



Feeling your body or feeling badly Evidence for the limited validity of the Somatosensory Amplification Scale as an index of somatic sensitivity

Keith R. Aronson^{a,*}, Lisa Feldman Barrett^b, Karen S. Quigley^a

^aDepartment of Psychology, The Pennsylvania State University, University Park, PA 16802, USA ^bDepartment of Psychology, Boston College, Chestnut Hill, MA 02467, USA

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Abstract

Objective: The Somatosensory Amplification Scale (SSAS) purports to measure the extent to which individuals are sensitive to their bodies. The present study examined the psychometric properties of the SSAS in two studies with university students. **Methods:** Participants completed the SSAS, various cross-sectional measures of somatic and psychological distress, longitudinal measures of somatic symptoms, daily hassles and mood, and participated in a heartbeat detection task (Study 2 only). **Results:** The SSAS was correlated with cross-sectional measures of somatic

symptom reporting, but not with somatic symptoms reported on a daily basis nor with an index of interoceptive sensitivity. The SSAS was also correlated with several indices of general distress including anxious and depressive symptoms, daily hassles, and negative emotionality. **Conclusions:** Taken together, the results suggest that the SSAS is more likely an index of negative emotionality and general distress than a valid measure of somatic sensitivity per se. © 2001 Elsevier Science Inc. All rights reserved.

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Introduction

Recently, Barsky et al. [1–3] introduced the notion of somatosensory amplification as a central predisposing factor in somatization and hypochondriasis. The hypothesis is that individuals who somatize perceive normal bodily sensations as unusually intense, noxious, and disturbing [2]. Compared to those who do not somatize, these individuals are thought to be particularly sensitive to both normative and ambiguous somatic sensations.

The Somatosensory Amplification Scale (SSAS) is a brief self-report questionnaire designed to measure the amplifying somatic style [3]. A number of studies have examined the psychometric properties of the SSAS. In general, the SSAS has demonstrated adequate internal

E-mail address: keith.r.aronson@vanderbilt.edu (K.R. Aronson).

consistency and test-retest reliability [3]. The validity of the measure, however, remains in question.

The SSAS has demonstrated good convergent validity with a number of related self-report measures. For example, the SSAS was a significant predictor of hypochondriacal and somatization tendencies [2–5]. The SSAS has also correlated with self-reported localized upper-respiratory symptoms [1], medical patient ratings of physical discomfort [1], and self-reported bodily preoccupation [3]. Consistent with the notion of somatic amplification, scores on the SSAS converge with the self-report of somatic symptoms and illness concerns [1].

While the SSAS has converged with related self-report measures, it has failed to converge with objective tests of somatic sensitivity. Three studies have examined the extent to which SSAS scores correlate with an individual's ability to sense resting heartbeats [6–8]. SSAS scores did not correlate with ability to detect resting heartbeats and, in one study, were inversely related with sensitivity to heartbeats [8]. A recent study found that

^{*} Corresponding author. Psychological and Counseling Center, Vanderbilt University, 1120 Baker Building, 110 21st Avenue South, Nashville, TN 37203, USA.

although hypochondriacal individuals obtained higher scores on the SSAS than did healthy controls, there were no differences between the two groups in tactile sensitivity [9]. Therefore, it may be that the SSAS actually measures the extent to which a person believes he is somatically sensitive.

The discriminant validity of the SSAS has not been well supported. The SSAS is associated with a number of measures tapping generalized distress. For example, the SSAS was significantly correlated with depressive and anxiety symptoms, hostility, and overall level of psychiatric symptoms [1,3,5,10]. It is important to note that in one study [3], this pattern of correlations only emerged for nonhypochondriacal individuals. A recent study found that neuroticism was the most potent predictor of SSAS scores for men [4], but was not predictive of SSAS scores for women. Another recent study also found that scores on the SSAS were best predicted by measures of depression, anxiety, and alexithymia in chronic pain patients [11]. These findings suggest that the SSAS does not discriminate well between somatic sensitivity, generalized distress, and negative emotionality. Indeed, individuals who are dispositionally vulnerable to experience pervasive distress tend to report more bodily symptoms and illness concerns than do those who are less vulnerable [12–15]. Negative emotionality is correlated with cross-sectional and daily report of somatic symptoms, illness worry, negative mood, psychological distress, and poor stress tolerance [14-18]. Thus, it may be that negative emotionality accounts for much of the demonstrated relationship between the SSAS, symptom reports, and generalized distress. Indeed, the pattern of correlations, which emerges in studies examining negative emotionality with respect to somatic symptoms and psychological distress is quite similar to the pattern that is demonstrated in individuals with an amplifying style [19-21].

