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Depressive Symptomatology, Rather than Neuroticism, Predicts Inflated Physical Symptom Reports

M. Bryant Howren, Ph.D.,
Center for Research in the Implementation of Innovative Strategies in Practice (CRIISP), Veterans Affairs Medical Center, Iowa City, Iowa

Jerry Suls, Ph.D., and
Department of Psychology, The University of Iowa

René Martin, Ph.D.
Center for Research in the Implementation of Innovative Strategies in Practice (CRIISP), Veterans Affairs Medical Center, Iowa City, Iowa, College of Nursing, The University of Iowa

Abstract

Objective—To examine the roles of depressive symptomatology and neuroticism/negative affect (N/NA) on common physical symptom reporting in a sample of community residents.

Methods—Community-residing adults (N = 108) participated in a combined concurrent-retrospective design. Physical symptoms were assessed concurrently over 21 consecutive days followed by a retrospective assessment of the collective symptom experience for the same time period.

Results—Based upon evidence of differences in cognitive processing of emotion-relevant material, it was predicted and found that depressive symptomatology (at baseline) was a stronger predictor of inflated physical symptom recall than N/NA. Depressive symptomatology was also a stronger, independent predictor of concurrent physical symptoms. Notably, these results were obtained even when depressive symptoms in both the physical symptom checklist and the baseline depression assessment were eliminated.

Conclusions—The results suggest that the classic *symptom perception hypothesis* should be refined and operationalized in terms of depressive symptomatology rather than N/NA. This study demonstrates how cognitive-affective processing differences associated with depressive symptomatology can shed additional light on the psychology of symptom perception. Implications for treatment seeking, medical diagnoses, and treatment decisions are discussed.

Keywords

symptom perception; depressive symptomatology; neuroticism; negative affect

Neuroticism (N) has been defined as “a broad dimension of individual differences in the tendency to experience negative, distressing emotions and to possess associated behavioral and cognitive traits” (1, p. 301). This individual difference has also been referred to as Negative Affectivity (NA;2). Measures of N/NA are moderately to highly correlated with reports of frequent and intense physical symptoms, often in the absence of objective illness (e.g., 1;3–5). The *symptom perception hypothesis* (SPH;1;4) explains this association by proposing that persons who are high in N/NA are more likely to attend to and exaggerate minor somatic

Author correspondence should be sent to Jerry Suls, Department of Psychology, E-11 SSH, University of Iowa, Iowa City, Iowa 52242.

sensations and/or are inclined to misattribute benign or ambiguous physical changes to illness (e.g., 4). This perspective emphasizes N/NA as an interpretative filter in the process of encoding somatic information. The SPH has considerable practical importance if these tendencies lead certain individuals to believe that they are ill when they actually are not, prompt inappropriate self-care, and/or increase unnecessary medical visits (1). In addition, researchers have been encouraged to statistically control for N/NA in self-reported illness outcomes because the SPH conceptualizes N/NA as a confounding variable (4).

The present study was motivated by an empirical fact and a conceptual issue that have not been fully acknowledged in discussions of individual differences in symptom perception. First, although N/NA is considered to represent a broad spectrum of different negative trait affects (2,6)—such as anxiety, depression, and anger the—vast majority of studies reporting significant associations between N/NA and physical symptom reports rely on measures of N/NA that predominately assess anxiety and distress. For example, Watson and Pennebaker (4) used Tellegen's (7) Negative Emotionality Scale (from the Multidimensional Personality Questionnaire), which includes several anxiety and tension items, but has very few items directly tapping depression or anger—two negative emotions also considered to be part of the N/NA spectrum. Several other studies employed the Eysenck Personality Questionnaire (EPQ; 8,9) which also has limited representation of dysphoric and angry affects.[1] The NEO-PI-R (10), which has an N scale consisting of several facet subscales (including anger and depression), has also been used in studies of symptom reporting, but researchers have failed to assess the effects of one facet while statistically controlling for the others (1). Consequently, the exact role of specific negative affects on symptom reporting is unclear.

