewinsohn et al., 1977). Hopelessness depression also includes some endogenous depression symptoms. Given the high degree of similarity between the two symptom clusters, one might question whether hopelessness depression and endogenous depression are two distinct forms of depression. These studies attempt to answer this question, at least in part, by inferring from theory (Abramson et al., 1989) that one cluster might be more reactive than another, and by framing relation to mood regulation as a kind of signal of "reactivity." Results demonstrated that the theoretically more reactive cluster (hopelessness depression) was more associated with mood regulation than was the theoretically less reactive cluster (endogenous symptoms). Taken together with past work from neurobiological and descriptive psychiatric perspectives (Parker et al., 1994), the results are supportive of the endogenous depression symptom cluster as a separate and cohesive syndrome, distinguishable from other aspects of depressive symptomatology.

Mood regulation deficits appear to be associated with various clinical problems, including suicidal ideation, parasuicidal behavior, diagnosis of personality disorder, alcohol abuse, deficits in problem-solving skills, and the development of hopeless cognitions (Catanzaro, 2000). According to Westen, Muderrisoglu, Fowler, Shedler, and Koren (1997), all *DSM-IV* personality disorder diagnoses include some type of deficit in mood regulation. A possible explanation for this is that the behavior associated with such a diagnosis has developed, in part, as a mechanism to regulate moods that cannot be done so in an adaptive way (Linehan, 1993; Westen, 1994). An interesting avenue for future theory development is the idea that hopelessness may arise from the belief that one is incapable of effectively regulating negative mood.

Findings warrant further research concerning their relationship to clinical treatment issues. Various studies demonstrate that those with endogenous depression have a different course, prognosis, and response to antidepressant treatment (Boyce et al., 1990; Peselow et al., 1992). The results suggest that mood regulation may be worth considering in the assessment and treatment of depression. Patients with clear endogenous symptom profiles may benefit less from psychotherapeutics focused on mood regulation, as compared to patients with clearly nonendogenous symptom profiles.

Some considerations and cautions are relevant to interpretation of study results. Although Joiner (2001) and Haslam and Beck (1994) provided support that the BDI indicators for endogenous and hopelessness depression symptoms are valid indicators, the present work was not meant to reify these items as the sole indicators of the respective types of depression. Indeed, present findings show that their reliabilities can be low. Future work would benefit from an assessment of symptoms that incorporated additional measures. Examples include measures better suited to assess endogenous depression symptoms (e.g. Inventory of Depressive Symptomatology; Rush, Gullion, Basco, & Jarrett, 1996) and symptoms of hopelessness depression, such as the Hopelessness Depression Symptom Questionnaire (Metalsky & Joiner, 1997). Other items used to assess depressive symptoms among adolescents include the Child Depression Inventory (Kovacs, 1980/1981) and the K-SADS-L diagnostic interview (Puig-Antich, Chambers, & Klein, 1993).

The issue of low reliability could also be considered in interpreting results. However, because reliability coefficients were roughly similar for the measures of endogenous and hopelessness symptoms, reliability issues cannot

explain their *differential* relations to mood regulation—differential relations that were highly similar across two separate studies. Additionally, current results were found within a nonclinical sample of adolescents, and applicability to nosologic depression is indeterminate. Study participants were, on average, in their teens, a time of life that precedes the average age of onset of forms of major depression, including endogenous depression (e.g., Smith & Weissman, 1992). On the other hand, age of depression onset is dropping (e.g., Smith & Weissman, 1992). Furthermore, the age of onset of hopelessness depression may be similar to that for endogenous depression, and if this is so, the average age of study participants would not explain why mood regulation skill was more correlated with one than with another symptom constellation. Finally, results of this study were obtained from Spanish high school students, and results may not generalize to other samples.

Despite the limitations discussed above, results of the present work provide support for the validity of the distinction between endogenous and nonendogenous depression, a distinction that has useful theoretical and clinical implications. The finding that mood regulation skill correlated with endogenous depression to a significantly lesser degree than with the highly associated hopelessness symptom cluster adds to evidence that endogenous depression symptoms may be separable from other forms of depression.

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