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ORIGINAL ARTICLE

Rumination and Vegetative Symptoms: A Test of the Dual Vulnerability Model of Seasonal Depression

Michael A. Young · Annemarie Reardon · Omar Azam

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Abstract The Dual Vulnerability Model of seasonal affective disorder proposes that the cognitive-affective symptoms of seasonal depression are the result of an interaction of a diathesis of cognitive vulnerability to depression and the stressor of seasonal vegetative change. Two studies examined this hypothesis employing a within-subject design with daily data on vegetative and cognitive-affective depressive symptoms. Study 1 included a subclinical sample and a trait measure of ruminative response style. Study 2 included a clinical sample and reports of actual ruminative thoughts and behaviors in response to fatigue. Results of mixed linear model analyses in both studies supported the hypothesis that rumination moderates the relationship between the vegetative symptoms and the cognitive-affective symptoms of seasonal depression. The extension of the model to other subtypes of depression is considered.

Keywords Rumination · Seasonal affective disorder · Dual vulnerability model

M. A. Young (✉) · A. Reardon · O. Azam
Institute of Psychology, Illinois Institute of Technology, 3105 S. Street, Room 252, Chicago, IL 60616-3793, USA
e-mail: youngm@iit.edu

Present Address:
A. Reardon
National Center for PTSD, Boston, MA, USA

Introduction

Seasonal Affective Disorder (SAD) is a pattern of recurrent major depressive episodes that begin in the fall and winter and remit in the spring (Rosenthal et al. 1984). Symptoms typically include vegetative changes of fatigue, increased appetite and weight, and increased sleep, as well as the other cognitive and affective symptoms of depression. Although SAD is a present–absent classification (American Psychiatric Association 2000), seasonal variation in physiology, mood, and behavior (“seasonality”) is a phenomenon that varies in magnitude across individuals in the general population (Hardin et al. 1991; Kasper et al. 1989; Murray et al. 2001).

The Dual Vulnerability Model of seasonal depression (Young et al. 1991) proposes that the symptomatology is the result of two distinct vulnerabilities. The first is a physiological vulnerability to experience large seasonal fluctuations in vegetative functions (sleep, appetite, energy) in response to seasonal changes in the physical environment (Young et al. 1997). The second is a psychological vulnerability to develop mood and cognitive symptoms of depression in response to the experience of seasonal vegetative symptoms. Thus, the psychological vulnerability can be considered a depressive diathesis that is triggered by the stressor of vegetative change. The model was originally proposed in response to findings that episodes of SAD begin with vegetative changes and that other symptoms occur later, with their onsets spread across the episode (Young et al. 1991). This pattern of symptom onset has been confirmed with prospective data (McCarthy et al. 2002).

Several lines of evidence support the Dual Vulnerability Model. Both between-subject (Madden et al. 1996) and within-subject (Schmitt and Young 2000) factor analyses have supported the distinction of vegetative and cognitive-affective symptom clusters in diagnosed SAD patients. In addition, the model suggests that a seasonal vegetative syndrome exists, which may or may not be accompanied by the cognitive and affective symptoms of depression (Young et al. 1991). White and Terman (2004) examined symptom reports from individuals completing a questionnaire on a public access website that provides information about SAD and related topics (www.cet.org). Consistent with the model, they found a subset of respondents who reported seasonal changes in vegetative symptoms only.

In recent years, the role of psychological factors in SAD has received increasing attention, with researchers investigating vulnerabilities to depression that are established in the nonseasonal depression literature. A number of studies have found an association between seasonality and neuroticism (Blodgett et al. 2001; Jang et al. 1998; Kane and Lowis 1999; Murray et al. 1995). Other studies have found that ruminative response style (Nolen-Hoeksema 1991)—the tendency to ruminate about one's symptoms and their possible causes, meanings and consequences—predicts the severity of winter depression (Young and Azam 2003; Rohan et al. 2003). Most recently, Enggasser and Young (2007) found that a ruminative response style, dysfunctional attitudes, and an internal causal attributional style all prospectively predicted the winter severity of cognitive and affective symptoms in individuals reporting a history of seasonal vegetative changes.

