The Evolutionary Significance of Depressive Symptoms: Different Adverse Situations Lead to Different Depressive Symptom Patterns

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Although much depression may be dysfunctional, the capacity to experience normal depressive symptoms in response to certain adverse situations appears to have been shaped by natural selection. If this is true, then different kinds of situations may evoke different patterns of depressive symptoms that are well suited to solving the adaptive challenges specific to each situation. The authors called this the *situation–symptom congruence* hypothesis. They tested this hypothesis by asking 445 participants to identify depressive symptoms that followed a recent adverse situation. Guilt, rumination, fatigue, and pessimism were prominent following failed efforts; crying, sadness, and desire for social support were prominent following social losses. These significant differences were replicated in an experiment in which 113 students were randomly assigned to visualize a major failure or the death of a loved one.

Keywords: depression subtypes, depressive symptoms, evolutionary psychology, psychopathology, Darwinian psychiatry

> We could never learn to be brave and patient if there were only joy in the world.
>
> —Helen Keller

Depression is not a unitary phenomenon. Different depressive episodes often have different symptoms profiles, even within the same person across time (Oquendo et al., 2004), and the precipitants of depression vary widely, from deaths of loved ones to failures at major goals to chronic stress (Kendler, Gardner, & Prescott, 2002). Thus, a central challenge in depression research has been to disaggregate it into meaningful subtypes, generally based on symptom profiles, precipitating causes, or both. In the present article, we review previous approaches for subtyping depression, and then introduce and test a new framework for understanding how and why depressive symptoms differ across episodes. Our focus is on unipolar depressive symptoms (hereafter, *depressive symptoms*) such as sadness, fatigue, pessimism, and so forth, but unless noted, our usage is agnostic as to whether these symptoms cross a clinical threshold of severity or duration. We provide evidence that different precipitants cause different depressive symptom patterns that are consistent with an evolutionary account of their origins.

Previous Approaches for Subtyping Depression

One straightforward way to subdivide depression is based on the depressive symptoms themselves. For instance, the subtype *depression with melancholia* is characterized by anhedonia, fatigue, chronically depressed mood, early morning insomnia, weight loss, and guilt (*Diagnostic and Statistical Manual of Mental Disorders*, 4th edition, text rev. [*DSM–IV–TR*]; American Psychiatric Association, 2000). Its previous designation, *endogenous depression*, was largely abandoned when it became clear that these symptoms were as likely to be precipitated by life events as other types of depression and that much depression originally reported as having "no cause" was found to be precipitated by events, often of an embarrassing nature (Leff, Roatch, & Bunney, 1970). Another reliably occurring cluster of symptoms, *atypical depression*, is in some ways the opposite of depression with melancholia, characterized by increased appetite and sleeping, heavy feeling limbs, rejection sensitivity, and perhaps mood reactivity (American Psychiatric Association, 2000).

Depression has also been subtyped by the kind of events that precipitate an episode (hereafter *adverse situations* or *precipitants*). For instance, *seasonal affective disorder* (SAD) is recurrent depression with typical onset in the fall/winter; it is characterized

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by fatigue, increased appetite and sleeping, and carbohydrate craving (Rosenthal et al., 1984; Young, 1991). Some SAD symptoms occur in most people in northern latitudes during the winter (Dam, Jakobsen, & Mellerup, 1998), suggesting that SAD may be an extreme of normal wintertime behavioral changes. *Bereavement* is a dysphoric reaction precipitated by the death of a loved one. Common symptoms include a profound sense of loss, emotional pain, crying, and loneliness (Archer, 1999). Bereavement is not considered pathological by *DSM–IV–TR* standards if it fits the expected symptom profile and lasts less than 2 months.

Finally, diathesis-stress models posit that depression subtypes arise from interactions between adverse situations and stable dispositional factors (Abramson, Metalksy, & Alloy, 1989; Beck, 1967). People who characteristically attribute adverse situations to stable, global causes are at higher risk for *hopelessness depression* following adverse situations (Abramson et al., 1989). Some evidence indicates that hopelessness depression is characterized by negative cognitions, decreased motivation, fatigue, psychomotor retardation, sleep disturbances, sadness, poor concentration, and suicidal ideation (Alloy, Just, & Panzarella, 1997; Joiner, 2001). Another diathesis-stress model posits that people who are high in need for approval (sociotropes) are at risk for depression following social losses, whereas people high in need for personal achievement (autonomous individuals) are at risk for depression following failures (Beck, Epstein, & Harrison, 1983). Beck et al. (1983) suggested that depression in sociotropes is characterized by emotional lability, helplessness, crying, anxiety, and concern over social desirability, whereas depression in autonomous individuals is characterized by pessimism, guilt, irritability, and social withdrawal. This symptom-specificity hypothesis generally has been confirmed for sociotropic but not autonomous depression (Burke & Haslam, 2001; Sato & McCann, 2000).

Although these and other previous attempts to subtype depression have captured important dimensions along which depressive reactions differ, we suggest that several factors limit their explanatory power. First, an individual's depressive symptoms are by no means consistent across different depressive episodes (e.g., Coryell et al., 1994; Oquendo et al., 2004), a finding that undermines symptom-specificity models based on individual trait differences, including symptom-specific predictions of diathesis-stress models. Second, previous attempts to subtype depression are not based on a unifying theoretical framework. The symptoms of hopelessness depression, for example, provide little insight into what symptoms we should expect in bereavement, SAD, or sociotropic depression. The symptoms of such descriptive subtypes have been thoroughly documented, but an answer to *why* these particular symptoms coexist remains elusive. Finally, previous attempts to subtype depression often focus on clinical depression. However, clinical depression requires the co-occurrence of a number of prespecified symptoms, which may artificially impose symptom uniformity and obscure heterogeneity that exists in less severe but more common (Judd, Akiskal, & Paulus, 1997) subclinical depressive episodes.

The following two sections introduce a theoretical framework grounded in evolutionary theory that attempts to explain why the capacity for depressive symptoms exists in the first place. Although we focus on normally expressed depressive symptoms—those that most people experience following adverse situations—rather than on clinical depression per se, we believe that this framework might also provide some theoretical coherence to the subtypes of depression reviewed above.

Evolutionary Explanations for Depressive Symptoms

Unpleasant and disabling states, such as fever, pain, nausea, and inflammation, are often assumed to be abnormal even though they are commonly aroused by specific negative situations. However, it has become increasingly clear that these states are controlled by evolved regulation systems that express the response when cues indicate the presence of particular kinds of situations (Nesse, 2005; Nesse & Williams, 1994). Affect states likewise were shaped by selection to deal with the challenges posed by certain situations (Nesse, 1990). In particular, depressive symptoms are consistently aroused in response to certain adverse situations (Monroe & Simons, 1991), and they appear closely regulated. This suggests that depressive symptoms are not necessarily maladaptive but rather can be useful in the types of negative situations that arouse them.

Several previous evolutionary hypotheses have argued for domain-specific functions of depression, such as a signal of submissiveness following a loss of status (Price, Sloman, Gardner, Gilbert, & Rhode, 1994), a strategy to conserve energy and resources (Engel, 1980), a way to avoid social losses (Allen & Badcock, 2003), as a means of social manipulation (Hagen, 1999; Watson & Andrews, 2002), or as a way to analyze complex social problems (Watson & Andrews, 2002). A more inclusive model suggests that depressive symptoms can be useful in unpropitious situations in any domain (Klinger, 1975; Nesse, 2000). These evolutionary models do not hypothesize that depressive symptoms are always adaptive but rather that the capacity to express them in certain adverse situations increased fitness among human ancestors, and so these capacities continue to be a part of human nature today.

Our own hypothesis can be differentiated from most previous evolutionary hypotheses in at least two ways. First, we focus on normally expressed depressive symptoms rather than on clinical depression per se, not only because clinical depression may conceal important symptom heterogeneity but also because clinical depression is more likely to be an inappropriate and maladaptive response (see also Allen & Badcock, 2003). Intense and prolonged depressive symptoms (depression) may sometimes be normal, nonpathological responses to chronic or severe precipitants (whether useful or not in the individual instance), but at other times may reflect defects or maladaptive "noise" in the mechanisms responsible for regulating normal depressive symptoms (Keller & Miller, in press). Second, we do not argue that depressive symptoms have a unitary cause or serve a unitary function (see also Watson & Andrews, 2002). Rather, given that highly varied situations can arouse depressive symptoms and that many depressive symptoms have little in common (e.g., crying vs. fatigue vs. pessimism), we hypothesize that different symptoms serve related but nevertheless distinguishable functions. We see depressive symptoms as partially differentiated branches on a phylogenetic tree (Nesse, 2004).