A second important point is that the SPH stresses how persons differentially attend to and encode somatic changes. However, the associations among N/NA and reports of more frequent and severe physical symptoms are consistently larger for global, retrospective reports (e.g., weeks, months) than for concurrent reports measured during the same day or with experience sampling techniques (11–14). The small to null associations between N/NA and concurrent symptom reports (compared to larger associations with retrospective accounts over weeks or months) suggest that memory bias, rather than attentional or encoding biases, may be the critical mechanism underlying the N/NA-symptom relationship.

Larsen (12) tested this idea by having participants report physical symptoms experienced during an 8-week period in two ways: three times daily and as a single retrospective report. He found only a weak tendency for those high (vs. low) in N/NA (as assessed by the EPQ-R; 9) to report more symptoms on a daily basis, consistent with what others have found for concurrent reports (e.g., 11;13,14). Most notably, persons who were high (vs. low) in N/NA recalled experiencing more symptoms eight weeks later than they had reported concurrently over that same time period. These findings further suggest that recall—rather than encoding—biases may be more critical for the N/NA-symptom association, at least regarding retrospective assessment.

Neuroticism, Depressive Symptomatology, and the SPH

If recall bias is the significant factor contributing to inflated symptom reporting, then we propose that the focus should center on individual differences in depressive symptomatology rather than on the broad dimension of N/NA. Although N/NA subsumes anxiety, depression, and other related negative affects, as noted, anxiety is the trait affect primarily assessed in individual difference measures of N/NA found in the symptom reporting literature.[2] Moreover,

[1]The Neuroticism scale of the EPQ contains 6 (out of 24) items tapping anger and depression. However, anhedonia, for example, is not assessed.

the most popular explanations for the N/NA-symptom relationship found in the psychological literature—such as the SPH—are based on the tendency of individuals high in N/NA to demonstrate heightened attentional vigilance for threatening (somatic) stimuli (4).[3] Experimental evidence indicates that such attentional vigilance, however, is specific to anxiety (16,17). Thus, the predominant view regarding exaggerated symptom reporting is more closely aligned with trait anxiety, rather than trait N/NA as a whole.

As we have noted, a closer look at the empirical literature suggests that an association between N/NA and physical symptoms is more likely to be observed when individuals are asked to report symptom experiences that occurred in previous weeks or months, suggesting that recall biases may contribute significantly to this relationship. Experimental evidence shows that exaggerated recall of negative events and experiences is actually more strongly connected to depressive symptomatology than to anxiety or other negative affects (18).

The Role of Depressive Symptomatology

Depressive symptomatology is characterized by low external engagement, high levels of rumination, and excessive self-focus, whereas the attentional biases associated with anxiety noted above are manifested as a heightened readiness to orient to negative stimuli (19,20). More specifically, individuals high (vs. low) in anxiety are faster at perceiving negatively-valenced stimuli (21) while, in contrast, depressed (vs. non-depressed) individuals exhibit comparable attention to both positive and negative stimuli (22).

In terms of recall, however, the patterns are reversed. Depression is related to greater memory for negative (vs. positive) material, including differential recall of both autobiographical and experimentally-manipulated stimuli (23). In contrast, anxious persons do not show differential recall of positive versus negative stimuli (23,24). Presumably, these information processing differences reflect the anxiety-prone individual's heightened vigilance for threatening stimuli versus the depressed person's rumination and intense self-focus.

We hypothesize that these distinctive orientations may lead to differences in cognitive processing of somatic changes. With respect to judgment and interpretation, both anxious and depressive symptomatology may facilitate misattribution of minor bodily changes to illness because both are associated with threatening interpretations of ambiguous stimuli (25–28). In terms of the heightened attention specific to anxiety, vigilance for bodily changes may be offset by vigilance for external threats. The differential memory biases specific to depressed individuals, however, may have the greatest implications for symptom reporting. In particular, depressive self-focus and rumination should contribute to exaggerated *recall* of past symptoms. This leads to the hypothesis that depressed persons will recall having experienced more symptoms over a specific timeframe than they actually reported on a daily basis.