Although these results are promising, the Dual Vulnerability Model hypothesizes a more specific relationship than a simple association between cognitive vulnerability and symptomatology, i.e., that depression vulnerability factors *interact* with SAD-specific vegetative symptoms to generate the cognitive and affective symptoms of seasonal depression. Thus, the magnitude of the cognitive-affective response to vegetative symptoms should be moderated by the depressive vulnerabilities. In addition, although the Dual Vulnerability Model was developed based on the sequence of symptom onset across the weeks of episode development, we believe that this phenomenon also can be observed on a daily basis in people with seasonality—that one's mood and thoughts during a given day are responses to the severity of one's vegetative symptoms on that day, moderated by cognitive vulnerabilities to depression. If correct, this effect should be observable during periods in which there is sufficient variability in vegetative symptom severity, which includes both onset (fall) and offset (spring) periods. It is also worth noting that, according to Response Style Theory, (Nolen-Hoeksema 1991), the effects of rumination occur whenever there are symptoms about which to ruminate and are not limited to periods of diagnosable disorder.

We report here on two studies that examined the hypothesis that cognitive vulnerabilities moderate the relationship between vegetative and cognitive-affective symptoms in terms of daily symptom levels across time within individuals. Both studies examined vegetative and cognitive-affective symptoms assessed on a daily basis. They differed in the number of days over which participants were followed, the specific symptoms assessed, and whether rumination was operationalized as a trait tendency or as a manifest daily behavior. In Study 1, college students with a self-described history of seasonality completed measures of trait neuroticism and trait ruminative response style and reported their vegetative and cognitive-affective symptoms daily across approximately 2 months. In Study 2, individuals diagnosed with SAD reported for 14 consecutive days their levels of fatigue and depressed mood, and their actual ruminative responses to fatigue and to mood. Thus, in Study 1 we examined how cognitive vulnerability traits moderated the relationship between vegetative symptoms and cognitive-affective symptoms; in Study 2 we examined how time-varying vulnerability behaviors moderated this relationship.

Data Analysis Approach

Data were analyzed with linear mixed models (Pinheiro and Bates 2004), also know as hierarchical linear modeling (Bryk and Raudenbusch 1992), using S-Plus 6.02 for Windows (Insightful Corporation

2003). These models represented our hypotheses regarding (a) the relationship over time within each person between vegetative symptoms and cognitive-affective symptoms, (b) trait moderators of this relationship, which vary only from person to person (Study 1), and (c) time-varying moderators of this relationship, which vary across time within each person (Study 2). Linear mixed models also have the advantage of being applicable to data sets with missing data and different numbers of times points per participant.

The specific models in our studies consisted of variants of the following model.

$$C_{it} = B_{0i} + B_{1i}V_{it} + [B_2M_{1i} + B_3V_{it}M_{1i}] + [B_4M_{2it} + B_5V_{it}M_{2it}]$$

The outcome C_{it} is the cognitive-affective symptom score of participant i at time t. B_{0i} is the intercept for participant i, V_{it} is the vegetative symptom score of participant i at time t, and B_{1i} is the vegetative regression coefficient for participant i.[1] The third term represents the interaction of vegetative symptoms with a trait moderator M_1, which varies across the i participants. The fourth term represents the interaction with a time-varying moderator M_2, which varies across the i participants and t times. As in standard multiple regression, the significance of the interaction (the moderation) is assessed by the significance of the second, product part of these expressions, i.e., B_3 and B_5. As will be seen, Study 1 included two trait moderators and Study 2 included one time-varying moderator. All models included linear and quadratic terms for time (in days) and a first-order autoregressive error term.[2]

[1] The effect of vegetative symptoms was modeled as a random effect, consistent with our hypothesis that their impact on cognitive-affective symptoms varies from person to person. Moderating variables terms were modeled as fixed effects. Maximum likelihood estimation was used so that the fits of the models with different fixed effects could be compared. The statistical significance of fixed effects was assessed by a *t*-test based on the estimate and its standard error. The statistical significance of the standard deviation of random effect variables was assessed by comparing the fit of the model to the model with the effect fixed (Pinheiro and Bates 2004).

[2] In both studies, all dependent and independent variables were examined for being approximately normally distributed. This was the case, except for the positively skewed cognitive-affective variable in study 1, which, therefore, was log-transformed. Time-varying independent variables were person

Study 1

We hypothesized that (1) within individuals across days, the severity of cognitive-affective symptoms is associated with the severity of vegetative symptoms; (2) the magnitude of the association between cognitive-affective symptoms and vegetative symptoms varies across individuals; and, most important, (3) the magnitude of this association is moderated by trait levels of neuroticism and ruminative response style.