The Situation–Symptom Congruence Hypothesis

If different depressive symptoms serve different functions, then different precipitants should give rise to different symptom patterns that increase the ability to cope with the adaptive challenges

specific to each situation. We term this predicted match *situation–symptom congruence* (SSC). Specific patterns of SSC can be predicted from the potential functions of 11 depressive symptoms and the utility of these functions in different situations.

1. *Emotional pain* or sadness should occur in response to losses of resources valuable to fitness (Nesse, 2000). As with somatic pain, the aversiveness of emotional pain is its raison d'être: It draws attention to and stimulates withdrawal from currently harmful situations, and it motivates avoidance of future actions that could lead to similar losses (Carver, 2004; Nesse, 2004). Given that social bonds have probably been among the most fitness-relevant resources throughout human evolution, social losses should be especially painful. Situations that do not represent a loss per se, and in which a specific and potentially avoidable event is not the cause, should elicit less emotional pain.

2. *Crying*, like many emotional signals, is expressed via configurations of facial musculature and vocal behaviors, and it elicits specific reactions in receivers of the signal—in this case, empathy and comforting behaviors (Hill & Martin, 1997). It seems likely therefore that crying requests and secures aid. Given that crying appears to strengthen social bonds (Frijda, 1986), we predict that crying will be especially prominent when social bonds themselves are threatened, lacking, or lost. This hypothesis may seem at odds with evidence that depression is often met with interpersonal rejection (e.g., Segrin & Abramson, 1994). However, these conclusions are relevant to extreme depressive symptoms rather than crying in appropriate contexts. Moreover, this research generally indicates that depression elicits rejection from strangers and loose associates; depression appears to elicit *solicitous* responses from people close to the depressed person (Sheeber, Hops, Andrews, Alpert, & Davis, 1998).

3. *Desire for social support* would also be adaptive when help is needed. As with crying, the motivation for forming/strengthening social bonds may be especially high following social losses to replace lost bonds. When the loss is not social, however, forming social bonds should be less important.

4. *Fatigue* refers to physical or mental weariness. Normally, fatigue results from exertion and motivates conserving energy and disengaging motivation. It is parsimonious to assume that fatigue serves the same functions when continued striving is unlikely to be rewarded, such as following failures (given that continued striving at failed goals is maladaptive), when one is unable to cope with all they are attempting to do, or when physical exertion should be minimized to conserve energy, such as might have occurred during ancestral winters.

5. *Pessimism* is the tendency to expect unfavorable future outcomes. Some evidence suggests that such depressive shifts are actually away from a baseline optimistic bias and toward more realistic appraisals (Alloy & Ahrens, 1987), although in certain domains, pessimism is clearly unrealistically negative (e.g., Stone, Dodrill, & Johnson, 2002). Given that goal pursuit reflects the perceived likelihood of success (Carver & Scheier, 2001), pessimism should diminish initiative and withdraw the organism from current and potential goals (Klinger, 1975) and should be most prominent when future efforts are unlikely to succeed.

6. *Guilt* refers to feelings of self-reproach and worthlessness. Guilt might motivate an individual to try to figure out how his or her actions led to the situation, and so should be prominent in proportion to the degree of control the individual had in the situation.

7. *Rumination*, or the obsessive replaying of negative events, feelings, and implications of those feelings, is a common concomitant of depression (Beck, 1967). Numerous studies have concluded that rumination is maladaptive, based on evidence that it increases other depressive symptoms (Nolen-Hoeksema, 1991). However, this conclusion only holds if other depressive symptoms are indeed maladaptive. Moreover, research in emotion regulation stresses the importance of working through rather than avoiding negative emotions (Stanton, Kirk, Cameron, & Danoff-Burg, 2000). Along with other theorists (Martin & Tesser, 1996; Watson & Andrews, 2002), we hypothesize that rumination aids in understanding the causes and consequences of the adverse situations to avoid such situations in the future and to reconsider strategies and goals themselves. If so, rumination should be most prominent when the best future course of action is uncertain or after an untoward event that is potentially avoidable and could recur.

8. *Anhedonia* refers to diminished mood reactivity and a decreased ability to experience positive emotions. Positive emotions, according to numerous theorists, facilitate approach behavior and increase risk-taking (see Fredrickson, 2001). An inability to experience positive emotions should decrease these tendencies and should be prominent when the environment is unpropitious.

9. *Anxiety* is a painful state of uneasiness or nervousness about possible future losses. Anxiety promotes wariness and hypervigilance, particularly toward potential threats, and so should be adaptive in threatening situations (Marks & Nesse, 1994).

10. *Appetite changes* can increase or decrease food intake during depressive episodes. In the most serious cases of depression, appetite is diminished (Beck, 1967). This lack of response to normally pleasurable cues can be seen as a concomitant of anhedonia. A temporary decrease in foraging could have adaptively reduced energy expenditure and risk exposure in unpropitious situations in which efforts would likely be wasted. An increase in appetite, on the other hand, might have been adaptive when food is in short supply, such as during ancestral winters.

11. *Sleep increases or decreases* often occur during depressive episodes. It seems possible, but tenuous at best, that wakefulness is a form of nocturnal hypervigilance in risky situations. More sleep, on the other hand, could adaptively conserve energy in unpropitious situations.

The 11 depressive symptoms above are ordered according to our subjective confidence about the proposed functions of each symptom. Certain depressive symptoms may simply be epiphenomena with no adaptive utility. For example, changes in sleep and appetite may be byproducts of more general changes in arousal. We have emphasized strictly functional accounts, in part, because they are more easily falsified. Support for nonadaptive explanations increases to the degree that empirical support for functional hypotheses is weak. We also note that functional accounts are not alternatives to proximate explanations about responsible mechanisms, either psychological or neurophysiological. Both proximal and ultimate/evolutionary explanations are essential for a full understanding of depressive symptoms.

The prediction made by the SSC hypothesis is not that symptoms will be present or absent depending on the situation but only that they should be more or less pronounced in the predicted patterns. The fuzzy boundaries arise in part because the varied precipitants have multiple overlapping adaptive challenges. For instance, over evolutionary time, failures were probably preceded more often by other failures than by social losses, so we predict

more fatigue, pessimism, and rumination following failures than following social losses. Nevertheless, social losses were also probably associated with future failures to some degree, and so also should elicit some degree of these symptoms. Depressive symptom patterns should differ quantitatively, not qualitatively, across precipitants.

Such partial differentiation of response specificity has precedents in other biological domains. Antigens arouse general responses (inflammation, fever, and malaise) helpful in fighting a wide range of infections, as well as specific responses that depend on the kind of threat (eosinophils to parasites, interferon to viral invasion, and natural killer cells to cancerous cells). Subtypes of anxiety may also be partially differentiated to cope with different kinds of dangers (Marks & Nesse, 1994). The fit between situation and response supports the hypothesis that the immune response and anxiety are defensive reactions that maximized ancestral fitness in negative situations. Likewise, evidence for a fit between different kinds of situations and specific depressive symptoms would support the hypothesis that depressive symptoms aided ancestral fitness during such situations.

In a previous study (Keller & Nesse, 2005), we found that students reported more fatigue and pessimism following failures or during the wintertime, and they reported more crying and sadness following social losses. These results are consistent with the SSC hypothesis but are preliminary for two reasons. First, symptom scales were derived from the Center for Epidemiologic Studies—Depression Scale (CES–D; Radloff, 1977) using face validity, meaning that many symptoms could not be assessed, and those that could had unknown reliabilities. Second, symptom–situation associations were based solely on retrospective reports that are open to noncausal explanations.

To circumvent problems inherent to using existing depression scales, we developed a scale designed to measure different depressive symptoms in Study 1. In Study 2, participants used this scale to retrospectively report the symptoms that followed a recent adverse situation. To increase confidence that different situations cause different symptom patterns, we randomly assigned participants in Study 3 to imagine either the death of a loved one or the failure of a major goal prior to reporting current depressive symptoms.

Study 1: Depressive Symptoms Scale Development

Depression inventories, such as the CES–D, Beck Depression Inventory (BDI; Beck, Steer, & Garbin, 1988), and Hamilton Depression Rating Scale (Hamilton, 1967), are designed and validated to measure a single underlying latent construct, depression severity, so they are poorly suited to measure specific symptoms of depression. The only scale designed to measure separate depressive symptoms, the Multiscore Depression Inventory (Berndt, Petzel, & Berndt, 1980), does not assess many of the symptoms for which we made predictions and was not developed using modern statistical methods. To investigate whether adverse situations lead to different symptom patterns, it was necessary to create our own scale of depressive symptoms, the Depressive Symptoms Scale (DSS).