Depression has been previously acknowledged to be an amplifier of somatic complaints (29, 30), but only one study has reported the independent effects of depressive symptomatology and N/NA on physical symptom recall.[4] Neitzert, Davis, and Kennedy (36) found that depressive symptomatology (as measured with the CES-D;37) significantly contributed to the variance in retrospective symptom reporting even after controlling for the effects of N/NA (as assessed with the EPQ-R). This study was limited, however, by the absence of concurrent

[2] In fact, many have used the terms "trait N/NA" and "trait anxiety" interchangeably (see 2,4).

[3] The medical literature has also focused on how individual differences in attention may explain elevated levels of symptom reporting (e.g., 15).

[4] The empirical literature on N/NA, depression, and objective indices of physical illness (e.g., 31–34) is inconsistent. The status of depression as a risk factor for cardiac disease has considerable support (e.g., 35), but the empirical evidence is considerably weaker and inconsistent with respect to other physical diseases. In any case, the status of depression as a risk factor for physical disease is an independent issue from the question of the role of depression and/or N/NA on biased symptom recall.

symptom reports and lack of statistical adjustment for overlapping physical symptoms in the depression and symptom measures. Another study involving a daily diary procedure in Type II diabetics found that the combination of high N/NA and low positive affect (PA) was the best predictor of physical symptoms(13). This is especially interesting because, according to one theory (38), the combination of high N/NA and low PA defines depression.

A Point of Clarification

Before proceeding, we want to anticipate a possible problem that may occur to the reader. If depressive symptomatology is, in fact, the critical element in inflated recall of physical symptoms—rather than anxiety or other negative affects—then why have past studies found associations between N/NA and physical symptom reporting (e.g., [1],3,4,12)? The answer is two-fold. For some studies, the N/NA measure did include items assessing depressive symptomatology (e.g., 1). More importantly, however, anxiety and depression are moderately to strongly correlated (39). Thus, even with N/NA measures predominantly consisting of fear and anxiety items, associations with symptoms may still be observed because of the indirect association with depression. To evaluate our hypothesis, it is essential to directly examine the separate and simultaneous roles of depressive symptomatology and N/NA on physical symptom reports.

The Present Study

Our predictions were based upon the experimental psychopathology literature on cognitive processing among individuals differing in depressive symptomatology (18). The main study aim was to assess the relative contributions of N/NA versus depressive symptomatology on retrospective accounts of common physical symptoms. Concurrent symptom reports—aggregated over 21 days—should independently predict retrospective symptom recall since participants should have some recollection of their recent symptom experiences. Beyond the contribution of concurrent symptoms, however, depressive symptomatology should subsume the effects of N/NA and independently inflate retrospective accounts of physical symptoms experienced during the previous three weeks. Our design also permitted an examination of the relationships among depressive symptomatology, N/NA, and concurrent symptom reports. As noted earlier, when associations between N/NA and concurrently-reported symptoms are significant, they tend to be small. The literature on cognitive processing differences associated with depression and other negative affects, particularly anxiety, provides no straightforward predictions about concurrent reports, thus, this remains an empirical question.

Methods

Participants

The sample consisted of community-residing females without acute or chronic illness, who responded to an announcement posted on a large university news webpage generally read by non-faculty employees. Those of age 18–55 with daily internet access were invited to respond by email concerning a study assessing “attitudes, beliefs, feelings, and symptoms.”

Participants were recruited from April through June of 2006. Informed consent was obtained from all study participants and all procedures were reviewed and approved by The University of Iowa’s institutional review board. The final sample consisted of 108 non-faculty women (*M* age = 34.47, *SD* = 11.03; range = 19–54) employed by The University of Iowa. Most were full-time employees (*n* = 104; 96.3%). In addition, the majority were white (*n* = 81, 75.0%), married/living with partner (*n* = 62, 57.4%), and had completed at least some college (*n* = 93, 86.1%; see also Table 1). Respondents who reported suffering from chronic illnesses (e.g., diabetes, cancer; *n* = 3) were excluded.

Procedures

Participants gave informed consent and were sent a packet containing individual difference measures and a baseline symptom checklist. After these were returned, participants were provided a website address and log-in instructions in order to begin completing the concurrent symptom checklists.