Methods

Participants

Participants were recruited for a study of daily thoughts and feelings from undergraduate psychology classes at an urban Midwestern university. Of 45 original participants, 15 reported a history of regular winter seasonal symptoms on the Seasonal Pattern Assessment Questionnaire (Rosenthal et al. 1987), a commonly used self-report of seasonality with satisfactory psychometric properties (Young et al. 2003), and are included in the analysis. Four participants were female and 11 were male, a distribution similar to that of the university. Age ranged from 18 to 24 years. Five participants were White, Not Hispanic; 4 were East/South Asian; 2 were Black, Not Hispanic; 1 was Native American; and 3 were Other/Missing. None of the participants were taking psychotropic medications or using light treatment at the time of the study; two reported currently seeing mental health professionals. The mean (SD) score on

Footnote 2 continued
centered; those that varied only across persons were grand mean centered. In both studies visual inspection of graphs of the dependent cognitive-affective variable suggested that linear and quadratic general trends over time, both varying from person to person (i.e., as random effects), should be accounted for. In both studies, models with these terms significantly improved model fit. Next, a first-order autoregressive error structure was added. In both studies this addition also significantly improved model fit (autocorrelation ranged from .224 to .296 across all the models reported). Consequently, all subsequent models representing our hypotheses included these effects and served as the base models for assessing further improvements in model fit. For simplicity of presentation, these terms are not shown in the equation above or reported further in the Results. Details are available from the authors.

the Center for Epidemiological Studies Depression scale (Radloff 1977), completed at the beginning of the study in late February and early March, was 15.8 (7.3).

Measures and Procedures

On the first day of the study, participants completed two measures of vulnerability to depression. Neuroticism (N) was measured with the short version of the Eysenck Personality Questionnaire (Eysenck and Eysenck 1964). This scale consists of 12 true-false items, 6 of which assess N. Ruminative response style (RRS) was assessed with the Response Style Questionnaire (Nolen-Hoeksema and Morrow 1991), the most commonly used scale for this purpose. The RRS subscale contains 22 cognitive and behavioral responses to depressed mood that focus on the self and the experience, causes, and consequences of depressive symptoms. For each item, respondents indicate the frequency with which they typically engage in the thought or behavioral response on a 5-point Likert scale ranging from 0 (“almost never”) to 5 (“almost always”). The RRS has demonstrated good internal consistency ($\alpha = .90$) and five-month test–retest reliability ($r = .80$; Nolen-Hoeksema et al. 1994). The internal consistencies of the RRS in this study was $\alpha = .79$.

After completing the initial packet of questionnaires, participants were given a supply of daily rating forms, which they completed each night before going to bed. Forms were returned in a sealed envelope on a weekly basis. Participants were instructed not to look at previous days’ ratings and that if they did not complete a questionnaire on the assigned day to just leave it blank. Participants received course credit and $20 upon completion of the data collection. In addition, a raffle was held every 2 weeks at which time the winner received $50.

The daily ratings consisted of 24 one hundred mm visual analogue scales (VAS) of depression-related phenomena. The VAS was a practical method for data collected daily for approximately 9 weeks. Participants completed the VAS each evening beginning between February 22 and March 5 and ending between April 21 and May 9. This resulted in 56–70 rating days per participant (mean, 65.3) and a total of 980 ratings administered. Data were missing for 68 ratings (6.9%), with participants missing a mean of 4.5 reports. This yielded data for a mean of 60.7 reports per person (range, 42–68). A within-subject factor analysis of these data (see Appendix) indicated the energy-motivation and cognitive-affective factors that were used in the analyses reported here. Energy-motivation items consisted of sluggish, sociable, interest in usual activities, energetic, able to concentrate, slow, tired, and confident (some items reverse scored). Cognitive-affective items consisted of discouraged, withdrawn, confused, worthless, sad, guilty, hopeless, and tearful.