The purpose of the following two studies was to examine the construct validity of the SSAS. We examined the convergent validity of the SSAS with self-report measures of somatization tendencies and somatic symptom reporting. This was the first study to examine the convergent validity of the SSAS with daily symptom reports. We also examined the discriminant validity of the SSAS with self-reports of negative emotionality and psychological distress. In Study 2, we included an objective measure of somatic sensitivity in our assessment of convergent validity. We hypothesized that the SSAS would demonstrate reasonable convergent validity with self-report measures, but not with an objective index of somatic sensitivity. Second, we hypothesized that the SSAS would demonstrate limited discriminant validity. Specifically, we predicted that individuals scoring high on the SSAS would also score high in negative emotionality and report a high degree of generalized distress and negative mood.

Method

Participants

Participants in Study 1 were 81 undergraduate students (males = 31, females = 50) enrolled in an introductory psychology course with a mean age of 18.4 years, with a range from 17 to 25 years of age. Over the course of this 60-day experience-sampling study, 33 participants left the study of their own accord, leaving a final sample of 48 (males = 14, females = 34) participants. Participants who left the study did not differ from those completing the study on any of the initial self-report measures, (Wilk's £=.58), P > .20). Participants in Study 2 were male (n=33) and female (n=98) undergraduate psychology students with a mean age of 19.8 years. Sixty-seven participants (22 males, 45 females) left the study of their own accord during the longitudinal portion of the study. This left a final sample of 11 males and 53 females. Participants who left the study did not differ from those completing the study on any of the initial personality or symptom report measures (Wilk's £=1.14, P > .30). Both samples were largely comprised of European Americans.

The substantive dropout rates in both studies were higher than those reported in three recent studies examining symptom-reporting in university students [15,17,18]. The present studies differed from these other studies in several respects. The class grades of participants in two of the aforementioned studies [i.e., Refs. 17,18] were dependent upon their completion of the study. In the present studies, participation was completely voluntary. In another related study [15], the researchers were less stringent in the number of days of participation required for inclusion in their study.

Materials

The Somatization Screening Index (SSI) was used to select participants in the initial screening battery [22]. The SSI is an 11-item scale asking respondents to indicate how many of 11 common physical symptoms they experience.

The SSAS is a 10-item scale designed to measure the extent to which people are somatically sensitive [3].

We measured cross-sectional symptom reports using the Somatization Subscale of the Symptom Checklist-90 (SCL-90) [23] and the Illness Worry Scale (IWS) [24].

The Symptom Interpretation Questionnaire (SIQ) is a 39-item measure of causal attributions individuals make of common somatic symptoms [25]. Three attribution dimensions have been identified: psychological, somatic, and normalizing.

We measured negative emotionality via the Mutidimensional Personality Questionnaire Negative Emotionality Subscale (NEM) [21,26].

We measured psychological distress using the Beck Depression Inventory (BDI) [27] and the Penn State Worry Questionnaire (PSWQ) [28].

Our experience-sampling measures included a 20-item checklist of common somatic symptoms [18], a modified version of the Daily Hassles Scale (DHS) [29], and a modified version of the Positive Affect Negative Affect Scale (PANAS-X) [30]. For the purposes of this study, and as has been done in previous research [31], the DHS was shortened to include only 37 hassles by removing items related to somatic complaint and health issues, leaving items more salient to university students. The modified measure demonstrated acceptable psychometric properties [16]. Following previous research [32], the PANAS-X was modified by adding 28 additional mood terms that allowed us to sample the entire mood circumplex space. The fear, sadness, hostility, and composite negative mood subscales were used in our analyses. In Study 2, the somatic symptom checklist was lengthened from 20 to 24 items to include allergy symptoms. This modification had little effect on the reliability and validity of the measure [16].

Procedure

Potential participants completed the SSI as part of a prescreening conducted with all undergraduate students enrolled in an introductory psychology course. Individuals who endorsed five or more somatic symptoms on the SSI and those who endorsed no somatic symptoms on the SSI were contacted via telephone and asked to participate in a longitudinal study examining the health of university students.

After obtaining informed consent, participants completed the cross-sectional measures. After completing the questionnaire battery, participants were instructed on how to complete and return experience sampling (i.e., daily) measures. The DHS and the Daily Symptom Report were completed once at the end of each day for a minimum of 54 days. In Study 1, the PANAS-X was completed twice per day (morning, evening), while in Study 2, the measure was completed three times per day (morning, afternoon, evening). Completed daily forms were returned three times per week. Each participant attended a final debriefing session and was then excused.

Study 2 extended Study 1 by including a heartbeat detection task, which is described below.

Electrocardiogram (ECG) and heartbeat detection task

Participants were asked to judge whether each tone in a series of 10 tones was coincident with, or delayed from their heartbeats using a modified Whitehead procedure [38]. A tone occurring within 200 ms of the R-spike of the QRS complex was considered a coincident signal, while a tone occurring 500 ms after the R-spike was considered a delayed signal. Each trial consisted of either 10 coincident or 10 delayed signals. The heartbeat detection session consisted of 100 trials (50 coincident, 50 delayed). A signal was considered to be present when the tone was coincident with heartbeat.