Symptom checklists were administered via a secure website. Participants were instructed to complete these "as close to the noon hour of each day as possible" for 21 consecutive days. Because participants had reliable internet access during the workday, we collected symptom reports in the middle of each day to facilitate compliance and reliability. Participants received a "reminder" email just before noon each day that contained the link needed for completion. Weekend reports were also requested during the noon hour; however, because weekends are often less structured and participants relied on their internet access at home, the hours that each report could be completed during those days were relaxed (i.e., between 11am and 1pm). Reminder emails were sent out just before 11am instead of just before noon as they had been Monday through Friday. Upon completion of each day's checklist, responses were automatically transferred onto a secure server.

Each day, participants were asked to endorse any of 15 common physical symptoms that they were currently experiencing or had experienced during the previous 24 hours and note any illness (e.g., cold or flu). Once 21 daily symptom reports were completed, a retrospective symptom checklist was completed on day 22. This retrospective report requested information concerning the presence and frequency of each symptom experienced during the previous 3-week period.

Measures

Concurrent symptom assessment—Concurrent symptoms were assessed using a checklist of 15 common symptoms. Participants endorsed (yes/no) symptoms that they were currently experiencing or had experienced during the preceding 24 hours and were asked to note any illness they may have been suffering. This checklist was developed and factor-analyzed by Larsen and Kasimatis (40) and includes symptoms corresponding to four distinct factors: *ache*, *depression*, *gastrointestinal*, and *upper respiratory* symptoms. Individual symptoms were backache, headache, muscle soreness (*ache factor*); loss of interest/bored, low energy/tired, trouble concentrating, urge to cry (*depression factor*); constipation/diarrhea, dizziness, nausea/upset stomach, poor appetite, trembling/shaking (*gastrointestinal factor*); congestion, sore throat, runny nose (*upper respirator factor*).

Retrospective symptom assessment—The retrospective symptom checklist, also developed by Larsen (12), assessed the collective symptom experience during the preceding 3-week period. Participants endorsed any of the 15 common symptoms that they recalled experiencing during the previous 21 days and rated the frequency of each endorsed symptom on a 7-point scale (0 = *not at all*; 6 = *extremely frequent*).

Beck Depression Inventory (BDI;41)—Participants rated the severity of depressive symptoms for 21 items presented in multiple choice format. Each item is representative of a category of depressive symptoms. Items 1–14 have been described as *cognitive-affective* items; items 15–21 have been labeled *physical* (or *somatic)* items (42). The BDI has high internal consistency for both clinical and nonclinical samples (43). In our sample, participants' responses were internally consistent ($\alpha = .82$). Scores ranged from 0–29 ($M = 8.29$, $SD = 6.11$).

Big Five Inventory (BFI;44)—The BFI is a brief, but comprehensive measure of the five-factor model of personality; participants rated 44 items in 5-point Likert format (1 = *disagree*

strongly; 5 = *strongly agree*). Although the BFI was administered in its entirety, the Neuroticism scale (BFI-N) was of particular importance for our study. The BFI-N scale consists of 8 items assessing, for example, anxiety ("Gets nervous easily"), depression ("Is depressed, blue"), and emotional instability (Is emotionally stable, not easily upset [R]). When the BFI-N scale is compared with Costa and McCrae's NEO-Five Factor Inventory N scale (NEO-FFI; 10), convergent correlations reach .90 (44). Further, Cronbach's alpha and three-month test-retest reliabilities of .83 and .85, respectively, have been reported (45). In the current sample, participants' responses on the BFI-N scale were internally consistent ($\alpha = .85$) with a mean of 22.67 ($SD = 6.12$; range = 10–36).

Results

Descriptive Statistics and Correlational Analyses

Tables 2 and 3 present descriptive and correlational information, respectively, for the concurrent (i.e., based on the number of symptoms reported over 21 days) and retrospective symptom totals (i.e., based on frequency ratings). On average, participants experienced symptoms on 13.88 ± 3.53 days. The average number of total concurrent symptoms aggregated across three weeks was 37.61 ($SD = 28.32$; range = 2–122). After depressive symptoms were removed from the concurrent checklist, the average aggregated total was 25.90 ($SD = 19.78$; range = 1–90). Zero-order correlations indicated that BDI and BFI-N scores were positively associated with both concurrent symptom totals and retrospective symptom frequency ratings. The magnitude of these correlations is similar to those reported by others (e.g., [1],4,12). BDI and BFI-N scores were also correlated, $r = .51$, $p < .001$.

