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Research report

Is low mood an adaptation? Evidence for subtypes with symptoms that match precipitants

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Abstract

Background: Although severe depression is dysfunctional, the capacity to experience normal low mood may be useful in certain fitness-threatening situations. Moreover, if specific kinds of situations recurred often enough in the course of evolution, natural selection may have shaped partially differentiated subtypes of low mood that are parallel to the subtypes of anxiety that protect against different kinds of danger. To test this hypothesis, we examined how symptoms of low mood differ depending upon the precipitating situation, and whether these differences match expectations of symptoms useful in each kind of situation.
Method: 337 subjects who experienced a period of low mood within the last year wrote accounts describing perceived causes of their low mood and they filled out the CES-D depression inventory. Seven symptom scales were derived from analysis of CES-D data. Independent judges blindly coded the accounts into one of six precipitant categories.
Results: Different untoward situations were associated with different symptoms that were predicted to be useful in those situations. Social losses (death of a loved one, romantic breakups, and social isolation) were associated with greater crying and arousal. Failure to reach a goal, stress, and winter seasons were associated with more fatigue and pessimism.
Discussion: These results suggest that natural selection shaped not only a generic state of low mood but also partially differentiated subtypes shaped to cope with specific situations that were associated with fitness losses among our ancestors.

Keywords: Depression subtypes; Evolution; Natural selection; Evolutionary psychology; Mood

There has been much debate about whether low mood is an evolved adaptation, what functions it might serve, and what kinds of evidence would test hypotheses about these functions. Most researchers who think low mood may be adaptive argue for a global function of low mood, such as a signal of submissiveness following a loss of status (Price et al., 1994), a strategy to conserve energy and resources (Engel, 1980; Beck, 1996), a way to reassess failing plans (Gut, 1989; Watson and Andrews, 2002), or as a means of social communication (Klerman, 1974; Watson and Andrews, 2002). We and others have

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suggested that natural selection may have shaped capacities for low mood to cope with fitness-relevant situations that have been recurred over evolutionary history-specifically, those that involve losses or efforts that are not paying off (Klinger, 1975; Carver and Scheier, 2001; Nesse, 2000). Less often, researchers have sought specific functions of subtypes of low mood that follow specific precipitants, such as depression following childbirth (Hagen, 1999) and depression following social versus material losses (Gilbert, 1992).

These differing hypothesized functions of low mood are not mutually exclusive. Low mood may be both a global response to a wide variety of situations that represented potential fitness threats in ancestral environments, and a set of more specific responses shaped by natural selection to cope with the particular adaptive challenges posed by different types of situations. If depression symptoms differ depending on the precipitating circumstances in ways that match functional expectations, this would support the more general thesis that low mood is an adaptation. It would also provide a new way to view subtypes of depression based on precipitants and context.

1. Low mood versus depression

An evolutionary view of psychopathology suggests the core importance of distinguishing defects from symptoms that are normal and useful (even if aversive). Fever, nausea, and somatic pain, for example, are aversive states that nonetheless serve important adaptive functions (Nesse and Williams, 1994), while chronic pain syndromes arise from defective neural mechanisms. Because the word depression usually refers to pathology, we use the more neutral phrase "low mood" to describe the cluster of symptoms usually associated with depression—depressed mood, anhedonia, crying, self-reproach, fatigue, pessimism, psychomotor retardation, somatic disturbances, and shifts in cognitive style (Diagnostic Statistical Manual of Mental Disorders, 1994, Fourth Edition). Normal low mood is precipitated by cues associated with fitness losses in ancestral environments, it subsides once the situation is dealt with, and its severity is proportional to the seriousness of the precipitant. As with fever or somatic pain, we hypothesize that normal low mood evolved as a response that is protective in certain kinds of fitness-threatening situations.

Depression, as typically used in the literature, refers to what we would term "extreme low mood". Depending upon the context, extreme low mood could be normal and adaptive or it could be pathological (Wakefield, 1997). For instance, in a hypothetical example (Licinio et al., 2002), a young woman catches her fiancé cheating on her and breaks up with him. For 2 weeks she cries, feels horribly sad, pessimistic, guilty, and has difficulty eating and sleeping, then her behavior returns to normal. Although this woman would receive a diagnosis of major depressive disorder by DSM-IV criteria, such behaviors are probably normal and (as argued below) adaptive given her situation. If, on the other hand, the woman experienced the same symptoms without a precipitant, her behavior would be pathological. Instead of an arbitrary cutoff between normal and pathological low mood based on severity and duration alone, this approach takes context into account in the same way that general medicine uses the context to determine when pain or immune responses are normal and when they are pathological.