Method

Participants

Because we wished to study normal reactions that follow adverse situations in ordinary people, we used nonclinical populations to validate the DSS. The exploratory sample, also used in Study 2, consisted of undergraduate students who completed the study for course credit. At the beginning of one fall and one winter semester, we prescreened 2,664 introductory psychology students (57% female) for the experience of a 2-week period when they felt "down, sad, or disturbed" during the previous 12 months; 1,127 of the 2,664 students (42%) reported such a period. They then indicated the situations (if any) they thought caused this episode from among the following: general stress or inability to cope (46.7%), social isolation (39.5%), romantic breakup (25.4%), failure at an important goal (19.7%), a specific situation not mentioned above (18.9%), death of a loved one (13.0%), the wintertime (9.7%), and no specific cause (8.4%). These categories were derived from our earlier study (Keller & Nesse, 2005), and the percentages sum to more than 100% because participants could choose more than one category.

To ensure adequate sample sizes for each precipitant category (important for Study 2), we prescreened participants to oversample those who experienced less common precipitants (deaths of loved ones, wintertime blues, and failures). We invited 623 of the 1,127 eligible students to participate for course credit, of which 473 agreed and 456 of these (96%) completed the study. A further 11 responses (2.5%) were later dropped because they had incomplete data, indicated on a probing question that they had not taken the survey seriously (see *Measures*), or visited the debriefing Web page before completing the survey. Of the 445 complete responses, 283 were female, 162 were male, and ages ranged from 18–23 years (M = 18.8, SD = 1.0).

We supplemented the exploratory sample with a cross-validation sample of 311 participants who volunteered to participate on a Web site dedicated to online psychological studies. Unlike the exploratory sample, the cross-validation sample was used solely in Study 1 because information regarding the precipitating situation was not collected. The data for 22 participants (7%) were dropped prior to any analysis for the same reasons mentioned for the exploratory sample. Of the remaining 289 eligible participants, 221 were female and 68 were male; 269 lived in North America, 14 lived in Europe, and 6 lived in Asia; ages ranged from 18 to 58 years (M = 27.1, SD = 9.9).

Procedure

Participants who met our prescreening criteria in the exploratory sample completed the survey over the Internet at a private location (usually at home) after receiving an e-mail with the Web address. Participants in the cross-validation sample chose the present study among many other study titles by clicking on a link titled "25 Minute Psychology Survey." After reading the consent form and filling out a short demographic questionnaire, participants from both samples identified the weeklong period when they felt the worst emotionally in the previous 12 months. To refresh their memories of this period, participants wrote a free-format paragraph about what events or situations, if any, they thought precipitated the depressive symptoms and another paragraph about how they felt during the weeklong period when they felt the worst. Participants in the exploratory sample provided additional information (see *Measures*, Study 2). Finally, all of the participants responded to items from the DSS, the CES–D, and then the BDI scales regarding the symptoms that occurred during the week when they felt the worst, and then answered a final probing question. The last page fully debriefed the participants.

Measures

DSS. Participants answered 66 questions, written by Matthew C. Keller, which assessed 11 depressive symptoms (6 questions per symptom): Emotional Pain, Anhedonia, Fatigue, Pessimism, Rumination, Crying, Guilt, Anxiety, Changes in Eating Habits, Changes in Sleeping Habits, and Desire for Social Support. The first 10 symptoms are commonly identified as symptoms of depression (Beck, 1967); Desire for Social

Support was added based on predictions of the SSC hypothesis. When awkward wording could be avoided, an equal number of positively and negatively worded items were included for each scale. Because participants reported on how they felt during a weeklong period, we used the duration response format from the CES–D (*rarely or none of the time* = 1; *some of the time* = 2; *a moderate amount of the time* = 3; *most or all of the time* = 4). We attempted to assess a range of symptom intensities across the 6 questions for each symptom. Items were presented in random order within groups of 11 items, such that each symptom was represented only once within each group, and groups were then randomly ordered.

The DSS instructions read as follows:

> Please think carefully about how you felt during the weeklong period when you felt the worst. After each statement, indicate how often you felt the ways described. Remember: (a) all responses are completely anonymous, so be as honest as possible; (b) answer each item separately from all others, even if some questions seem redundant; (c) there are no right or wrong answers; try to indicate how you actually felt rather than how you think you "should" have felt.

Other depression inventories. Participants also filled out the BDI and CES–D. The BDI is a 21-item measure of depression that uses a 4-point, statement-anchored response format. The CES–D scale is a 20-item measure of depression that uses a 4-point, frequency response format. The CES–D is often considered a more sensitive measure of less severe, subthreshold depression compared with the BDI. The BDI and CES–D items were reworded to be in the past tense for this study.

Probing questions. Participants answered the question "How seriously have you answered questions on this survey up to this point (all responses are 100% anonymous . . . knowing this really helps us out)" using a 5-point, description-anchored scale (*very seriously* = 1 to *not seriously at all* = 5). Ninety-eight percent of participants indicated that they had taken the task seriously or very seriously.

Analysis

The analysis proceeded in three phases. In the first phase, conducted on responses from the exploratory sample, we used exploratory factor analysis (EFA) and then confirmatory factor analysis (CFA) to uncover the latent structure of the 66 depressive symptom items and to drop items and factors that did not fit this structure. In the second phase, we cross-validated the final (primary) model from the exploratory sample on the cross-validation sample using CFA. In the final phase, we compared the primary model with several alternative models. We treated item responses as ordinal rather than continuous data by fitting all factor models using robust weighted least squares on polychoric correlations with Mplus 3 software (L. K. Muthén & Muthén, 1998).

The data set from the combined sample ($N = 724$) contained 71 missing values out of a total of 47,784 (724×66) possible values (.0015 of the total data frame). We imputed the missing values using PRELIS 2 (Jöreskog & Sörbom, 1996). To check that this imputation did not alter the model fits, we reran final models using listwise deletion. Changes in fit were extremely minor and are not reported.

Results and Discussion

Refinement of the Primary Model in the Exploratory Sample

We determined the number of factors using scree plots from principal-axis EFA with oblique promax rotation. As expected, the first eigenvector explained a large amount of the variation (31%) in item responses. Nevertheless, much of the latent factor substructure was not captured by this single factor; solutions of between 11 and 13 latent factors fit the data best (accounting for 69% to 74% of the overall variation). The 13-factor solution was the most interpretable, having factors corresponding to nine of the symptoms that we had expected, as well as four factors from questions we originally thought would tap into just two symptoms: sleepiness and quality of sleep (from Changes in Sleeping Habits questions) and loss of appetite and increased eating/weight gain (from Changes in Eating Habits questions).

We used CFA to refine the 13-factor solution suggested by EFA. Because part of our interest was to understand how depressive symptoms relate to each other, we allowed the 13 latent factors to be intercorrelated. Items that loaded poorly on the factors (standardized loadings $< .50$), that were factorially complex (as judged by modification indices showing large cross-loadings to other factors) or for which the model explained little item variation ($R^2 < .30$) were dropped sequentially, and the model was rerun. Factors that ended up having fewer than 3 items (quality of sleep and increased eating/weight gain) were dropped. This procedure was iterated until no more items or factors could be dropped. The primary model for the DSS in the exploratory sample retained 47 items that loaded onto 11 depressive symptoms (see Table 1).

Table 2 shows the maximum likelihood correlation matrix as well as the descriptive statistics for the 11 DSS symptom scales from the exploratory sample. The average coefficient alpha (Cronbach, 1951) for the 11 DSS subscales was .86. We conducted a second-order EFA with oblique promax rotation on the 11×11 correlation matrix shown in Table 2. Fatigue, Anhedonia, Emotional Pain, Pessimism, Crying, Low Appetite, and Sleepiness loaded most highly (in order) onto the first factor (Overall Dysphoria); Guilt, Rumination, Pessimism, and Anxiety loaded most highly onto the second factor (Brooding/Agitation); Crying, Emotional Pain, and Desire for Social Support loaded most highly onto the third factor (Signal for Help). We used the factor loadings from the Overall Dysphoria factor to create an Overall Dysphoria score for each participant, which appeared to tap into the same construct as the overall scores of the BDI and CES–D (see Table 2).