Results

We first examined the model including energy-motivation symptoms predicting cognitive-affective symptoms, but with no moderators (hypotheses 1 and 2). Results supported hypothesis 1 that the severity of cognitive-affective symptoms and energy-motivation symptoms are related within subjects across time[3] (B = 3.32, SE = .34, $P < .001$). Day-to-day variability in energy-motivation symptoms accounted for 30.4% of the within subject variability in cognitive-affective symptoms. In addition, the coefficients varied significantly across individuals (SD = .97, $P < .001$), supporting hypothesis 2. This model provided a significant improvement in fit compared to a model without energy-motivation as a predictor ($\chi^2 = 306.6$, $P < .001$). When RRS and N were added to the model as moderators, consistent with hypothesis 3, the RRS interaction term was significant, indicating that higher levels of RRS were associated with a stronger relationship between cognitive-affective symptoms and energy-motivation symptoms (B = .08, SE = .029, $P = .005$). Contrary to our hypothesis, the N interaction term was not significant (B = .20, SE = .22, $P = .37$). With N removed from the model, RRS alone accounted for 56.1% of the between subject variance in the magnitude of the relationship between energy-motivation symptoms and cognitive affective symptoms.

In order to provide some evidence regarding the direction of causality of energy-motivation and cognitive-affective symptoms, we reversed the direction of the model so that cognitive-affective symptoms predicted energy-motivation symptoms

[3] For ease of presentation, in Study 1 coefficients and standard errors have been multiplied by 1000.

and this effect was moderated by RRS. However, the RRS interaction term was not significant (B = .802, SE = 1.34, P = .548), supporting the original model in which rumination moderates an effect of energy-motivation symptoms on cognitive-affective symptoms.

Study 2

The primary focus of Study 1 (hypothesis 3) was whether individual differences in the magnitude of the relationship between cognitive-affective symptoms and energy-motivation symptoms were related to individual differences in trait levels of RRS and N. In contrast, Study 2 focused on whether daily levels of mood were related to the interaction between the level of fatigue on that day and the ruminative response to fatigue on that day. We hypothesized that within SAD patients across time (1) daily mood level is predicted by daily fatigue level and, most important, (2) this association is moderated by daily ruminative responses to fatigue.

Methods

Participants and Procedures

Participants were community members who had been diagnosed with SAD in an earlier randomized clinical trial of light treatment (Eastman et al. 1998). At that time, all participants were interviewed with the Schedule for Affective Disorders and Schizophrenia (Endicott and Spitzer 1978) and met the SAD diagnostic criteria of Rosenthal et al. (1984): DSM-IIIR criteria for recurrent major depressive disorder, depressive episodes in at least two consecutive winters, no seasonally varying psychosocial factors that could account for the recurrent depressions, and regularly occurring non-depressed periods in the spring and summer. Participants met criteria for no other current Axis I disorders. Additional inclusion criteria were the presence of the typical SAD symptoms of increased sleep and appetite or weight and a score 21 or more on the first 24 items of the Structured Interview Guide for the Hamilton Depression Rating Scale, SAD Version (SIGHSAD) (Williams et al. 1992, i.e., the original 17-item Hamilton Depression Scale plus 7 atypical symptom items).

For the current study, our intention was to assess these diagnosed individuals in a new period of winter symptomatology. Symptomatology in a future winter would be likely if a person's seasonal affective disorder had continued and they had been symptomatic the previous winter. Therefore, participants were recontacted by telephone 2–7 years after their original participation (mean, 4.1; SD, 1.6) and received an unstructured interview by a clinical psychology Ph.D. student (OA). Twenty-eight individuals were successfully contacted, reported this continuing symptomatology, and agreed to participate in the new study. Of these twenty-eight, 79% were female, 86% were Caucasian, and their mean (SD) age was 44.1 (9.9) years. The median age of onset of SAD was 18 years (range, 6–42) and the median time between onset and intake into the current study was 26 years (range, 8–43).

Questionnaires were completed on 14 consecutive nights. Participants were instructed to place each daily packet in an envelope, not to look at forms from previous days, and that if they missed completing a questionnaire on the assigned day to leave that day's ratings blank. All forms were returned by mail at the end of the 14 days. Starting dates ranged from September 2 to November 17 (median, September 29). The mean (SD) total score on the Beck Depression Inventory (BDI; Beck et al. 1961) completed at the end of the two-week data collection was 14.1 (10.0). For a modified BDI with four items added so that increases as well as decreases in vegetative functions could be assessed the mean (SD) was 16.7 (11.6).

Measures

All data were collected on a modified Daily Emotion Report (DER; Nolen-Hoeksema et al. 1993), a self-report of actual daily ruminative responses and distracting responses (not reported on here) parallel to the trait assessment on the Response Styles Questionnaire used in Study 1. The participant first rated the severity of sad or depressed mood on that day on a scale of 0–10. Following a non-zero response, the participant endorsed as applicable 16 ruminative thoughts and behaviors in which he or she had engaged that day in response to his or her mood. If mood was rated zero, ruminative responses to mood were assumed to be zero. The daily rumination-to-mood score is the total of the items checked and thus each has a potential range of 0–16, with higher scores indicating greater use of rumination.