The utility of low mood is more apparent when considering the normal human reaction to ordinary events rather than extreme reactions. Nevertheless, because most research is on depression instead of ordinary low mood, we briefly review relevant depression findings.

2. Causes and subtypes of depression

It is increasingly clear that situational factors play a large role in depression etiology. Brown and Harris (1978) estimated that 83% of depressive episodes were preceded by one or more severe negative life events. Relevant to the present research, depressive symptom patterns show little within-person stability across time (Coryell et al., 1994; Oquendo et al., 2004), implying that mood symptom patterns are more governed by situational than interpersonal differences. Consistent with this, some evidence suggests that negative events perceived as being global and stable are associated with specific kind of symptoms: lack of motivation, fatigue, psychomotor

retardation, sleep disturbances, sadness, and poor concentration (Alloy et al., 1997, Joiner, 2001). Similarly, Beck's (1987) hypothesis of sociotropic depression suggests that social losses among people high in need of approval are associated with feelings of loss, loneliness, crying, and lability (Robbins and Luten, 1991).

The DSM-IV distinguishes only two subtypes of depression on the basis of their precipitants—bereavement and seasonal affective disorder (SAD). Typical bereavement symptoms include crying, agitation, sense of loss, rumination, and somatic symptoms. However, there is controversy about the overlap between normal bereavement and depression, and the DSM appears poor at discriminating between the two (Gilbert, 1992). SAD tends to be characterized by 'atypical' symptoms, such as increased appetite and longer sleep duration (Rosenthal et al., 1984) in addition to 'typical' depressive symptoms (sadness, fatigue, pessimism). Some degree of SAD symptoms occur in the majority of non-depressed people in northern latitudes during the winter (Dam et al., 1998), implying that SAD is one extreme along a continuum of normal wintertime behavioral changes.

3. Evolution and subtypes of low mood

Low mood exhibits several hallmarks of a reaction that has been sculpted by natural selection. First, although the prevalences vary dramatically, extreme low mood is observed in most, if not all, cultures (Hill and Martin, 1997; Howell, 1979; Andrade et al., 2003; Weissman et al., 1996). Second, a reanalysis of Brown and Harris' (1978) original data suggested that severe enough situations cause severe low mood (depression) in almost anyone (Monroe and Simons, 1991). Third, low mood is expensive, complex, and carefully regulated, and thus unlikely to be a mere byproduct. Fourth, depression rates, and presumably therefore low mood rates, reach their highest prevalence during the early reproductive years (Kessler et al., 1993), a pattern most unlike the vast majority of diseases (Nesse, 2000). Finally, like fever and nausea, the situations that arouse low mood–deaths, failures, and so forth–probably represent ancestrally recurrent fitness threats. It would be amazing if natural selection had not shaped specialized states to deal with such recurrent and fitness-relevant situations. Of course, the defining feature of an adaptation is its function. Because low mood is a collection of many different types of symptoms, a functional account of low mood must begin with an understanding of the likely functions of its different symptoms, which we explore below.

(1) *Sadness* should motivate avoidance of actions that might lead to future losses in much the same way somatic pain motivates avoidance of actions that might lead to future tissue damage. People can imagine affective reactions to hypothetical scenarios, so the aversiveness of sadness is also useful preemptively; anticipation of the anguish from losing a child can motivate precautions that prevent the loss. We expect sadness to follow discrete fitness losses that organisms could potentially avoid.

(2) *Crying*, like other emotional signals, is expressed on the face. It elicits empathy and comforting behaviors in observers (Labbot et al., 1991; Cornelius, 1997), and may help strengthen bonds between people (Frijda, 1986). Thus, we hypothesize that one function of crying in adults is to solicit "help" and to strengthen weakened social networks. We therefore expect more crying when the precipitant involves social losses or a lack of social support.

(3) *Self-reproach* refers to feelings of worthlessness and guilt. Like sadness, it probably motivates avoidance of actions that might lead to similar future scenarios. Unlike sadness, however, self-directed reproach might motivate a mental search for understanding how one's own actions led to the situation, and thus is more likely to occur when the actor is directly at fault. Self-reproach may also signal culpability to others in order to avoid the loss of important social bonds.