Heart rate was recorded using a modified Lead II configuration with Ag/AgCl electrodes attached to the distal right collarbone and lower left rib. This placement maximized appearance of the QRS complex of the ECG. A reference electrode was attached to the forearm. The ECG signal was sent to an amplifier A/D converter and then to a computer. The computer was set to trigger a timer when an R-spike was detected. When the timer reached the desired time interval for the heartbeat detection task, a tone sounded.

The participant sat in a comfortable chair. Participants were then attached to the physiological recording equipment. A resting 5-minute baseline measure of heart rate was then taken. Following the baseline period, the experimenter read instructions for the heartbeat detection task. Following completion of the task, the experimenter removed the electrodes from the participant. The participant was then given a 30-day supply of the experience-sampling measures and dismissed.

We calculated "hit" and "false alarm" rates, as well as the number of total correct responses for each subject based on their responses to the 100 trials in order to compute nonparametric indices of sensitivity (A') and response bias (B') [39–41]. Typical values for A' vary between 0.50 (indicating chance discrimination) and 1.00 (perfect discrimination). B' values range from 0.00 indicating the absence of bias, with increasing positivity reflecting an increasingly cautious or strict criterion (tendency to report absence of the target stimuli), whereas an increasingly lax or risky criterion (tendency to report presence of the target stimuli) is indicated with increasing negativity.

Using the typical formulas for computing A' and B' [e.g., Ref. 39] it became apparent that a number of participants (n=46) displayed less than chance performance (i.e., their false positive rate exceeded their hit rate) on the task. Recently, however, it has been shown that the application of traditional signal detection formulas to data from individuals who perform below chance yield nonsensical values [42]. Following established statistical procedures, we adjusted the A' and B' values of those 46 participants who performed at less than chance level [42]. These adjustments did not change A' and B' values to the point where they reflected above chance performance, but merely reduced or increased the magnitude of the initial values to bring them closer to chance performance.

Results

In Study 1, the mean score on the SSAS (Mean = 15.7, S.D. = 6.4) was within two S.D. of the hypochondriacal, and within one S.D. of the nonhypochondriacal medical patients in the original SSAS standardization sample [3]. A similar mean score on the SSAS (Mean = 16.2, S.D. = 6.2) was found in Study 2. SSAS scores in both studies using university student samples were comparable to those demonstrated by medical outpatients [1,33], psychiatric out-

Table 1 Mean scores and correlations of Study 1 cross-sectional measures (N=81)

Measure	1	2	3	4	5	6	7	8	9	Mean	(S.D.)
1. SSAS	1.00									15.7	(6.4)
2. NEM	.54 **	1.00								7.6	(4.0)
3. SCL-90	.52 **	.51 **	1.00							18.1	(5.2)
4. IWS	.23 *	.22 *	.29 **	1.00						2.3	(3.8)
5. PSYCH	.51 **	.64 **	.48 **	.27 *	1.00					27.7	(7.7)
6. SOM	.27 *	.24 *	.39 **	.14	.36 **	1.00				19.7	(4.8)
7. NORM	.14	.10	.16	02	.37 **	.26*	1.00			34.6	(6.8)
8. BDI	.28 *	.42 **	.26	.46 **	.30 *	07	.14	1.00		7.9	(6.9)
9. WORRY	.40 **	.65 **	.30 **	.11	.40 **	.10	.24	.47 **	1.00	50.9	(14.3)

SSAS = Somatosensory Amplification Scale; NEM = Negative Emotionality Subscale of the Multidimensional Personality Questionnaire; SCL-90 = Symptom Checklist-90 Somatization Subscale; IWS = Illness Worry Scale; PSYCH = Psychological Attribution Subscale of the Symptom Interpretation Questionnaire; SOM = Somatic Attribution Subscale of the Symptom Interpretation Questionnaire; BOI = Beck Depression Inventory; WORRY = Penn State Worry Questionnaire.

patients diagnosed with panic or depressive disorder [5], and the general adult population [33]. The comparability of scores suggests that the results of this study can be generalized beyond university students.

Convergent validity of the SSAS

All participants (Study 1 N=81; Study 2 N=131) were included in analyses using cross-sectional measures, while only those who completed the studies (Study 1 n=48; Study 2 n=64) were used in the analysis using daily reports. In Study 1, mean SSAS scores were significantly higher for participants who reported five or more somatic symptoms in the previous month (Mean=17.2, S.D.=5.6) than those who reported no symptoms (Mean=13.4, S.D.=6.8) over the same period, t(77)=2.73, P<.05. This finding was replicated in Study 2 as mean scores on the SSAS for individuals who reported five or more somatic symptoms during the previous month (Mean=17.4, S.D.=5.7) were higher than those who reported no somatic symptoms (Mean=12.6, S.D.=6.5) during the same time

period, t(129) = -3.98, P < .001. As shown in Tables 1 and 2, individuals in both studies who scored high on the SSAS reported more symptoms (SCL-90) and worried more about being sick or having a serious illness (IWS), replicating previous findings [1,5]. These findings are consistent with others that demonstrate the association between the SSAS and somatizing tendencies and illness preoccupation. Both studies revealed that SSAS scores were related to making somatizing (SOM), as well as psychological (PSYCH) attributions to somatic symptoms on the SIQ. In Study 2, the SSAS was also related to making normalizing (NORM) attributions.