A second version of the DER was created for this study in order to assess responses to fatigue. This form was the same as the original DER except that the participant rated the daily experience of, and responses to, "fatigue or low energy" instead of "sad or depressed mood." Responses to fatigue were the same as those for mood, with a few small wording modifications. The questionnaire is available from the authors. The internal consistencies of the rumination-to-mood and rumination-to-fatigue scales were both $\alpha = .80$.

In summary, the variables analyzed were low mood, ruminative response to low mood, fatigue, and ruminative response to fatigue. Data were collected for 28 participants assessed on 14 days. On one day each, two participants failed to complete ratings. Therefore, there were 390 days of ratings.

Results

We first examined the model including fatigue as a predictor of mood, but with no moderators (hypotheses 1). Consistent with the hypothesis, low mood was associated with fatigue (B = 0.247, SE = 0.083, $P = .003$). This model provided a significant improvement in fit ($\chi^2 = 90.2$, $P < .001$) compared to a model without fatigue as a predictor, and fatigue accounted for 24.4% of the within person variance in mood. When the rumination-to-fatigue moderator was added to the model, consistent with hypothesis 2 the interaction of fatigue and rumination-to-fatigue was significant (B = 0.086, SE = 0.027, $P = .001$), indicating that the effect of fatigue on mood depended on the amount of rumination-to-fatigue. In order to interpret the interaction, simple slopes were calculated based on model parameter values (Cohen et al. 2003). As can be seen in Fig. 1, in general as fatigue increased, low mood increased. However, the magnitude of this effect depended on the degree of rumination to fatigue: on days in which there was more rumination, the mood response is greater. In fact, if the amount of rumination was as low as two SDs below the mean, increasing fatigue was *not* associated with increasing low mood.

In order to enhance the interpretation of these findings, we conducted two further analyses. First, in

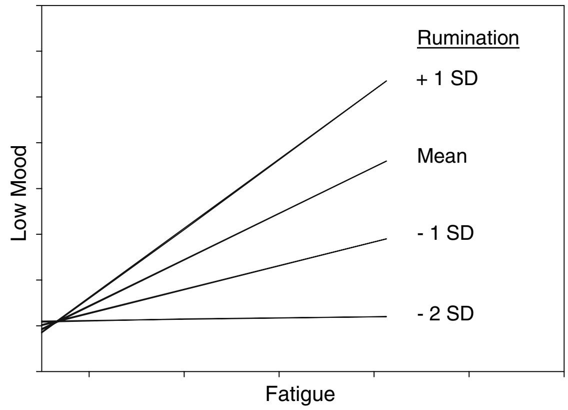

Fig. 1 Regression of low mood on fatigue at 3 levels of rumination-to-fatigue

order to test whether the moderation was specific to ruminations about *fatigue*, we hypothesized that the effect of fatigue on mood was *not* moderated by rumination about *mood*. Rumination-to-mood and its interaction with fatigue were entered into the regression in place of rumination-to-fatigue and its interaction with fatigue. As hypothesized, this interaction was not significant (B = .008, SE = .021, $P = .704$), indicating that rumination-to-fatigue but not rumination-to-mood moderated the effect of fatigue on mood. (As might be expected, rumination-to-mood did have a significant direct effect on mood; this also suggests that sufficient rumination-to-mood occurred to potentially interact with fatigue.) As a point of information, the within-subject across-time correlation of rumination-to-fatigue and rumination-to-mood[4] was .42. This indicates that although there was some consistency in rumination across content, these two variables were far from being identical.

Finally, in order to provide some evidence regarding the direction of causality of fatigue and mood, we reversed the direction of the model so that mood predicted fatigue and this effect was moderated by rumination-to-mood. The interaction of mood and rumination-to-mood was not significant (B = .032,

[4] Models were fit regressing rumination-to-mood on rumination-to-fatigue and vice versa, including, as before, linear and quadratic time effects and autocorrelated errors. The within-subject correlations were computed based on the reduction in variance from models without the independent variable. The value reported is the mean of the two values obtained, .4306 and .4023.

SE = .028, P = .248), supporting the original model in which rumination moderates an effect of fatigue on mood.