(4) *Fatigue* signals depletion of energetic resources and motivates energy conservation. During low mood, the fatigue threshold is lower and the fatigue is not relieved by rest. Fatigue decreases exertion, conserves resources, and reduces goal pursuit in ways that should be adaptive when future effort is unlikely to pay off or when the environment is generally less propitious, such

as might have occurred during the winter among human ancestors.

(5) *Pessimism* is the tendency to think that favorable future outcomes are unlikely. Given that goal pursuit is largely a function of the perceived likelihood of success (Carver and Scheier, 2001), pessimism should diminish initiative and withdraw the organism from efforts towards unreachable goals (Klinger, 1975). Pessimism is adaptive when past failure predicts future failure.

(6) *Changes in appetite* during bouts of low mood are usually characterized by decreased appetite. A temporary decrease in foraging would reduce risk-exposure in the kinds of uncertain situations that arouse many types of low mood. An increase in appetite, on the other hand, might have been adaptive during ancestral winters when food was limited.

(7) *Changes in sleep patterns* usually result in nocturnal wakefulness that could protect against attacks by animals or humans. Although modern life has become relatively safe, dangers from predators were very real for our ancestors.

Several potential objections to these hypotheses deserve consideration. First, changes in appetite and sleep could be epiphenomena without adaptive utility. Sympathetic arousal may be a useful component of low mood, but it may interfere with normal sleeping and eating. Indeed, some evidence suggests that somatic depression symptoms differ qualitatively from other depressive symptoms (Beach and Amir, 2003). Second, the hypothesized function of crying may seem at odds with findings that depressive symptoms are often met with interpersonal rejection (Segrin and Abramson, 1994). However, these findings are for depression, not for normal low mood expressed in appropriate contexts. Finally, recent findings indicate that depressives are no more likely to cry than non-depressives in response to a sad movie clip (Rottenberg et al., 2002). We find it likely that crying in response to another's pain in a movie clip stems from empathy, and differs qualitatively from crying during normal low mood, which is about requesting aid for the self.

Given the hypothesized functions of low mood symptoms, an organism would gain a selective advantage by using environmental cues to more precisely match its mood responses to the specific kind of untoward situation. This is analogous to the body's immune defense system, which uses both general reactions and differentiated components that cope with toxins, viruses, and bacteria. It is even more closely analogous to anxiety subtypes, which appear to be partially differentiated to cope with different kinds of dangers (Marks and Nesse, 1994). Table 1 summarizes the symptoms hypothesized to be functional given the different precipitating situations in the present study.

Table 1
Precipitant categories coded from free responses with expected adaptive symptoms

Precipitant and description of coding scheme	Expected adaptive symptoms
Death of loved one (*n*=34). The death of a family member or close friend.	Sadness, crying.
Romantic loss (*n*=88). Ending romantic union, fear of breaking-up, or unrequited love.	Sadness, crying.
Social isolation (*n*=64). Need for greater social support, not "fitting in", or feeling homesick.	Sadness, crying, self-reproach.
Failure at an important goal (*n*=33). The past and likely future failure of a goal.	Fatigue, pessimism, self-reproach, sadness.
Stress (*n*=38). An inability to cope with many things going wrong or anxiety about the present or future.	Fatigue, pessimism, disturbed sleep.
Wintertime (*n*=33). Self-report that wintertime instigated the low mood.	Fatigue, pessimism, high appe tite, more sleep.
Other cause (*n*=44). No precipitant (*n*=22) or specific precipitant other than one of those above (*n*=22).	–

4. Method

4.1. Sample

We prescreened 2791 undergraduate students (57% female) for the occurrence of a 2-week period of low mood during the previous 12 months. In all, 920

students (33%) reported they had experienced a period of low mood in the previous year. We invited 542 of these students to participate for course credit. Asking participants what they thought caused their low mood allowed us to over-sample participants who had experienced less common precipitants to assure adequate sample sizes for each precipitant. Three-hundred and seventy-six participants agreed to participate and 337 (90%) completed the study. Participants had a median age of 20 years and were 62% female.