Cross-Validation of the Primary Model

In the second phase of the analysis, we assessed the degree to which the primary model developed with the exploratory sample explained item covariance in the cross-validation sample. The chi-square statistic is not a good index of fit in this case because even trivial lacks of fit tend to be significant with large sample sizes. Better fit indices are the Tucker–Lewis index (TLI), the comparative fit index (CFI), and the root-mean-square error of approximation (RMSEA). For both continuous and categorical data (Yu & Muthén, 2001), TLI $> .95$, CFI $> .95$, and RMSEA $< .06$ suggest "good fits" (Hu & Bentler, 1999), whereas TLI $> .90$, CFI $> .90$, and RMSEA $< .10$ have historically suggested "acceptable fits" (Browne & Cudeck, 1993; though see Hu & Bentler, 1999).

For the exploratory sample, two of the indices from the primary model indicated good fits and two indicated acceptable fits. We expected a decent fit for the exploratory sample because the model was refined using this sample; better information of a model's generalizability comes from the same model run on an independent

Table 1
Means and Factor Loadings for the Depressive Symptoms Scale

Scale and item	Exploratory sample		Confirmatory sample	
	M	SL	*M*	SL
Emotional Pain				
I felt really sad	3.39	.87	3.38	.80
I "hurt" inside, even though the pain wasn't physical	3.28	.74	3.32	.78
I felt fine emotionally	1.42	−.87	1.41	−.90
I was in agony	2.40	.77	2.87	.82
I was free from emotional pain	1.34	−.71	1.31	−.65
Pessimism				
Things seemed hopeless	2.98	.85	3.00	.91
I felt pessimistic about the future	2.94	.81	2.98	.80
I felt like things were going to turn out really well	1.49	−.73	1.61	−.86
I felt discouraged about things	3.23	.76	3.23	.77
I felt hopeful for the future	1.75	−.74	1.76	−.81
Fatigue				
I felt as energetic as I normally do	1.58	−.70	1.58	−.72
Everything seemed like such an effort	2.72	.64	2.99	.77
I felt active and full of "pep"	1.30	−.80	1.33	−.74
I could not "get going"	2.71	.76	2.91	.82
It was easy to get a lot of things done	1.53	−.58	1.59	−.62
Anhedonia				
I was still able to feel happy	2.00	−.79	1.86	−.81
I enjoyed life	1.69	−.87	1.66	−.89
Nothing could make me smile	2.26	.76	2.46	.78
Things that normally gave me joy continued to give me joy	2.15	−.68	1.91	−.74
I was incapable of feeling anything pleasant	2.09	.81	2.57	.78
Rumination				
I couldn't "let go" of certain thoughts	3.52	.72	3.52	.76
I was able to clear problems from my mind	1.52	−.61	1.52	−.67
I thought about how I could have done things differently	3.26	.61	3.24	.62
I would catch myself thinking about the same issue	3.55	.64	3.58	.81
Crying				
I felt like crying	3.21	.91	3.12	.92
I cried really hard	2.43	.94	2.48	.92
I got teary-eyed	2.82	.89	3.02	.89
I sobbed	2.40	.95	2.64	.97
It took effort to fight off tears	2.64	.83	3.00	.73
Guilt				
I felt ashamed	2.41	.77	2.53	.82
I felt guilt-free	1.79	−.60	1.81	−.63
I was angry at myself	2.68	.88	2.75	.77
Rational or not, I blamed myself	2.76	.83	2.80	.85
Low Appetite				
The thought of food was not appealing	2.10	.89	2.39	.85
I lost my appetite	2.01	.93	2.47	.91
Food didn't taste as good as it usually did	2.13	.86	2.58	.93
Anxiety				
I was free from fear	1.86	−.54	1.73	−.61
Things made me nervous	2.26	.68	2.65	.80
I was free from worrying	1.34	−.76	1.41	−.83
I was more afraid than usual	2.08	.88	2.61	.83
I felt anxious	2.38	.85	2.71	.78
Sleepiness				
I wanted to sleep all day	2.62	.66	2.48	.60
I slept more than I normally do	2.31	.97	2.16	1.03
I felt sleepy even when I had gotten plenty of sleep	2.69	.86	2.62	.72
Desire for Social Support				
I felt like having a heart-to-heart with a close friend or relative	2.65	.92	2.58	.45
I wanted to share how I felt with someone	2.75	.88	2.71	1.70
I wanted to be with close friends or family for support	2.63	.82	2.50	.37

Note. Item means are reported on a 1–4 scale. SL = standardized loadings from freely estimated pathways in threshold confirmatory factor models.

Table 2
Correlations and Descriptive Statistics Among 11 Depressive Symptoms Scale (DSS) Subscales, Overall Dysphoria, the CES-D, and the BDI

Scale	1	2	3	4	5	6	7	8	9	10	11	12	13	14
1. Emotional Pain	—													
2. Pessimism	**.82**	—												
3. Fatigue	**.71**	**.77**	—											
4. Anhedonia	**.81**	**.79**	**.82**	—										
5. Rumination	**.81**	**.75**	**.63**	**.64**	—									
6. Crying	**.79**	**.50**	**.46**	**.54**	**.54**	—								
7. Guilt	**.52**	**.64**	**.47**	**.46**	**.81**	**.26**	—							
8. Low Appetite	**.53**	**.44**	**.53**	**.57**	**.42**	**.41**	**.33**	—						
9. Anxiety	**.56**	**.55**	**.43**	**.34**	**.59**	**.37**	**.60**	**.35**	—					
10. Sleepiness	**.36**	**.44**	**.73**	**.39**	**.27**	**.30**	**.25**	**.39**	**.25**	—				
11. Desire for Social Support	.08	**−.16**	**−.16**	**−.25**	**.12**	**.22**	−.04	−.04	**.13**	−.07	—			
12. Overall Dysphoria	**.94**	**.96**	**.93**	**.92**	**.86**	**.68**	**.65**	**.61**	**.61**	**.58**	**−.11**	—		
13. CES-D	**.82**	**.83**	**.84**	**.83**	**.76**	**.59**	**.60**	**.61**	**.57**	**.54**	**−.13**	**.88**	—	
14. BDI	**.75**	**.80**	**.78**	**.77**	**.75**	**.51**	**.68**	**.61**	**.57**	**.52**	**−.17**	**.82**	**.86**	—
No. items in scale	5	5	5	5	4	5	4	3	5	3	3	47	20	21
M	3.27	3.19	3.21	2.71	3.46	2.70	2.77	2.08	2.71	2.55	2.66	2.89	2.65	2.08
SD	0.64	0.69	0.58	0.66	0.55	0.94	0.84	0.96	0.73	0.98	1.00	0.74	0.55	0.50
Coefficient α	.89	.87	.82	.88	.73	.95	.84	.91	.84	.88	.90	n/a	.89	.89

Note. Significant correlations ($p < .01$) in bold. The DSS is made up of Subscales 1–11. Statistics for scales and subscales are based on participants' means of the questions making up the scales after relevant items were reversed. CES-D = Center for Epidemiologic Studies–Depression Scale; BDI = Beck Depression Inventory.

sample. We ran two types of models on the cross-validation sample. The factor pattern invariant model (Table 3, row 2a) fixed the pathways to be the same as they were in the exploratory sample but allowed the loadings to vary. The fits of this model were similar to the fit for the exploratory sample, with the RMSEA index showing a slight decrement. The factor loading invariant model (Table 3, row 2b) fixed both the pathways and their loadings to be the same as in the exploratory sample. The fits of this more stringent model were somewhat degraded.

Taken together, the cross-validation results indicate that the same basic latent factor structure existed in both samples but that the specific values of the factor loadings differed slightly between them. Because the patterns were similar but loadings differed between the samples, we combined the two samples for further analyses but allowed the factor loadings as well as the means of the latent variables to differ between them.