Discussion

Consistent with the Dual Vulnerability Model, both studies found that cognitive vulnerabilities to depression interacted with vegetative symptoms to predict the cognitive and affective symptoms of seasonal depression. Thus, both studies found support for rumination moderating the effect of vegetative symptoms on cognitive-affective symptoms. In addition, the results indicate that these processes occur on a daily basis, extending the original theory. In Study 1 this was the case in a non-clinical sample of college students who described typically experiencing seasonal symptoms. In Study 2 this was the case in a clinical sample of individuals who had been diagnosed with SAD during a prior episode.

Given the methodological differences between the two studies, it is significant that their results converged. Study 2 examined the more specific constructs of "fatigue" and "low mood" assessed with single items, whereas Study 1 employed the broader vegetative and cognitive-affective constructs, each assessed with multiple items. The Study 1 items that most closely corresponded to "fatigue" and "low mood" in Study 2 were "energetic" and "sad", which had the highest loadings on the vegetative and cognitive-affective factors (.76 and .78, respectively; see Appendix). Thus, the symptom constructs in the two studies are probably largely overlapping. It is worth noting that both the commonly used RSQ and the measure of rumination in Study 2 ask about the occurrence of ruminative thoughts and behaviors and not their intensity or duration, the assessment of which would probably be useful in future research.

Another important difference was that Study 1 was based on a trait measure of typical ruminative responses to low mood. In contrast, Study 2 provided a more specific test of the model by assessing reports of actual daily ruminative thoughts and behaviors, and in response to the specific vegetative symptom fatigue. Study 2 found that fatigue was associated with low mood only when substantial rumination to fatigue occurred. In addition, it was specifically ruminations about fatigue, and not ruminations about mood, which functioned as a moderator. Thus, not only did rumination moderate the impact of fatigue on mood, it appeared to be an essential ingredient.

Furthermore, the size of the observed effects was substantial. In Study 1, vegetative symptom severity accounted for almost one-third (30.4%) of the daily variability in cognitive-affective symptom severity and rumination accounted for over half (56.1%) of the individual variability in the strength of this relationship. In Study 2, severity of fatigue, as moderated by rumination about fatigue, accounted for more than one-quarter (27.8%) of the daily variability in mood. Overall, these findings support the idea that the cognitive-affective symptomatology of SAD is the result of vegetative symptoms and depressive vulnerabilities that interact in a diathesis-stress manner.

Contrary to expectations, the relationship between vegetative symptoms and cognitive-affective symptoms was not moderated by neuroticism (Study 1). Several studies have found that the relationship between neuroticism and depressive symptoms is mediated by rumination (Roberts et al. 1998; Spasojevic and Alloy 2001; Blodgett et al. 2001). However, this could not account for our results because we failed to find a neuroticism effect even excluding rumination. It is possible that failure to find this effect was due to a lack of power because of the small sample size.

It is noteworthy that our studies examined the sources of day-to-day changes in cognition and affect *within individuals*, in contrast to many studies of symptom–trait relationships across individuals at one point in time or from one to a second point in time. Our findings suggest that treatments for SAD and subclinical seasonality might effectively target psychological vulnerabilities, such as ruminative response style, to ameliorate the impact of seasonal changes in vegetative symptoms. Results of a study of cognitive behavior therapy tailored to SAD (Rohan et al. 2007) indicate that this may be the case.

The designs of our studies had a number of limitations. First, the number of participants was small, although the number of time points per participant was relatively large in Study 1. Despite the small samples, all but one of the hypothesized relationships was statistically significant. Nevertheless, our confidence in these encouraging results will be enhanced by replications with larger samples.

Second, the studies were correlational in nature and so provide limited evidence for the causal relationships proposed in the Dual Vulnerability Model. Some evidence suggestive of causality going from fatigue to mood comes from the finding in both studies that when the entire model was reversed, ruminating about mood did not moderate the effect of mood in predicting fatigue. In addition, we cannot rule out the possibility that variables not included in the study play a causal role. However, in Study 2 we were able to exclude rumination-to-mood as such a third variable. More conclusive evidence regarding causality could be provided by experimental studies.