4.2. Procedure and measures

The survey was conducted over the internet from a private location (usually at home) and took 25–45 min to complete. Participants read the consent form, filled out a demographic questionnaire, and identified the weeklong period during which they felt the "most down, sad, or disturbed" in the last 12 months. They then wrote a free-format paragraph about what, if anything, they thought caused this period of low mood. The first author and two research assistants independently coded these paragraphs into one of the seven categories shown in Table 1. Because participants were asked not to write about symptoms associated with their low mood during this phase, raters were blind to the participants' symptoms. Raters agreed on the same category 73% of the time. When exactly two raters agreed (23% of cases), the majority category was used. Situations coded differently by every rater (4% of cases) were included in the "other" category.

Participants then completed the 20-item Center for Epidemiologic Studies Depression Scale (CES-D; Radloff, 1977) based on how they felt during the weeklong period when they felt the worst. The CES-D, often used to assess mood in non-clinical populations, was reworded for this study to be in the past tense. The symptom scales derived from analysis of CES-D data are shown in Table 2. Single item symptoms scales are simply the item in question, standardized. Otherwise, symptom scales were the standardized sum of relevant scores after reversal of necessary items. We used face validity to inform the creation of the symptom scales rather than a statistical method because many of the interesting symptoms–crying, pessimism, appetite change, and sleep disturbances–are measured by single items in the CES-D.

Table 2
Items from CES-D summed then standardized to create symptom scores

Symptom scale	Items
Sadness	CES-D 3, "I felt that I could not shake off the blues even with the help of my family or friends", CES-D 6, "I felt depressed", CES-D 18, "I felt sad"
Crying	CES-D 17, "I had crying spells"
Self-reproach	CES-D 4[a], "I felt I was just as good as other people", CES-D 9, "I felt my life had been a failure"
Fatigue	CES-D 7, "I felt that everything I did was an effort", CES-D 20, "I could not get going"
Pessimism	CES-D 8[a], "I felt hopeful about the future"
Low appetite	CES-D 2, "I did not feel like eating, my appetite was poor"
Sleep disturbance	CES-D 11, "my sleep was restless"

[a] Items reverse scored.

The fact that different numbers of items were summed for the different symptom scales should add random noise to the results, degrading test sensitivity, but should not introduce systematic biases. We did not use nine items from the CES-D because we had no functional predictions about these symptoms or because they seemed more likely to be causes (i.e., precipitants) rather than consequences of low mood.

4.3. Data analysis

The first six precipitants shown in Table 1 served as the (between-subjects) independent variables. The seven low mood symptom scales from Table 2 served as the (within-subjects) dependent variables. The precipitant-by-symptom omnibus interaction term from a MANOVA profile analysis tested whether the patterns of the low mood symptoms differed depending upon the precipitant of the low mood. We used six planned interaction contrasts (Tabachnick and Fidell, 2001), one for each precipitant, to test if the observed symptom profiles were consistent with the predicted symptom profiles. Interaction contrasts compare the precipitant in question versus all other precipitants on the predicted symptoms for that precipitant versus all other symptoms. Because these were planned contrasts, we did not use a redundancy correction. Using conservative criteria, we did not detect multivariate outliers or heterogeneity of the variance–covariance matrixes.

While the present methodology makes it impossible to state conclusively that the precipitants caused the symptom patterns, we statistically controlled for several obvious non-causal explanations through the use of covariates (see below). Age was not a covariate because of the narrow age range (18 to 26) in the sample. None of the conclusions substantively changed in a supplementary analysis in which we omitted all covariates.

5. Results

Of the 920 prescreened participants who had experienced low mood, 93% indicated that it was caused by a specific precipitant. Controlling for gender, weeks since the precipitant occurred, number of times depressed in the past, antidepressant usage, and mood in the last week, different precipitants led to different reported levels of overall low mood, $F(5, 276)=2.99$, $p<0.05$. About 5% of the variance in overall low mood was explained by different precipitants (partial $\eta^2=0.051$). The seemingly small amount of variation predicted by different precipitants probably underscores (a) the degree of heterogeneity within precipitants (e.g., the "death of a loved one" category included deaths of parents and deaths of friends), (b) the variation in how similar situations affected different people, and (c) the noise in our symptom measures. Failure at a goal caused the most