Comparisons With Alternative Models

As recommended by Cliff (1983), we compared the primary model with several plausible alternative models to gauge the uniqueness of the primary model and to better understand the structure of the items and latent factors. All items loaded directly onto a single Overall Dysphoria factor in the first alternative model (Table 3, row 4). The very poor fit of this model indicates that items from similar symptoms do indeed cluster together. The second alternative model (Table 3, row 5) is the same as the primary model except that items from four symptoms having the

Table 3
Goodness-of-Fit Summaries for Confirmatory Factor Models

Model	χ^2	*df*	CFI	TLI	RMSEA
Primary model: Eleven intercorrelated subscales					
1. Exploratory sample ($n = 446$)	596*	176	.971	.913	.073
2. Cross-validation sample ($n = 289$)					
2a. Pattern invariant	381*	101	.964	.902	.098
2b. Loading invariant	187*	37	.947	.947	.119
3. Full sample ($N = 735$)	927*	265	.969	.908	.083
Alternative models (full sample)					
4. No subscales; all items load directly onto Overall Dysphoria	3,606*	150	.532	.818	.202
5. Eight intercorrelated subscales model	1,285*	265	.858	.952	.103
6. Eleven subscales correlate only through Overall Dysphoria	892*	163	.874	.954	.101
7. Three second-order intercorrelated latent factors	1,005*	245	.962	.895	.093

Note. χ^2 and *df* statistics are approximations due to fitting of robust weighted least squares using polychoric correlations. The *df* statistics differ between identical models (rows 1 and 3) because sample sizes are used in these *df* approximations (B. O. Muthén, 2004). See text for explanations of terms and descriptions of models. CFI = comparative fit index; TLI = Tucker-Lewis index; RMSEA = root-mean-square error of approximation.
* $p < .001$.

highest intercorrelations—Emotional Pain, Fatigue, Anhedonia, and Pessimism—loaded instead onto a single latent factor, Core Dysphoria. The poor fit of this model indicates that although these four symptoms appear to be core depressive symptoms, items tapping into them are not interchangeable, and these four symptoms are differentiable. In the third alternative model (Table 3, row 6), the items related to the symptoms as in the primary model, but the 11 symptoms loaded onto a single Overall Dysphoria factor, such that the intercorrelations between the symptoms were explained only by their associations with Overall Dysphoria. The relatively poor fit of this model indicates that there is substructure even between the 11 symptoms themselves; the relationships between symptoms cannot be captured along a single dimension. The fourth alternative model (Table 3, row 7) was similar to the third, except that the 11 symptoms loaded onto the three intercorrelated second-order latent factors (Overall Dysphoria, Brooding/Agitation, and Signal for Help) that were suggested by the second-order factor analysis described above. The similar fit of this model compared with the primary model suggests that the relationships between symptoms fall mainly along three dimensions, and this model might be considered a viable alternative to the primary model.

Summary

The DSS primary model fit the exploratory sample well to adequately depending on the criterion. The differences in fits between the exploratory and cross-validation samples were not large, which is noteworthy given the major demographic differences between the two samples. The factor structure of the DSS also appeared preferable to several plausible alternative factor structures. Thus, the basic DSS structure reported here—11 intercorrelated subscales—should generalize to the nonclinical populations from which these samples were drawn. Although the DSS is sufficiently reliable for the present study, the present version should not be considered final. Future revisions of the DSS need to improve the model's overall fit by rewording items with low loadings and including more items for symptoms with few items.

Study 2: Test of the Situation–Symptom Congruence Hypothesis

In Study 2, we used the DSS to investigate whether the patterns of depressive symptoms differed depending on the precipitating situation, and if so, whether these patterns were consistent with the SSC hypothesis.

Method

Participants and Procedure

The participants of Study 2 were the exploratory sample described in Study 1. The procedure is also described above, although participants provided more detailed information, as described below.

Measures

Categorical precipitants. After writing the free-format paragraphs about the causes of their depressive symptoms (see *Procedure*, Study 1), participants chose the *single most likely* cause from among the following eight (mutually exclusive) precipitants: *Death of Loved One* ($n = 44$), *Romantic Loss* ($n = 92$), *Social Isolation* ($n = 112$), *Failure at Important Goal* ($n = 44$), *Stress or Difficulty Coping* ($n = 83$), *Wintertime* ($n = 30$), *No Cause* ($n = 13$), and *Other Cause* ($n = 27$). Privacy protections did not allow matching prescreening data to participants' responses collected during the study, and so we could not assess the degree to which these responses corresponded to their earlier, nonmutually exclusive responses provided during prescreening.

Properties of precipitants. Using 6-point scales (*not at all* = 1 to *completely* = 6), participants answered the following five questions about the precipitant: (a) "To what degree was the situation due to a social loss (e.g., losing someone close to you through a death or a breakup, losing a friend after a fight)?" (*Social Loss*; $M = 3.6$, $SD = 1.8$); (b) "To what degree was the situation caused by your effort at something not working?" (*Failed Effort*; $M = 3.0$, $SD = 1.6$); (c) "To what degree was the situation due to being shamed?" (*Shamed*; $M = 2.1$, $SD = 1.4$); (d) "To what degree did the situation occur suddenly (vs. gradually)?" (*Suddenness*; $M = 3.7$, $SD = 1.6$); and (e) "To what degree did you have control over the situation?" (*Control*; $M = 2.5$, $SD = 1.4$). Participants then indicated the date when the precipitant occurred or when they began to feel bad if no precipitant occurred. *Time Since Precipitant* ($M = 39.5$, $SD = 30.4$) was defined as the number of weeks between this date and when the survey was completed.

Depressive symptom scores. We obtained 11 standardized symptom scores for each participant from the primary DSS model on the exploratory sample (see Study 1) using the SAVE = FSCORES command in Mplus 3. We also obtained an overall dysphoria score based on a weighted combination of the 11 symptoms (see Study 1).

Information on present mood, antidepressant usage, and depression history. Following completion of the DSS, CES–D, and BDI surveys (see *Measures*, Study 1), participants rated their mood over the last week using a description-anchored, 9-point scale (*completely depressed* = 1 to *completely euphoric* = 9; $M = 5.4$, $SD = 1.7$). Participants then indicated how often they had been depressed in their life from among the following: "I have never been depressed" (10%), "I have been depressed once in my life" (17%), "I have been depressed a few times in my life" (65%), "I have been depressed most of my life" (7%), and "I have been depressed for as long as I can remember" (1%). Finally, participants indicated whether they were currently taking antidepressant medication (10% were).

Analysis

The global prediction that different precipitants would be associated with different depressive symptoms patterns was tested by the Precipitant × Symptom interaction term in repeated measure multivariate analysis of variance (MANOVA) using the GLM command in SPSS software. The 11 depressive symptoms from the DSS were within-subject dependent variables, and the 7 categorical precipitants served as between-subjects independent variables. The *Other Cause* precipitant was not included in these analyses, reducing the sample size from 445 to 418. This repeated measure MANOVA analysis is similar to a mixed (split-plot) analysis of variance (ANOVA), with one between-subjects variable (precipitant type, 7 levels) and one within-subject term (symptom type, 11 levels), but it does not require the rarely met statistical assumption of sphericity (Tabachnick & Fidell, 2001). We tested predictions of the SSC hypothesis using both within- and between-subjects follow-up ANOVA contrasts. We used structural equation modeling (SEM) in Mplus 3 software to test whether the ratings on the degree to which the precipitants involved social loss and failed effort (from *Properties of Precipitants*) were differentially related to the 11 DSS symptoms.

With sample sizes as large as the present one, multivariate normality is not crucial for statistical inference with MANOVA or SEM, but the presence of outliers can be problematic (Tabachnick & Fidell, 2001). We found no multivariate outliers using a conservative $p < .001$ criterion for Mahalanobis distances, which compared the highest scores and those

expected from a chi-square distribution with degree of freedom equal to the number of variables. Because the sensitive Box's *M* test indicated that the assumptions of equality of the variance–covariance matrices were violated on the omnibus MANOVA analyses, we used Pillai's approximation to the *F* (hereafter Pillai's *F*) for omnibus tests, which is robust to this assumption (Olson, 1979).

Results and Discussion

Tests of the Situation–Symptom Congruence Hypothesis

The Precipitant × Symptom MANOVA interaction term was highly significant across the 11 DSS symptoms, Pillai's $F(60, 2448) = 4.77$, $p < .001$, partial $\eta^2 = .11$, indicating that different precipitants aroused different patterns of depressive symptoms. Controlling for gender, time since the precipitant, number of previous dysphoric episodes, antidepressant usage, and mood in the last week, the Precipitant × Symptom interaction remained significant, Pillai's $F(60, 2406) = 4.64$, $p < .001$, partial $\eta^2 = .10$.

We used the hypothesized functions of each symptom (outlined in the introduction) to predict which symptoms should be prominent following the particular precipitants investigated in Study 2 (see Table 4). Anxiety was left out because none of its predictions corresponded well to the six precipitant categories. The fourth column of Table 4 shows 10 between-subjects contrast tests, one per symptom, which compared the mean symptom levels of precipitants expected to have high levels of that symptom versus the mean of the other precipitants. SSC predictions were supported for 4 of 10 symptoms. However, main effects of precipitants can obscure precipitant differences within symptoms (Tabachnick & Fidell, 2001). For example, fatigue levels may not have differed between the romantic loss and winter precipitants, as predicted, simply because virtually *all* symptoms were higher for romantic loss (see Figure 1). Controlling for overall dysphoria, the predicted precipitants had significantly higher mean levels, relative to their overall symptom levels, for 8 of the 10 symptoms (fifth column, Table 4).