Finally, since our study did not include non-seasonally depressed or never-depressed groups, it is unclear whether the phenomenon of cognitive-affective symptoms being a response to vegetative symptoms moderated by cognitive vulnerabilities is limited to individuals with seasonal depressive symptoms. Such a Dual Vulnerability Model also could apply to other kinds of depression that are primarily "biologically-driven", as SAD is believed to be. For example, perhaps in endogenous-melancholic depression core physiological symptoms are responded to with the more psychological depressive symptoms and this response also is moderated by cognitive processes. Furthermore, it may be possible to use this approach to identify different depressive subtypes. Current approaches mostly rely on defining subtypes based on what symptoms are present. But a more beneficial approach maybe to posit a structure in which certain "primary" symptoms serve as stressors which trigger diatheses (moderating vulnerabilities) for "secondary" symptoms. For example the hopelessness subtype of depression (Abramson et al. 1989), proposes that there are core cognitive symptoms, from which vegetative symptoms emerge; vulnerabilities that moderate the emergence of vegetative symptoms are not part of the current hopelessness theory model. Other subtypes also might be defined based on what are the primary symptoms, what are the secondary symptoms, and what are the moderators that link them. Models like this can be tested empirically with studies such those presented here and with longitudinal studies in which the time lag between primary and secondary symptoms is appropriate to the causal processes hypothesized. It is also worth noting a subtle but important difference between the Dual Vulnerability approach and the traditional Response Style Theory (Nolen-Hoeksema 1991). In the latter, rumination as a moderator addresses, the question of who, after experiencing initial depressive symptoms, goes on to a period of sustained and more severe depression (Teasdale 1985). In the Dual Vulnerability approach moderators connect particular primary and secondary symptom clusters, addressing the question of who, after experiencing initial primary symptoms, goes on to expand their symptomatology, potentially to a full depressive syndrome. Exploring the value of Dual Vulnerability-type models is a valuable area for future research.

Appendix: Factor Analysis of Visual Analogue Scale (VAS) Items

Data for the factor analysis came from the 45 original participants in Study 1. Participants completed visual analogue scales (VAS; items are in Table A1) on 42–70 days ($M = 51.6$, $SD = 7.7$). This sample's demographic characteristics were similar to those of the Study 1 subsample. First, a covariance matrix was calculated for each participant; then these matrices were averaged to yield a single covariance matrix representing the typical within-subject covariability. An exploratory principle components factor analysis with varimax rotation was conducted on the covariance matrix to assess which symptoms clustered together as they changed across time.

Between-subject factor analyses of SAD symptom data have found vegetative and cognitive-affective factors (Madden et al. 1996). In a within-subject factor analysis of data from 10 SAD patients with the same VAS variables in this study, Young and Schmitt (2000) found two similar factors, labeled energy-motivation (a subset of vegetative) and cognitive-affective, plus a doublet of the two appetite items that neither loaded on these factors nor generated a clear separate factor. Based on these results, we examined a two-factor model and excluded the two appetite items. (Models including the two appetite variables produced estimation and interpretation problems commonly found with doublet items.) This solution produced the expected energy-motivation and cognitive-affective factors. Examination of a three-factor model yielded the same cognitive-affective factor and the energy-motivation items split into a factor with the positively worded items and a factor with the negatively worded items We considered the separation of the energy-motivation items to represent method variance. We therefore adopted the two-factor, energy-motivation and cognitive-affective solution (Table A1). The energy-motivation factor accounted for 20.5% of the total variance the cognitive-affective factor accounted for 23.4% of the total variance. Six items (forgetful, difficulty making decisions, happy, trouble doing daily activities, irritable, and optimistic) did not unambiguously load on one factor.

Table A1 VAS items and factor loadings from the two-factor solution

Item	Energy -motivation	Cognitive -affective
Energy & motivation variables		
Sluggish	**.58**	.26
Sociable	**−.65**	−.16
Interest in usual activities	**−.66**	−.12
Energetic	**−.76**	−.12
Able to concentrate	**−.61**	−.10
Slow	**.59**	.27
Tired	**.56**	.10
Confident	**−.59**	−.33
Affect & cognition variables		
Discouraged	.26	**.72**
Withdrawn	.30	**.63**
Confused	.23	**.63**
Worthless	.11	**.77**
Sad	.23	**.78**
Guilty	.08	**.62**
Hopeless	.23	**.74**
Tearful	.09	**.71**
Ambiguous variables		
Forgetful	.23	.40
Difficult making decisions	.27	.42
Happy	−.61	−.40
Trouble daily activities	.43	.39
Irritable	.39	.45
Optimistic	−.43	−.22
Appetite items		
Level of appetite		
Carbohydrate craving		

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