As shown in Tables 3 and 4, the SSAS was not significantly related to mean aggregated daily symptom reports (SYM) in Study 1 (r=.17, P<.30) or Study 2 (r=.19, P<.20). It is important to note that although the prospective daily symptom report (SYM) was not identical to both cross-sectional somatization measures, it was very similar in format and content to the SCL-90.

As predicted, the SSAS was not correlated with A' (r = -.04, P > .05) or number of correct responses

Table 2 Mean scores and correlations of Study 2 cross-sectional measures (N=131)

Measure	1	2	3	4	5	6	7	8	9	Mean	(S.D.)
1. SSAS	1.00									16.2	(6.2)
2. NEM	.46 **	1.00								6.8	(3.4)
3. SCL-90	.41 **	.46 **	1.00							18.6	(4.6)
4. IWS	.37 **	.38 **	.32 **	1.00						1.8	(1.7)
5. PSYCH	.40 **	.54 **	.35 **	.24 **	1.00					28.5	(7.5)
6. SOM	.38 **	.33 **	.34 **	.33 **	.42 **	1.00				19.2	(5.3)
7. NORM	.31 **	.20 *	.31 **	.04	.33 **	.34 **	1.00			35.4	(5.8)
8. BDI	.21 *	.45 **	.51 **	.21 *	.18*	.22 *	.08	1.00		7.0	(5.5)
9. WORRY	.43 **	.69 **	.38 **	.42 **	.52 **	.22 *	08	.35 **	1.00	48.8	(12.9)

SSAS = Somatosensory Amplification Scale; NEM = Negative Emotionality Subscale of the Multidimensional Personality Questionnaire; SCL-90 = Symptom Checklist-90 Somatization Subscale; IWS = Illness Worry Scale; PSYCH = Psychological Attribution Subscale of the Symptom Interpretation Questionnaire; SOM = Somatic Attribution Subscale of the Symptom Interpretation Questionnaire; NORM = Normalizing Attribution Subscale of the Symptom Interpretation Questionnaire; BDI = Beck Depression Inventory; WORRY = Penn State Worry Questionnaire.

^{*} *P*<.05.

^{**} P<.01.

^{*} *P*<.05.

^{**} P<.01.

Table 3 Mean scores and correlations of Study 1 experience-sampling measures (N=81)

Measure	1	2	3	4	5	6	Mean	(S.D.)
1. SSAS	1.00						15.7	(6.4)
2. HASS	.41 **	1.00					11.7	(7.1)
3. HASSEV	.34*	.90 **	1.00				27.5	(21.3)
4. HASSINT	.26	.44 **	.71 **	1.00			2.0	(0.7)
5. SYM	.17	.58 **	.63 **	.53 **	1.00		2.7	(1.9)
6. NMOOD	.43 **	.65 **	.78 **	.53 **	.54 **	1.00	13.8	(13.4)

SSAS = Somatosensory Amplification Scale; HASS = Hassles Frequency; HASSEV = Hassles Severity; HASSINT = Hassles Intensity; SYM = Daily Somatic Symptoms; NMOOD = Negative Mood Subscale of the Positive Negative Affect Scale.

(r=-.06, P>.05) in the heartbeat detection task. Thus, it appears that the SSAS is not related to interoceptive sensitivity to resting heartbeats. Furthermore, the SSAS was not related to B' (r=-.05, P>.05), indicating that individuals who report being somatically sensitive do not have a characteristic response style.

Discriminant validity of the SSAS

As shown in Tables 1 and 2, the SSAS and NEM were highly correlated. The strength of these associations is impressive when one considers that the NEM does not include items tapping somatic content. Indeed, the SSAS and NEM were similarly correlated to the measures of somatizing tendencies (SCL-90, IWS, SOM) and generalized distress (BDI, WORRY). Therefore, it appears that individuals who report being somatically sensitive, report somatic and psychological distress.