An alternative to testing if precipitants differ within each symptom is to test if symptom levels differ within each precipitant. This approach does not suffer from the analogous problem discussed in the previous paragraph—main effects of symptom levels obscuring symptom pattern differences in this case—because symptoms, having means of zero, necessarily had no main effects. For each precipitant, we performed a repeated measures contrast test that compared the combination of symptoms expected to be prominent versus the combination of all other symptoms. Except for the symptom pattern following an inability to cope, SSC predictions were supported (contrasts shown in Figure 1). These effects held or grew stronger after controlling for the same five variables described in the previous paragraph. The SSC-inspired contrasts predicted a substantial amount of the variation in depressive symptom patterns ($\eta^2 = .03$ to .33, depending on the precipitant). This is impressive given that different people must react differently to similar situations and that each participant had to choose a single, mutually exclusive precipitant, which may not always reflect reality.

SEM Tests of Situation–Symptom Congruence

In addition to assessing whether symptom patterns differ between mutually exclusive precipitant categories, the SSC hypothesis can also be tested by assessing whether symptom patterns differ as a function of the *degree* to which relevant dimensions were perceived to play a role in causing the depressive symptoms. We chose to focus on two dimensions, Social Loss and Failed Effort (from *Properties of Precipitants*), because these dimensions are predicted by the SSC hypothesis to lead to much different symptom profiles. To test this, we began with a fully saturated SEM model in which Social Loss and Failed Effort had pathways

Table 4
Results of Follow-Up Between-Subjects Contrast Tests for Each Symptom

Symptom	Proposed function of symptom	Symptom should be prominent following:	Are mean symptom levels significantly higher among predicted precipitants?	
			Not controlling for overall dysphoria[a]	Controlling for overall dysphoria[b]
Emotional pain	To make fitness-relevant losses aversive	Death, romantic loss, social isolation, failure	No, $t = 0.73$, $p = .532$, $\eta^2 = .00$	Yes, $t = 5.33$, $p < .001$, $\eta^2 = .07$
Crying	To signal a need for help and succor	Death, romantic loss, social isolation	Yes, $t = 2.91$, $p = .004$, $\eta^2 = .02$	Yes, $t = 5.27$, $p < .001$, $\eta^2 = .06$
Social support	To make or re-form social bonds	Death, romantic loss, social isolation	Yes, $t = 4.19$, $p < .001$, $\eta^2 = .04$	Yes, $t = 4.06$, $p < .001$, $\eta^2 = .04$
Fatigue	To down-regulate effort	Failure, can't cope, winter	No, $t = -1.76$, $p = .186$, $\eta^2 = .00$	Yes, $t = 3.17$, $p = .002$, $\eta^2 = .03$
Pessimism	To give up on failing goals	Failure, can't cope	No, $t = 0.93$, $p = .352$, $\eta^2 = .00$	Yes, $t = 5.99$, $p < .001$, $\eta^2 = .08$
Guilt	To learn from one's role in current situations	Romantic loss, failure, can't cope	Yes, $t = 5.09$, $p < .001$, $\eta^2 = .06$	Yes, $t = 6.88$, $p < .001$, $\eta^2 = .10$
Rumination	To analyze current situations to avoid similar future situations	Romantic loss, failure, can't cope	Yes, $t = 2.45$, $p = .015$, $\eta^2 = .01$	Yes, $t = 5.41$, $p < .001$, $\eta^2 = .07$
Anhedonia	To decrease approach behaviors	Failure, can't cope, winter	No, $t = -3.12$, $p = .002$, $\eta^2 = .02$	No, $t = -1.64$, $p = .101$, $\eta^2 = .01$
High appetite	To increase calories	Winter	No, $t = 1.75$, $p = .110$, $\eta^2 = .01$	No, $t = 0.06$, $p = .951$, $\eta^2 = .00$
Sleepiness	To conserve energy	Winter	No, $t = 0.796$, $p = .426$, $\eta^2 = .00$	Yes, $t = 3.02$, $p = .003$, $\eta^2 = .02$

[a] For *t* tests, the degree of freedom is 412. [b] For *t* tests, the degree of freedom is 411.

to all 11 depressive symptoms, and we then dropped pathways that did not reach marginal significance ($p < .10$) one at a time, rerunning until all such pathways had been dropped. The final model (see Figure 2) shows that Failed Effort related significantly to (in order of strength of association) Guilt, Rumination, Pessimism, Fatigue, Anxiety, Sleepiness, Anhedonia, and Emotional Pain. Social Loss, on the other hand, related significantly to Desire for Social Support, Crying, and Emotional Pain and was negatively associated with Guilt. The fit of this final model was almost perfect, $\chi^2(9) = 7.29$, $p = .61$ (CFI = 1.00, TLI = 1.00, RMSEA = .00) because all 9 degrees of freedom came from pathways that were dropped due to being nonmarginally significant in previous models. In a second model controlling for three likely mediating variables, we essentially replicated these results (the Failed Effort–Anxiety pathway was nonsignificant), indicating that Shamed, Suddenness, and Lack of Control do not mediate the relationships in Figure 2.

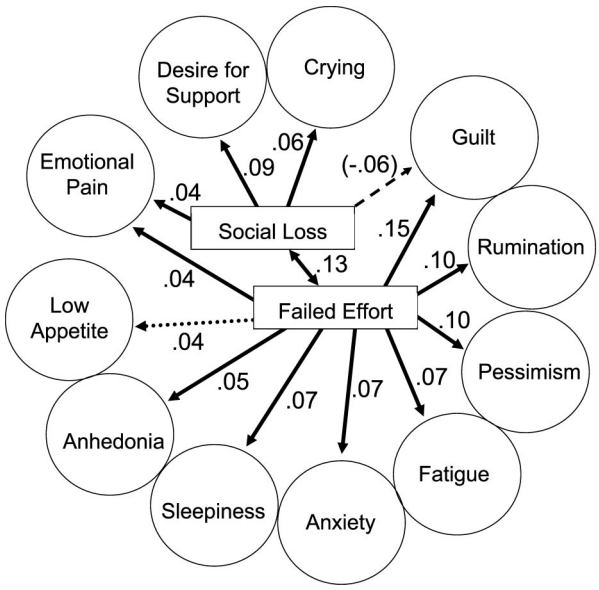

Figure 2. Structural equation model relating 11 depressive symptoms to the degree to which social losses and failed efforts played roles in causing the depressive symptoms. Path coefficients for the bold, dotted, and dashed pathways are significantly positive ($p < .05$), marginally positive ($p < .10$), and significantly negative ($p < .05$), respectively. Pathways that were not marginally significant ($p > .10$) were dropped from the model.

Study 3: Depressive Symptoms Following Random Assignment to Imagined Precipitants

Study 2 found that retrospective reports of depressive symptom patterns differed depending on the precipitant in ways consistent with predictions based on a functional hypothesis of depressive symptoms. However, these data could not assess the direction of causation. People who are characteristically fatigued and pessimistic may be more likely to fail at goals, for example. Study 3 attempted to control for such third-variable and reverse causation explanations by an experimental manipulation using imagined precipitants.

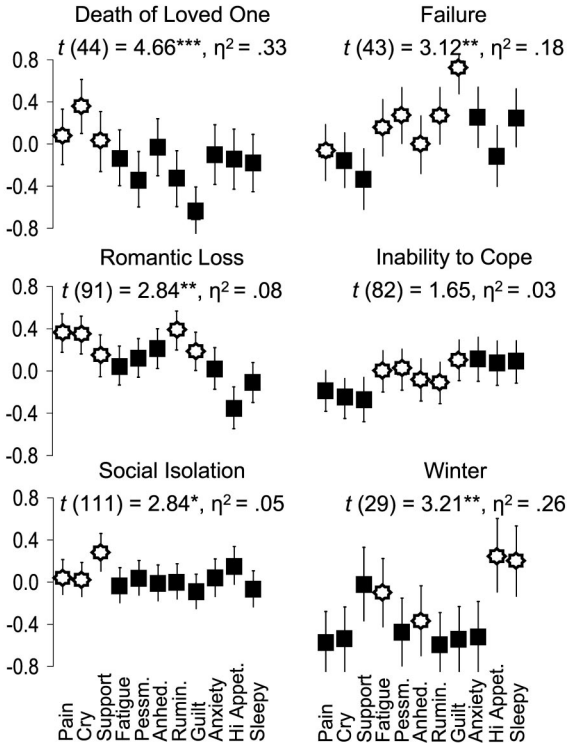

Figure 1. Mean levels (boxes and stars) within each precipitant (Low Appetite was reversed for visual clarity). Stars represent symptoms predicted to be prominent in that precipitant, according to the situation–symptom congruence hypothesis. Error bars represent 95% confidence intervals. Repeated measures contrasts (*t* tests) compare the mean of the predicted symptoms with the mean of all other symptoms for each precipitant. Pessm. = pessimism; Anhed. = anhedonia; Rumin. = rumination; Hi Appet. = High Appetite. * $p < .05$, ** $p < .01$, *** $p < .001$.