Regression Analyses

Both simultaneous and hierarchical regression analyses were used to assess the influence of baseline depressive symptomatology (as assessed by the BDI) and N/NA on physical symptom reports. Symptoms contained in the checklist related to depressive symptomatology (i.e., *depression factor*, see above) as well as the physical symptom items in the BDI (i.e., items 15–21) were removed to ensure the results were not due to overlap between these two measures.[5]

Depressive Symptomatology, N/NA, and Concurrent Symptoms as Predictors of Total Retrospective Symptoms—Table 4 summarizes the standardized coefficients, model R^2, and ΔR^2 for each successive block in a hierarchical multiple regression analysis. In addition to concurrent symptom totals, depressive symptomatology ($\beta = .22$, $sr^2 = .034$, $p < .05$), but not N/NA ($\beta = .09$, $sr^2 = .009$, *ns*), independently predicted retrospective symptom reports. Although prior evidence found N/NA biased retrospective symptom reports (e.g., [1]; 3,4;12), BFI-N scores did not independently predict retrospectively-recalled symptoms. As this seemed inconsistent with previous research, a regression analysis with BFI-N score as the only predictor was conducted. Entered alone, N/NA predicted retrospective symptom reports ($\beta = .20$, $sr^2 = .040$, $p < .05$). Thus, it seems that concurrent symptoms and baseline depressive symptomatology (i.e., BDI Cognitive score) played more substantial roles in the recollection of symptom experience. Further, depressive symptomatology inflated the recollection of symptom occurrence beyond concurrently-reported physical symptomatology.[6]

[5]Regression analyses including these items were also conducted; however, these additions did not alter the results reported here.

[6]An alternative explanation is that more frequent physical symptoms resulted in higher levels of depressive symptomatology, rather than elevated depressive symptoms resulting in inflated symptom reports. However, as the BDI was administered at baseline and the symptom recall measure 21 days later, this does not seem viable. Additionally, supplementary analyses indicated that the frequency of daily physical symptoms did not differ at any point over the 21-day reporting period. Thus, because there was no increase in the level of symptoms reported over time, a greater experience of physical symptoms could not have resulted in increased levels of depressive symptomatology at retrospective recall.

Depressive Symptomatology and N/NA as Predictors of Total Concurrent Symptoms—BFI-N and BDI Cognitive scores were simultaneously entered as predictors of the concurrent symptom totals (Table 5). Depressive symptomatology ($\beta = .23$, $sr^2 = .039$, $p < .05$), but not N/NA ($\beta = .07$, $sr^2 = .003$, *ns*), predicted concurrent symptoms. Thus, although zero-order correlations indicated simple associations of concurrent symptoms with N/NA, depressive symptomatology was the main factor contributing predictive variance.

Nature of Symptom Inflation

These results suggest that individuals with elevated depressive symptomatology inflated the occurrence of their negative somatic experiences. However, there exists another possible, although not mutually exclusive, explanation for the observed symptom inflation. Because those individuals with higher BDI scores also experienced more concurrent physical symptoms (see above), they may have been confused about *which* symptoms they experienced, thereby inflating how *often* each was experienced.

To evaluate this possibility, we compared the percentages of each of the three physical symptom factors reported concurrently versus those reported retrospectively. As expected, individuals with BDI scores of 9 or less were quite accurate in their recollection of previous symptom experiences. Specifically, over the 21-day concurrent phase, these individuals reported 46.0% *aches*, 14.8 % *GI*, and 39.2% *upper respiratory* symptoms. Comparable results were observed for the retrospective report as well—48.4% *aches*, 14.3% *GI*, and 37.3% *upper respiratory* symptoms.