To further examine our contention that the SSAS measures both somatizing tendencies, as well as a significant negative emotionality component, we conducted a hierarchical multiple regression analysis. This analysis allowed us to compare the predictive strength of both somatization tendencies and negative emotionality on SSAS scores. Some studies have found small and inconsistent gender differences in somatization and amplification tendencies [2,3,5]. Since we had more women than men in our samples, we controlled for any possible effects of gender by entering sex at Step 1. We entered a composite somatization index (i.e., Z-

score transformations of the SCL-90 Somatization Subscale and IWS) at Step 2, and a composite negative emotionality index (i.e., Z-score transformations of NEM, BDI, WORRY) at Step 3. The results for Study 1 are shown in the top-half of Table 5. In this analysis, sex was not a predictor of SSAS scores. The somatization composite index (SOMATIC) was a significant predictor of SSAS scores, accounting for approximately 19% of the variance in SSAS scores. The negative emotionality composite index (NEGEMOT) was also a significant predictor, explaining an additional 10% of the variance in SSAS scores. The entire model accounted for approximately 29% of the variance in SSAS scores. The findings were similar in Study 2 (see the top-half of Table 6). Sex was a significant predictor of SSAS scores, explaining 8% of the variance. The SOMATIC composite index again explained 19% of the variance, and the NEGEMOT composite predicted only 3% of the variance. We conducted the same analysis including only those participants completing the studies. In Study 1 (n=48), sex did not explain variance in SSAS scores. The SOMATIC explained approximately 17% of the variance in SSAS scores, while the NEGEMOT explained an additional 20% of the variance. The entire model explained approximately 36% of the variance in SSAS scores. Similar results were found in Study 2 (n=64). Sex remained a significant predictor of SSAS scores, explaining 11% of the variance in SSAS scores. The SOMATIC composite explained 20% of the variance, and the NEGEMOT composite explained only 6% of the variance in SSAS scores.

Table 4 Mean scores and correlations of Study 1 experience-sampling measures (N=131)

Measure	1	2	3	4	5	6	Mean	(S.D.)
1. SSAS	1.00						16.2	(6.2)
2. HASS	.39 **	1.00					10.9	(8.0)
3. HASSEV	.26 *	.80 **	1.00				22.0	(19.1)
4. HASSINT	03	.19 **	.56 **	1.00			1.9	(1.1)
5. SYM	.19	.20 **	.29 **	.24 **	1.00		1.9	(2.6)
6. NMOOD	.10	.57 **	.52 **	.20	.36 **	1.00	10.7	(9.2)

SSAS = Somatosensory Amplification Scale; HASS=Hassles Frequency; HASSEV=Hassles Severity; HASSINT=Hassles Intensity; SYM=Daily Somatic Symptoms; NMOOD=Negative Mood Subscale of the Positive Negative Affect Scale.

^{*} *P*<.05. ** *P*<.01.

^{*} *P*<.05.

^{**} P<.01.

We conducted a similar analysis, but entered only NEM on Step 3. As shown in the bottom-half of Table 5 (Study 1), sex did not explain variance in SSAS scores. The somatization index explained approximately 17% of the variance in SSAS scores, while NEM also explained approximately 17% of the variance. The entire model accounted for approximately 35% of the variance in SSAS scores. Entering NEM on Step 3 had virtually no effect on the results of Study 2 (see bottom-half of Table 6). When considering only the 48 participants who completed Study 1, we found that sex was not a predictor of SSAS scores, the SOMATIC continued to explain approximately 17%, while NEM explained approximately 30% of the variance in SSAS scores (vs. only 17% when considering all participants). The entire model explained approximately 46% of the variance in SSAS scores. When considering only the 64 participants who completed Study 2, results were virtually identical to those found when considering all participants who completed Study 2.

We also conducted regression analyses entering the negative emotionality index before the somatization index. This allowed us to determine whether somatization tendencies predicted variance in the SSAS after accounting for variance explained by negative emotionality. In this analysis, the negative emotionality indices (NEGEMOT and NEM alone) explained more than twice as much variance in SSAS than did the somatization index. For example, in Study 1 NEGEMOT accounted for 22% of the variance in SSAS scores, while SOMATIC explained a nonsignificant 5%. NEM alone accounted for 28% of the variance, while SOMATIC explained only 8%. Similar percentages were found in Study 2. We also conducted all the above regressions no longer including sex as a predictor. Results for both studies remained essentially unchanged. However, in Study 1, the somatization composite explained a small but significant proportion of variance in the SSAS (6% total sample, 8% completers only), when the somatization composite was entered into the regression at Step 2, and only when NEM alone was entered at Step 1.

Next, we examined the relationship between scores on the SSAS and the experience-sampling indices. As shown in Tables 3 and 4, the SSAS was significantly related to the

Table 5 Summary of hierarchical regression analysis for Study 1 variables predicting SSAS scores (N=81)

Step	Variable	R^2	R ² change	P
1.	SEX	.02		.370
2.	SOMATIC	.19	.17	.003
3.	NEGEMOT	.29	.11	.010
1.	SEX	.02		.373
2.	SOMATIC	.19	.17	.003
3.	NEM	.35	.17	.001

SOMATIC = Composite Somatization Index (SCL-90 Somatization Subscale; Illness Worry Scale), NEGEMOT = Composite Negative Emotionality Index (NEM, BDI, PSU Worry Scale), NEM = Negative Emotionality Subscale of the Multidimension Personality Questionnaire.