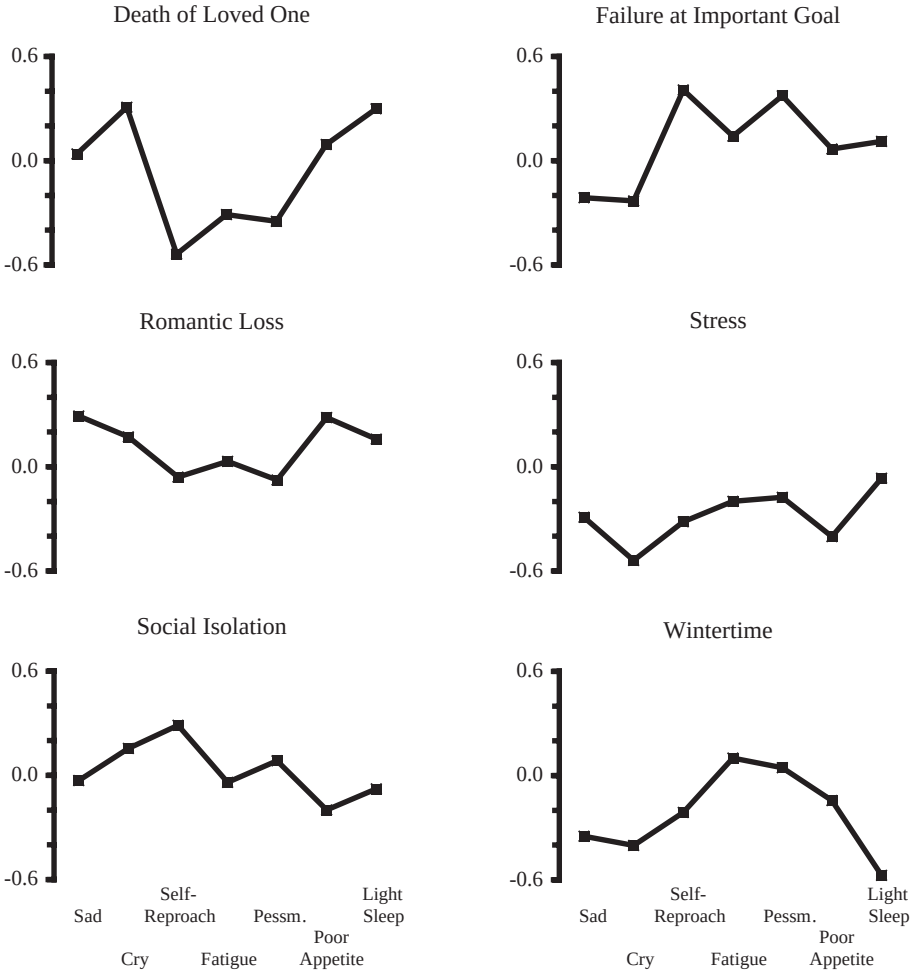

Fig. 1. Low mood symptom profiles across the six precipitants. Scales are standardized.

severe overall low mood (covariate-corrected standardized mean, $\bar{x}$=0.15) followed by a romantic breakup ($\bar{x}$=0.10), social isolation ($\bar{x}$=−0.02), the death of a loved one ($\bar{x}$=−0.08), stress ($\bar{x}$=−0.18), and wintertime ($\bar{x}$=−0.23). These differences should be interpreted in the context of the demographics of this population. For instance, at age 20, a romantic loss may have greater impact than the death of a grandparent.

Although 72% of participants in the present study had CES-D scores above the depression threshold of 27 (Roberts et al., 1991), considerations of the likely depression base-rate among our sample and the specificity and sensitivity of the CES-D suggest that less than 40% of our sample was clinically depressed during the week in question (from Baye's formula; details available from the authors upon request). It is important to note, therefore, that our results are most relevant to normal low mood and not necessarily to clinical depression.

The central focus of the present study is on the symptom patterns following precipitants. Controlling for the same variables as above as well as for overall low mood (the participant's CES-D score), the precipitant-by-symptom interaction term was highly significant using Wilk's approximation to the F, Wilk's F (30, 1082)=3.23, p<0.001, indicating that the patterns of low mood symptoms differed by precipitant. The size of this effect suggests that the amount of variance of low mood symptom *patterns* explained by precipitants (partial η^2=0.066) is comparable to the amount of variance of overall low mood symptoms explained by precipitants (partial η^2=0.051). Fig. 1 presents the symptom profiles associated with the six precipitants not corrected for overall low mood so that overall levels of low mood can be compared.