Method

Participants

Because imagining depressing scenarios has less emotional impact than real-world situations investigated in Study 2, we preselected participants who had a good chance of being emotionally affected by the experimental manipulations. To this end, we prescreened 1,211 introduction to psychology students to identify 509 participants who rated both accomplishments of goals and personal attachments as being important or very important and who had not participated in Study 2. Of the 353 invited to participate, 129 responded, and 116 (90%) completed the study for course credit. Of these participants, 64 were female, 52 were male, and ages ranged from 18 to 22 years ($M = 18.5$, $SD = 0.93$).

Procedure

Participants completed the survey over the Internet at a private location (usually at home) between 10 a.m. and 7 p.m. After reading the consent form and filling out a demographic questionnaire, participants clicked on a link that randomly assigned them to either identify their most important goal over the next 3 years (failure condition, $n = 60$) or to identify the person whom they felt closest to (death condition, $n = 56$). We dropped 1 participant in the failure condition and 2 participants in the death condition because they indicated on a probing question (see *Measures*) that they had

not taken the task seriously. Participants in the death condition read the following instructions:

> The first task of this experiment will be for you to write a fictional first-person story. The reason for writing this story is to induce an emotional reaction in you, so we encourage you to let yourself go emotionally and to allow yourself to feel any and all emotions that writing about this event elicits. In particular, we would like for you to imagine that you receive news in November that the person whose initials you placed in the box above has been diagnosed with brain cancer. Over the next few months the doctors try several promising procedures. However, by March, it becomes clear that things are not going well, and in late March this close person to you dies. Keep the following in mind as you write: (a) Write the story in the first person, and be as descriptive as possible. (b) The story should begin by describing the person you placed in the box above, why this person is important to you, and what you and this person have been doing (in the fictional future) together. The story should end in April, 2 weeks after you have learned of this person's death. (c) Make the story as realistic as possible—something that could actually happen in the future. (d) Try to cultivate the emotional reaction that your story elicits.

Instructions for the failure condition were similar, except that aspects related to the loved one were replaced by the important goal that participants had identified, and aspects related to the death were replaced by a definitive failure at the goal. Enough clarifying information was given to ensure that the failure participants only identified goals that they could conceivably fail at over the next few years (e.g., abstract goals, such as "world peace," were not allowed). The time frames were made explicit (the death/failure occurring 6 months after the stories began) to remove this as a potential confound. All participants were asked to write their fictional account until the input box was full (about 350 words). Participants then completed a modified DSS questionnaire and answered two probing questions. Participants were fully debriefed on the final page of the study. The study took 15–40 min to complete.

Measures

Depressive symptom scores. The DSS item wordings, instructions, and response format were altered for Study 3. DSS items were worded in the present tense. Questions from the Rumination, Sleepiness, and Low Appetite scales—symptoms unlikely to meaningfully change over the brief course of this study—were omitted. Likewise, the intensities of several questions were altered when judged necessary (e.g., "I feel like I could cry really hard" rather than "I cried really hard"). The modified DSS instructed read: "Think carefully about how you actually feel *right now* compared to how you felt on average today. We are interested in what types of feelings and emotions that you are experiencing, *not in how you think that you should or would feel*" (italics in original). We also altered the response anchor descriptions (*a lot less than before the study* = 1 to *a lot more than before the study* = 5). Such self-perceived deviations of mood state scales have been shown to be reliable and to correlate highly with repeatedly measured mood states (Eid, Schneider, & Schwenkmezger, 1999). Moreover, self-perceived deviations of mood state scales effectively control for stable interpersonal differences (Eid et al., 1999), which was important given interpersonal differences in baseline symptoms likely to exist in any sample.

Residual factor covariance matrices were not positive definite in CFA models, a common situation when the ratio of sample size to number of ordinal items (113:44) is as low as it is in the present sample (Flora & Curran, 2004). Rather than saving factor scores from Mplus, DSS symptom scales were the standardized sums of relevant items.

Probing questions. Using the same probing question from Study 1, 93% of participants indicated that they took the task "seriously" or "very seriously." We also asked participants to "choose the number that describes how much of an emotional effect writing the story had on you" (*big effect* = 1 to *no effect* = 5; $M = 2.64$, $SD = 1.02$).

Analysis

We found no violations of the assumption of equal variance–covariance matrices. We ran analyses both including and excluding 2 participants who had outlying Mahalanobis distances using the same criterion from Study 2, but because there was little difference between these models, only the models including both are reported. Participants reported being more emotionally involved in writing the death story ($M = 2.3$, $SD = 0.90$) than the failure story ($M = 3.0$, $SD = 1.0$), $t(108) = 3.60$, $p < .001$, so this variable was statistically controlled in analyses. Gender had no additive or interactive effects in the analyses and so was not included as a covariate.

Results and Discussion

The Precipitant × Symptom interaction was highly significant across the eight symptoms assessed in this study, Pillai's $F(7, 105) = 5.97$, $p < .001$, partial $\eta^2 = .29$, indicating that visualizing the death of a loved one led to a different pattern of depressive symptoms than visualizing a major failure. Controlling for how emotionally involving writing the story had been, the effect remained strong, Pillai's $F(7, 103) = 4.02$, $p = .001$, partial $\eta^2 = .22$. The symptom profiles and repeated measures contrast tests for the two conditions are shown in Figure 3. Although both deaths of loved ones and failures were predicted to lead to emotional pain, we predicted that social losses would lead to more emotional pain than would failures. The results of the contrast tests (see Figure 3) support the hypothesis that deaths of loved ones and failures cause patterns of depressive symptoms that are consistent with the SSC hypothesis.

General Discussion

This study tested the hypothesis that depressive symptom patterns differ depending on the precipitating cause in ways consistent

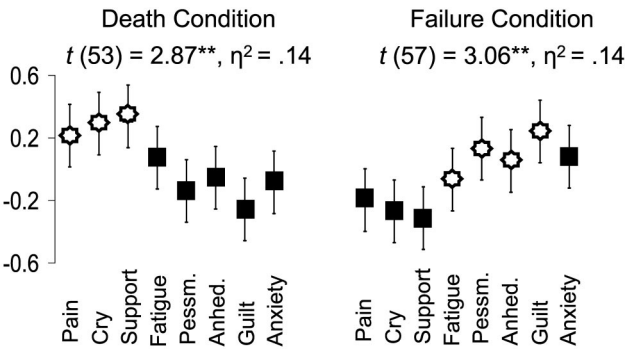

Figure 3. Mean depressive symptom levels of participants randomly assigned to visualize the death of the person they are closest to or the failure at their most important life goal. Stars represent symptoms predicted to be *more* prominent in that condition compared with the other condition. Error bars represent 95% confidence intervals. Repeated measures contrasts (*t* tests) compare the mean of the predicted symptoms with the mean of all other symptoms for each condition after controlling for emotional involvement in visualizing the scenario. Pessm. = pessimism; Anhed. = anhedonia. ** $p < .01$.

with a functional account of different depressive symptoms (the SSC hypothesis). Using the measure of depressive symptoms developed in Study 1, Study 2 found that retrospective reports of depressive symptom patterns matched the precipitants as predicted by the SSC hypothesis. Emotional pain, which makes losses painful and should thereby motivate avoidance of them, was common to all the precipitants except for the winter season, but it was especially prominent following social losses. We hypothesize that this is because social bonds have been especially important to fitness throughout human evolution. Social losses were also strongly associated with crying and a desire to be with friends and family, responses that may help establish or strengthen lost social bonds. Failing efforts were most strongly associated with guilt, rumination, pessimism, and fatigue—reactions that may have been shaped by natural selection to minimize wasted effort and to reassess failing strategies. Anhedonia, fatigue, sleepiness, and (unexpectedly) desire for social support were prominent symptoms of the winter blues. Such "hibernation" symptoms may have protected against starvation and exposure during ancestral winters. Reactions of participants in Study 3, who were randomly assigned to imagine either the death of a loved one or the failure of a major goal, were very similar to reactions reported by participants who actually experienced these situations.