Table 6 Summary of hierarchical regression analysis for Study 1 variables predicting SSAS scores (N=131)

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Step	Variable	R^2	R ² change	P
1.	SEX	.08		.001
2.	SOMATIC	.28	.19	.001
3.	NEGEMOT	.30	.03	.036
1.	SEX	.08		.001
2.	SOMATIC	.28	.19	.001
3.	NEM	.34	.06	.001

SOMATIC = Composite Somatization Index (SCL-90 Somatization Subscale; Illness Worry Scale), NEGEMOT = Composite Negative Emotionality Index (NEM, BDI, PSU Worry Scale), NEM = Negative Emotionality Subscale of the Multidimension Personality Questionnaire.

report of hassles frequency (HASS) and hassles severity (HASSEV), while in Study 1, there was a trend for hassles intensity (HASSINT). It is important to note that our hassles measure did not include any physical or health hassles, thus making the association between the SSAS and hassles more impressive. In Study 1, the SSAS was also significantly related to a global index of daily negative mood (NMOOD), as well as the hostility, fear, and sadness subscales that comprise the global index. This finding was not replicated in Study 2.

Discussion

We examined the psychometric properties of the SSAS as an index of somatic sensitivity in two longitudinal studies. While the SSAS purports to be a measure of somatic sensitivity, we have found only partial evidence to support this notion. Consistent with previous research [e.g., Refs. 1–3,5,33], the SSAS demonstrated reasonable convergent validity with cross-sectional symptom and illness worry reports. However, scores on the SSAS were not related to daily symptom reports, which are more accurate than retrospective ratings [34,35], nor were they related to objectively measured interoceptive sensitivity, replicating previous findings [6–8]. Furthermore, while the SSAS converged with a measure of somatic attributions, it also correlated with psychological attributions, replicating previous findings [36].

The discriminant validity of the SSAS was weak. The SSAS was correlated with self-report measures of negative emotionality, depressive tendencies, anxiety, and with daily negative mood reports in Study 1. The pattern of correlations between the SSAS and NEM with respect to symptom reporting, psychological distress, and somatic attributions was almost identical. This implies that the SSAS and measures of psychological distress and negative emotionality are likely tapping similar constructs. In both studies, scores on the SSAS were related to the frequency and severity of daily hassles. It is important to remember that our hassles measure included no items reflecting somatic or health issues. Taken

together, these findings clearly indicate that participants responded to this scale as another measure of distress or, at best, a measure of anxious beliefs about somatic symptoms. Furthermore, although the SSAS was correlated with a tendency to make somatic attributions about somatic symptoms, it was also correlated with a tendency to make psychological and normalizing attributions (Study 2 only). In fact, the SSAS was most strongly correlated with psychological attributions. That the SSAS correlated to all three kinds of attributions again suggests that the measure does not reflect somatic processes, but rather psychological ones.

In multiple regression analyses, both somatization tendencies and negative emotionality predicted scores on the SSAS. There was some inconsistency between Study 1 and 2 in these analyses. In Study 1, somatization and negative emotionality explained nearly identical proportions of variance in SSAS scores. In Study 2, somatization tendencies explained more than twice as much of the variation in SSAS scores than did negative emotionality. However, in Study 1, when the somatization composite was entered at Step 3 in the regression analysis, it no longer explained any variance in SSAS scores, except for a small percentage of variance when NEM alone was entered at Step 2. Entering the somatization composite at Step 3 yields a more stringent test of the notion that the SSAS measures somatic sensitivity. This test found that even in the best case, the somatization composite only explains between 5% and 11% of the variance. Overall, the weight of the evidence from the regression analyses suggests again that the SSAS is not a measure of somatic sensitivity, but rather one of psychic distress.

Consistent with previous findings [i.e., Refs. 6-8,37], we found no relationship between scores on the SSAS and interoceptive sensitivity to resting heartbeat. It is important to note that we used a slightly different heartbeat detection methodology than has been used in the previous studies (i.e., Kluviste vs. Whitehead procedure). Thus, the failure to replicate has now generalized to various methodological approaches to measuring somatic sensitivity. The lack of relationship between scores on the SSAS and sensitivity to resting heartbeat is consistent with the view that the SSAS does not measure interoceptive sensitivity. Recently, the SSAS has been criticized for potentially confounding somatic activity with the experience of symptoms and thus measures the outcome but not the process of somatosensory amplification [43]. Our results lend support for this contention. Based on these findings, it seems reasonable to conclude that the SSAS is not related to cardiac sensitivity. It is important to note, however, that most individuals are not particularly sensitive to resting heartbeat [44]. It is conceivable that scores on the SSAS may be related to sensitivity to other interoceptive and exteroceptive organ systems. Further studies should use signal detection tasks to assess the relationship between SSAS scores and sensitivity to various organ systems.