We performed six planned interaction contrasts (covariate corrected) to test whether the symptom levels differed within each of the six precipitant categories in the ways predicted from the functional considerations in Table 1. For all of the precipitants except for stress, the predicted symptom patterns were observed. Consistent with expectations, crying and sadness were prominent following deaths of loved ones, Wilk's F (1, 275)=14.03, p<0.001, η^2=0.05, and romantic losses, Wilk's F (1, 275)=7.02, p=0.000, η^2=0.03. Crying, sadness, and self-reproach were prominent following social isolation, Wilk's F (1, 275)=5.20, p=0.02, η^2=0.02. Self-reproach, pessimism, and fatigue were prominent following failed goals, Wilk's F (1, 275)=11.9, p<0.001, η^2=0.04. Also as predicted, participants who felt down in relation to the winter season were fatigued and pessimistic and showed higher levels of sleeping and eating, Wilk's F (1, 275)=15.7, p<0.001, η^2=0.05. This finding is consistent with the symptom profile of SAD. Only the symptom profile following stress was different than predicted, Wilk's F (1, 275)=1.90, *ns*, which is not surprising given that this precipitant category was the most heterogeneous (see Table 1).

6. Discussion

This research follows in the footsteps of many previous studies that have found that precipitants play a large role in the risk for developing depression (Brown and Harris, 1986; Kessler, 1997). To our knowledge, however, this study is the first to compare symptom profiles across a wide range of precipitating situations based on functional predictions. Moreover, this study focused on normal human reactions following adverse situations, with no attempt made to screen participants based on severity or duration of their reactions. The results indicate that the symptoms of normal low mood differ depending on the kind of situation that precipitated the low mood. These symptom differences correspond to those we might expect if the symptoms were partially differentiated to respond to the specific situations that recurred during our ancestral past. This "solution-to-problem" structure in nature suggests adaptive design.

Precipitants directly related to social losses engendered much higher levels of crying—useful among our ancestors, we think, in the process of building or strengthening social ties to replace lost ones. This effect remained if we controlled for differences in overall low mood intensity. Fatigue and pessimism, on the other hand, were relatively higher in response to failure, stress, and wintertime. In our ancestral past, we hypothesize that these responses conserved energy and decreased initiative because vigor and ambition were maladaptive on average during such times. This type of low mood, characterized by fatigue and

pessimism, shares features with endogenous/melancholic depression.

This study has several limitations. First, additional data are needed to confirm that the results generalize beyond the student population studied. Second, self-report data is subject to demand characteristics. It may, for instance, be socially acceptable to report crying after a death or a breakup but not after failing to get into business school. Third, like the great majority of studies on the precipitant–depression link, both "reverse causation" and "third variable" explanations are possible alternatives to the observed symptom–situation associations (Kessler, 1997). We controlled for third variables that we thought could be important, but others could not be controlled so easily. For example, pessimism may cause failures rather than vice versa. Furthermore, memory biases could have distorted the true symptom patterns. For example, sadness following discrete events (such as a romantic breakup) may be easier to remember than sadness following protracted events (such as stressful events). For these reasons, the present findings, while promising, should be considered preliminary.

The hypothesis that low mood is an adaptation raises the question of why depression (extreme low mood) is so prevalent. When depression is caused by an extreme situation, the situation is so abnormal that it is difficult to tell if the reaction is adaptive, albeit extreme, or merely maladaptive. Additionally, as with many defenses, low mood may be activated more often or more intensely than seems necessary because false alarms are often less costly than failing to respond when a response was warranted (Nesse, 2001). Furthermore, low mood evolved to deal with contexts much different than modern ones, which may explain why much normal low mood may nevertheless be maladaptive now (Glantz and Pearce, 1989). Finally, mood regulation mechanisms can be disrupted by many factors including toxins, injuries, extreme experiences, and especially genes. A robust finding from evolutionary genetics is that traits influenced by many genes tend to show high levels of maladaptive genetic variance (Houle, 1998; Charlesworth and Hughes, 1999). If mood reactions are influenced by many genes, recurrent mutation could lead to dysfunction of mood regulation mechanisms (Keller and Miller, 2004).

The capacity for normal low mood is, we contend, as adaptive as the capacity for normal pain. The finding that specific low mood symptoms are most frequent and intense in the kinds of situations where they are likely to be most useful is consistent with the hypothesis that selection has partially differentiated subtypes of low mood to cope with different kinds of untoward situations. The existence of these predicted subtypes provides the strongest evidence to date that low mood is a useful defense shaped by natural selection.

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