For individuals with BDI scores of 10 or more, the percentages reported during the concurrent period were 44.4% *aches*, 17.4 % *GI*, and 38.2% *upper-respiratory* symptoms.[7] Most interestingly, retrospective reports were similarly distributed—47.5% *aches*, 16.4% *GI*, and 36.0% *upper respiratory* symptoms.[8] To illustrate, if *aches* were the most common physical complaints endorsed over three weeks for someone with elevated depressive symptomatology, then *aches* were also the symptoms whose occurrence were most often inflated at recall.

Thus, individuals both high and low in depressive symptomatology were equally accurate in recalling *what* symptoms they experienced over the concurrent reporting period, but the former inflated the frequency of symptom occurrence.[9] Such results do not support the idea that the symptom inflation was due to confusion regarding the experience of specific types of symptoms.

Discussion

Depressive symptomatology played a more critical role in physical symptom reporting—especially for retrospective reports—than did N/NA. This result was obtained even when overlapping depression symptom items contained in the symptom checklist and the BDI were eliminated. N/NA predicted retrospective symptom reports only when evaluated in isolation (i.e., the usual practice in previous research). N/NA played no predictive role when entered with concurrent symptoms and/or baseline depressive symptomatology in multiple regression analyses.

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[7] A score of 10 or above on the BDI indicates possible mild depression (46).

[8] Comparisons between concurrent and retrospective reports for those scoring both high and low on the BDI were nonsignificant.

[9] It is noteworthy that participants with high BFI-N scores were also similarly accurate in their recall of previous symptoms experiences. Specifically, the distribution of concurrent reports was 47.8% *aches*, 20.5% *GI*, and 31.7% *upper respiratory* symptoms. For retrospective reports, the percentages were 47.2% *aches*, 19.4% *GI*, and 33.4% *upper respiratory* symptoms. Again, all comparisons were nonsignificant.

We had no firm predictions about concurrent reports, but these were also influenced by depressive symptomatology, perhaps because our daily reports captured both contemporaneous symptoms and those experienced during the preceding 24 hours. Because this method taps long-term memory to some extent, these reports may also have been subject to the effects of depression on recall. It is of interest that experience sampling, which measures only symptoms occurring at the time of report, does not yield associations with N/NA (11). This suggests that neither depressive symptomatology nor N/NA may inflate symptom reporting at the level of encoding—a possibility that requires additional research.

The current findings are consistent with experimental studies on differential attentional and recall biases associated with anxiety and depression (18;21). Anxiety directs attention to negative or threatening stimuli, whereas depression potentiates and magnifies the recall of negative experiences. Ironically, external vigilance among persons high in N/NA may prompt them to be less focused on bodily changes. Perhaps depressive symptomatology leads to stronger associations even with respect to concurrent symptom reports because of its characteristic self-focus and rumination.

Our exclusively female sample might be seen as a limitation. In general, women report higher levels of depression and more physical symptoms, even after eliminating menstrual symptoms or complications from pregnancy. However, Neitzert and colleagues (36) found the same pattern of associations with depressive symptomatology, N/NA, and symptom reports in both sexes, although women had somewhat higher depression and symptom scores.

The decision to recruit a population without serious acute or chronic illness was motivated by the need to reduce confounding factors such as symptoms and side effects associated with chronic disease, medication use, medical procedures, etc. This limitation, of course, confines our results to “well” individuals. N/NA and depressive symptomatology might operate differently in the context of serious chronic illness or illness-induced somatic changes, which is a subject for future research.

Some researchers have claimed that greater symptom reporting leads to greater treatment-seeking behavior (1), but empirical support for this assertion has been mixed (e.g., 4). If depression is really the amplifying factor in symptom perception, the passivity, anhedonia, and pessimism characteristic of depression may actually suppress actions such as seeking treatment in some contexts (see 47). This speculation needs empirical validation, but suggests that the classic SPH should be refined and operationalized in terms of depression rather than N/NA.

Although this conclusion applies mainly to the recall of symptoms, it does not limit its substantive or practical implications. For example, our results suggest that health researchers should be more concerned about controlling for the influence of depression, rather than N/NA, in physical symptom reports. In addition, depression is quite common in those suffering chronic illness (48). If patients experiencing depressive symptomatology inflate the duration and/or number of physical symptoms previously experienced, this may create problems for healthcare providers when formulating specific diagnoses as well as decisions regarding the need for specific treatments during clinical encounters. In conclusion, the present results suggest that depressive symptomatology plays a more prominent role in symptom recall than thought heretofore and this has significant implications for patients, practitioners, and health researchers alike.