SSAS scores obtained by the university students in both studies were comparable to those demonstrated in samples

of medical outpatients, as well as the general adult population [1,5,33]. This finding contradicts Barsky et al.'s (1988) [1] notion that the SSAS only be used with individuals using medical resources. University students report a significant number of somatic symptoms, express illness worry and concern, and use health care resources [15–18]. University students have proven a fruitful population in which to study the symptom reporting process. Results of our study support the continued use of university students in studies examining somatizing tendencies.

Based on our results, the SSAS should not be used as a measure of somatic sensitivity. The measure appears to capture nonspecific distress and a tendency toward negative emotionality. It is important to note that due to the number of statistical comparisons made in our studies, we have increased the chance of Type I error. There is current debate among researchers regarding the relative importance of significance level as compared to effect size and replication of results. Recently, researchers have argued persuasively that correction for the number of comparisons increases the chances of Type II error, a methodological/statistical result they consider more problematic [45-47]. These researchers and others have also suggested that replicating results is more important than significance levels [48]. Other researchers continue to support the adjustment of significance levels to account for multiple comparisons [49]. A post hoc analysis adjusting the significance level for comparisons (i.e., P < .01) did not change the overall interpretation of our findings.

While it may be possible to measure somatic sensitivity via self-report, no such measure currently exists. Until such a measure is developed, future researchers should use objective indices of somatic sensitivity, such as signal detection tasks, to operationalize such sensitivity. These tasks should be used with different organ systems in the body, since somatic sensitivity may differ from one organ system to another. Furthermore, daily measurement of somatic symptom reporting should be used to remove retrospective bias in cross-sectional measures [34,35]. Although undoubtedly challenging, future researchers should also work on the development of a reliable and valid self-report measure of somatic sensitivity, since such a measure would be efficient and cost effective.

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References

 Barsky AJ, Goodson JD, Lane RS, Cleary PD. The amplification of somatic symptoms. Psychosom Med 1988;50:510-9.

- [2] Barsky AJ, Wyshak G. Hypochondriasis and somatosensory amplification. Br J Psychiatry 1990;157:404-9.
- [3] Barsky AJ, Wyshak G, Klerman GL. The Somatosensory Amplification Scale and its relationship to hypochondriasis. J Psychiatr Res 1990;24:323–34.
- [4] Wise TN, Mann LS. The relationship between somatosensory amplification, alexithymia, and neuroticism. J Psychosom Res 1994; 38:515-21.
- [5] Spinhoven P, van der Does AJW. Somatization and somatosensory amplification in psychiatric outpatients: an exploratory study. Compr Psychiatry 1997;38:93-7.
- [6] Barsky AJ, Brener J, Coeytaux RR, Cleary PD. Accurate awareness of resting heartbeat in hypochondriacal and non-hypochondriacal patients. J Psychosom Res 1995;39:489–97.
- [7] Barsky AJ, Cleary PD, Brener J, Ruskin JN. The perception of cardiac activity in medical outpatients. Cardiology 1993;83:304–15.
- [8] Mailloux J, Brener J. Somatosensory amplification does not reflect sensitivity to heartbeat sensations. Paper presented at the annual meeting of the American Psychological Society, 1998 June.
- [9] Haenen M, Schmidt AJM, Schoenmakers M, van den Hout MA. Tactual sensitivity in hypochondriasis. Psychother Psychosom 1997; 66:128–32.
- [10] Wyshak G, Barsky AJ, Klerman GL. Comparison of psychiatric screening tests in a general medical setting using ROC analysis. Med Care 1991;29:775–85.
- [11] Kosturek A, Gregory RJ, Sousou AJ, Trief P. Alexithymia and somatic amplification in chronic pain. Psychosomatics 1998;39:399-404.
- [12] Costa PT, McCrae RR. Hypochondriasis, neuroticism, and aging: when are somatic complaints unfounded. Am Psychol 1985;40:19–28.
- [13] Costa PT, McCrae RR. Neuroticism, somatic complaints, and disease: is the bark worse than the bite? J Pers 1987;55:299-316.
- [14] Watson D. Intraindividual and interindividual analyses of positive and negative affect: their relation to health complaints, perceived stress, and daily activities. J Pers Soc Psychol 1988;54:1020-30.
- [15] Watson D, Pennebaker JW. Health complaints, stress, and distress: exploring the central role of negative affectivity. Psychol Rev 1989; 96:234-54.
- [16] Aronson KR. The role of emotional reactivity in symptom reporting: Somatic sensitivity, biased responding, or negative reporting style? Unpublished doctoral dissertation, Pennsylvania State University, 1999.
- [17] Larsen RJ. Neuroticism and selective encoding and recall of symptoms: Evidence from a combined concurrent–retrospective study. J Pers Soc Psychol 1992;62:480–8.
- [18] Larsen RJ, Kasimatis M. Day-to-day physical symptoms: Individual differences in the occurrence, duration, and emotional concomitants of minor daily illnesses. J Pers 1991;59:387–423.
- [19] Watson D, Clarke LA. Negative affectivity: the disposition to experience aversive emotional states. Psychol Bull 1984;96:465–90.
- [20] Watson D, Clarke LA, Tellegen A. Development and validation of brief measures of positive and negative affect: the PANAS Scales. J Pers Soc Psychol 1988;54:1063-70.
- [21] Waller NG, Tellegen A, McDonald RP, Lykken DT. Exploring nonlinear models in personality assessment: development and preliminary validation of a negative emotionality scale. J Pers 1996;64:545-76.
- [22] Swartz M, Hughes D, George L, Blazer D, Landerman R, Bucholz K. Developing a screening index for community studies of somatization disorder. J Psychiatr Res 1986;20:335–43.
- [23] Derogatis LR, Rickels K, Rock A. The SCL-90 and the MMPI: a step in the validation of a new self-report scale. Br J Psychiatry 1976; 128:280-9.
- [24] Kirmayer LJ, Robbins JM. Three forms of somatization in primary care: prevalence, co-occurrences, and sociodemographic characteristics. J Nerv Ment Dis 1991;179:647–55.