Taken together, these results provide strong evidence that different precipitants cause different depressive symptom patterns, and they are consistent with the hypothesis that depressive symptoms serve situation-specific functions. This supports the more global thesis that depressive symptoms are defensive reactions designed by natural selection to cope with certain kinds of adverse situations. Whether or not one agrees with this interpretation, we hope to have demonstrated that evolutionary approaches can stimulate the formation of testable and useful hypotheses in psychiatry and psychology.

Our findings are relevant to normally expressed depressive symptoms—symptoms that most people would feel in response to adverse situations—and may or may not generalize to depression per se. Nevertheless, we do not think that it is a coincidence that the patterns of depressive symptoms found in our studies resemble several depression subtypes previously uncovered in psychiatric research. Symptoms that we found to be aroused by deaths of loved ones, romantic losses, and social isolation resemble bereavement and share some features with sociotropic depression. Symptoms that we found to be aroused by failures resemble symptoms of depression with melancholia, hopelessness depression, and autonomous depression. Symptoms that we found to be aroused by the winter season are generally consistent with SAD symptoms.

Although we think that finding previously identified symptom clusters in our own data bolsters confidence in our findings, our results are not simply replications of previous findings or confirmations of previous theories of depression subtypes. First, we have investigated a broader array of both situations and symptoms than has previously been done, allowing us to test symptom pattern differences systematically. Moreover, we have introduced a unifying framework that may help explain why particular symptoms often co-occur and that may also provide a novel way to subtype depression based on the precipitating situation. Along with evidence that depressive symptom patterns show little within-person stability (Coryell et al., 1994; Oquendo et al., 2004), and contrary to many previous theories of depression subtypes, our results suggest that situational rather than dispositional factors may be central to explaining symptom pattern differences between episodes.

Limitations

The conclusions from the present set of studies are subject to several limitations. First, the SSC hypothesis did not predict a number of findings (e.g., loss of appetite following romantic breakups), and much variation in symptom patterns therefore remains unexplained. We also recognize that alternative explanations of our results exist, and we hope that such alternatives make new predictions that discriminate between the SSC and alternative explanations.

Second, Studies 1 and 2 used retrospective reports of symptoms and precipitants. Although this is the norm in life events research, including longitudinal research (Kessler, 1997), several studies have found that retrospective reports that were taken as soon as a week after concurrent reports suffer from poor reliability and contamination due to self-enhancement and anchoring biases (e.g., Henry, Moffitt, Caspi, Langley, & Silva, 1994; Smith, Leffingwell, & Ptacek, 1999). It is important to know if the present results replicate when symptoms are measured using daily assessment procedures. Nevertheless, retrospective recall bias was not a limitation of Study 3, which substantively replicated two symptom patterns observed in Study 2.

Third, the self-reported data on the precipitants could have been unreliable or even biased. The single forced-choice response format gave data on precipitant categories that were no doubt less reliable than what could be obtained by extensive life event interviews, such as those collected by Brown and Harris (1978). Moreover, strictly speaking, we investigated the participants' causal *attributions* and not necessarily the true causes of their depressive symptoms. Although one can argue that attributions are the most relevant criteria for testing our hypothesis, we cannot rule out the possibility that experiencing certain symptoms altered the participants' causal attributions. Once again, however, this limitation does not apply to Study 3.

A fourth limitation, potentially more important because it also applies to Study 3, is that the self-reported data on *symptoms* could have been biased (see, e.g., Rottenberg, Gross, Wilhelm, Najmi, & Gotlib, 2002). In our studies, participants may have been more likely to remember or incorrectly report certain symptoms in conjunction with certain precipitants. For example, participants might associate the death of a loved one with crying because crying behavior is intertwined with the memory of a funeral. Although the vast majority of research on depression and affect involves self-report data, different methods of data collection would be required to address this issue.

Fifth, self-report of certain symptoms may necessarily overlap with self-report of certain precipitants. For example, reporting that social isolation caused the depression is not much different from reporting a desire for social support during this period. Similarly, failing at a goal may have been perceived as being similar to certain pessimism items. Nevertheless, inspection of the symptom profiles makes this an unlikely explanation for the totality of our results.

Sixth, the present research was conducted on student samples and may not generalize to other populations experiencing depres-

sive symptoms. Failures to replicate these findings in different age groups or cultures using the types of symptoms and precipitants investigated here might indicate that our findings were somehow unique to student populations and would be problematic for our evolutionary hypothesis.

Future Research

This research needs to be replicated in different populations and cultures. Such studies could also investigate depressive symptom patterns for several precipitants predicted by the SSC but not investigated in the present study, such as postpartum depressive symptoms, physical illness, and being shamed. For example, both epidemiological and experimental studies show that the body's own defensive response to infections—specifically, cytokines secreted by immune system cells—can cause depressive symptoms (Schiepers, Wichers, & Maes, 2005; Yirmiya et al., 2000). The SSC hypothesis predicts that symptoms related to reduced energy expenditure, such as fatigue and anhedonia, will be prominent during illnesses or following cytokine administration, whereas other depressive symptoms, such as crying, emotional pain, guilt, and rumination, will be much less prominent.

Tests of SSC predictions are but one way to assess the more global hypothesis that depressive symptoms are functional. A seemingly more direct test would be to measure whether depressive symptoms increase fitness or lead to positive outcomes. However, such investigations would likely be inconclusive. First of all, fitness in modern environments, replete with birth control, medication, and other evolutionary novelties, may correlate poorly with ancestral fitness, which is the relevant criterion (Tooby & Cosmides, 1990). More important, depressive symptoms are only hypothesized to be useful *given already adverse situations*. Comparing the outcomes of people suffering from depressive symptoms with those not suffering them would be as meaningless as comparing the outcomes of people suffering from fever with those of healthy controls. Virtually any biological defensive reaction would appear maladaptive by such a standard. The correct comparison would be between people who did and did not have depressive symptoms in the same adverse situation, but correctly equating adversity across situations may be devilishly difficult.

Another obvious next step in testing the SSC hypothesis is to investigate whether depressive symptoms have the effects hypothesized. For example, do fatigue, anhedonia, and pessimism reduce motivation, goal pursuit, and energy expenditure? Second, in the same way that blocking fever may prolong infections (Nesse & Williams, 1994), blocking normal depressive symptoms with antidepressant medication could increase the risk of chronic negative life situations or poorer outcomes in such situations, even as the sufferers feel better. Similarly, individuals who lack a capacity for depressive symptoms (who have *pathological euthymia*) should be more likely to lose valuable attachments, more likely to persist at unachievable pursuits, less able to learn from mistakes, and less able to recruit friends during adverse situations.

Conclusions

Researchers and clinicians routinely presume that depressive symptoms, or at least extreme ones, are maladaptive. However, many aversive biological defenses, such as pain, are highly functional, in part *because* they are aversive. The fact that they cause disability and death does not undermine this argument; diarrhea is a useful defense that nonetheless is related to thousands of deaths each years. We propose that the genes of those ancestors who responded to deaths, failures, and losses with indifference tended to be displaced by the genes of those ancestors who responded to these precipitants with emotional pain, crying, anhedonia, guilt, pessimism, fatigue, and rumination. Such depressive symptoms appear to be neither abnormal nor spontaneous; in our study, 42% of college undergraduates reported experiencing them in the previous year, and 92% identified a specific cause. The patterns of depressive symptoms depended on the precipitating situation in a way consistent with the hypothesis that depressive symptoms serve specific functions during adverse situations.

Depending on the situation, some or even many episodes of depression may be normal reactions to highly adverse situations. Individual differences in tendencies to get depressive symptoms may have the same significance as variations in tendencies to get a fever during a cold. This in no way implies that depression is "good" or that treating it is "bad." Patients wanting treatment may not care, understandably, that depressive responses to adverse situations helped their ancestors survive and have offspring. Moreover, the desire to support friends, loved ones, and (in modern environments) patients in times of need—and to extricate them from adverse situations—may be as natural and adaptive as the depressive symptoms themselves. While an evolutionary approach raises questions about the wisdom of routinely blocking depressive symptoms as opposed to treating their causes, the scientific basis for distinguishing pathological from useful depressive symptoms will require a much better understanding of how they were shaped by natural selection.

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