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Glossary

BDI	Beck Depression Inventory
BFI	Big Five Inventory
CES-D	Center for Epidemiologic Studies Depression Scale
EPQ	Eysenck Personality Questionnaire
N	Neuroticism
NA	Negative Affect
NEO-PI-R	Revised NEO Personality Inventory
PA	Positive Affect
SPH	Symptom Perception Hypothesis

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Table 1

Characteristics of the Sample (N=108)

	Study Sample
Mean age (years)	34.47 ± 11.03
White	75.0%
African American	14.8%
Asian American	2.8%
Hispanic/Latino	3.7%
Other	3.7%
Completed at least some college	86.1%
Income less than 20K	16.7%
Income 20K-40K	44.4%
Income greater than 40K	37.1%
Not reported	1.9%
Married/Living with partner	57.4%
Single	36.1%
Divorced/Widowed	6.5%
Mean BDI Total Score	8.29 ± 6.11
Mean BDI Cognitive Score	4.87 ± 3.93
Mean BFI-N Total Score	22.67 ± 6.12

Note. BDI = Beck Depression Inventory; BFI-N = Neuroticism scale of the Big Five Inventory; BDI Cognitive Score refers to items 1–14 of the BDI and reflects the removal of physical symptom items from the BDI that may overlap with the symptom checklist.

Table 2

Descriptive Statistics for Concurrent & Retrospective Symptom Measures

	M	SD	Range
Overall Concurrent Symptom Total	37.61	28.32	2–122
Concurrent Total (minus Depressive Symptoms)	25.90	19.78	1–90
Overall Retrospective Symptom Recall[a]	17.05	11.97	0–53
Retrospective Recall (minus Depressive Symptoms)[b]	8.53	7.72	0–33

Note. Concurrent Symptom Period = 21 days;

[a]Refers to the average total symptom frequency across all 15 symptoms;

[b]Refers to the average total symptom frequency excluding any depressive symptoms from the symptom checklist.

Table 3

Correlations among the BDI Total Score, BDI Cognitive Score, BFI-N, Concurrent & Retrospective Symptom Measures

	Concurrent	Retrospective
BDI Total	.31**	.39**
BDI Cognitive	.27**	.36**
BFI-Neuroticism	.19*	.20*

Note. BDI Cognitive refers to items 1–14 of the Beck Depression Inventory; BFI-N = Neuroticism scale of the Big Five Inventory. All values reflect the removal of depression symptoms from the symptom checklists. Concurrent and retrospective symptoms were correlated, $r = .58$, $p < .001$.

* $p < .05$;

** $p < .01$

Table 4

Hierarchical Regression: Depressive Symptomatology & Neuroticism Predicting Retrospective Symptoms

	Retrospective Symptoms		
Block and variable entered	**R^2**	**ΔR^2**	**β**
Block 1: Concurrent Symptoms	.339**	.339***	.58
Block 2: BFI-Neuroticism	.348**	.009	.09
Block 3: BDI Cognitive Items	.382**	.034*	.22

Note. β = the standardized coefficient of each variable at the block it was entered. Values for R^2 and ΔR^2 indicate the total variance at each block and the incremental variance at each block, respectively. BDI Cognitive refers to items 1–14 of the Beck Depression Inventory; BFI-Neuroticism = Neuroticism scale of the Big Five Inventory.

* $p < .05$;

*** $p < .001$.

Table 5

Simultaneous Regression: Depressive Symptomatology and Neuroticism Predicting Concurrent Symptoms

	β	t	sr^2
BFI-Neuroticism	.07	0.59	.003
BDI Cognitive	.23	2.01*	.039

Note. β = the standardized coefficient of each variable; BDI Cognitive refers to items 1–14 of the Beck Depression Inventory; BFI-Neuroticism = Neuroticism scale of the Big Five Inventory.

* $p < .05$.