- [25] Robbins JM, Kirmayer LJ. Attributions of common somatic symptoms. Psychol Med 1991;21:1029–45.
- [26] Tellegen A. Brief manual for the differential personality questionnaire. Unpublished manuscript, University of Minnesota, 1982.
- [27] Beck AT, Rush AJ, Shaw BF, Emery G. Cognitive therapy of depression. New York: Guilford Press, 1979.
- [28] Meyer TJ, Miller ML, Metzger RL, Borkovec TD. Development and validation of the Penn State Worry Questionnaire. Behav Res Ther 1990;28:487–95.
- [29] Kanner AD, Coyne JC, Schaefer C, Lazarus RS. Comparison of two modes of stress measurement: daily hassles and uplifts versus major life events. J Behav Med 1981;4:1–39.
- [30] Watson D, Clarke LA. Preliminary manual for the positive affect and negative affect schedule (expanded form). Unpublished manuscript, Southern Methodist University, 1991.
- [31] Wu KK, Lam DJ. The relationship between daily stress and health: replicating and extending previous research findings. Psychol Health 1993;8:329-44.
- [32] Feldman Barrett L. Discrete emotions or dimensions? The role of valence focus and arousal focus. Cognit Emotion 1998;12:579–99.
- [33] Speckens AEM, Spinhoven P, Sloekers PPA, Bolk JH, van Hemert AM. A validation study of the Whitley Index, the Illness Attitude Scales, and the Somatosensory Amplification Scale in general medical and general practice patients. J Psychosom Res 1996;40:95–104.
- [34] Wheeler L, Reis HT. Self recording of everyday life events: origins, types, and uses. J Pers 1991;59:339–54.
- [35] Stone AA, Shiffman S. Ecological momentary assessment (EMA) in behavioral medicine. Ann Behav Med 1994;16:199–202.
- [36] Wise TN, Mann LS. The attribution of somatic symptoms in psychiatric outpatients. Compr Psychiatry 1995;36:407–10.
- [37] Barsky AJ, Cleary PD, Sarnie MK, Ruskin JN. Panic disorder, palpitations, and the awareness of cardiac activity. J Nerv Ment Dis 1994;182:63-71.
- [38] Whitehead WE, Drescher VM, Heiman P, Blackwell B. Relation of heart rate control to heartbeat perception. Biofeedback Self Regul 1997;2:371–92.
- [39] Boice R, Gardner RM. A computer program to generate parametric and nonparametric signal-detection parameters. Bull Psychon Soc 1988;26:365-7.
- [40] Hodos W. Nonparametric index of response bias for use in detection and recognition experiments. Psychol Bull 1970;74:351–4.
- [41] Pollack I, Norman DA. A non-parametric analysis of recognition experiments. Psychon Sci 1964;1:125–6.
- [42] Aaronson D, Watts B. Extensions of Grier's computational formulas for A' and B' to below chance performance. Psychol Bull 1987; 102:439-42.
- [43] Kirmayer LJ, Robbins JM, Paris J. Somatoform disorders: personality and the social matrix of somatic distress. J Abnorm Psychol 1994; 103:125–36.
- [44] Harver A, Katkin ES, Bloch E. Signal-detection outcomes on heartbeat and respiratory resistance detection task in male and female subjects. Psychophysiology 1993;30:223-30.
- [45] Cohen J. The earth is round (p < .05). Am Psychol 1994;49:997–1003.
- [46] Cohen J. The earth is round: Rejoinder (p < .05). Am Psychol 1995;50:1103.
- [47] Sohn D. Statistical significance and replicability: why the former does not presage the latter. Theory Psychol 1998;8:291–311.
- [48] Thompson B. If statistical significance tests are broken/misused, what practices should supplement or replace them. Theory Psychol 1999; 9:165–81.
- [49] Chow SL. Statistical significance: rationale, validity and utility. London, England: Sage Publications, 1996.