# Somatic Awareness and Tender Points in a Community Sample

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## Abstract

Somatic awareness (SA) refers to heightened sensitivity to a variety of physical sensations and symptoms. Few attempts have been made to dissociate the relationship of SA and affective symptoms with pain outcomes. We used a validated measure of mood and anxiety symptoms that includes questions related to SA to predict the number of tender points found on physical examination in a large cross-sectional community sample (the Midlife in the United States [MIDUS] Biomarker study). General distress, positive affect, and SA, which were all significantly associated with tender point number in bivariate analyses, were used as predictors of the number of tender points in a multivariate negative binomial regression model. In this model a greater number of tender points was associated with higher levels of SA ($p = .02$) but not general distress ($p = .13$) or positive affect ($p = .50$). Follow-up mediation analyses indicated that the relationship between general distress and tender points was partially mediated by levels of SA. Our primary finding is that SA is strongly related to the number of tender points in a community sample. Mechanisms linking SA to the spatial distribution of pain sensitivity should be investigated further.

**Perspective**—This article presents an analysis of three overlapping psychological constructs and their relationship to widespread pain sensitivity on palpation. The findings suggest that somatic awareness is most strongly related to the spatial distribution of pain sensitivity and that further assessing it may improve our understanding of the relationship between psychological factors and pain.

## Keywords

Somatic Awareness; Tender Points; Positive Affect; Distress; Pain

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## 1. Introduction

Heightened Somatic Awareness (SA) refers to a greater than average awareness for a variety of physical sensations and symptoms. Individuals high in SA have a tendency to notice and report non-specific symptoms, such as feeling shortness of breath, faint or having the sensation that one's muscles are trembling. Higher levels of SA have been linked to the presence of a variety of chronic pain conditions including fibromyalgia (FM)[26, 47], irritable bowel syndrome (IBS)[48], and temporomandibular disorder (TMD)[23]. Within pain conditions, higher levels of SA have also been linked to greater painful symptom severity[1, 41, 43] and experimental pain testing outcomes such as more tender facial areas on palpation in TMD patients,[58] lower heat pain thresholds in women with provoked vestibulodynia,[61] and higher pressure sensitivity in FM and TMD.[28] This has led to speculation that SA reflects some combination of psychological and neurobiological vulnerability to pain.[47]

However, the nature of this vulnerability continues to be debated, in part because SA is strongly associated with negative affect (i.e., depressive and anxious symptoms)[68] which is also strongly associated with pain[9, 10, 15, 22, 27, 33, 65]. Complicating matters, the relationship between different types of affective processes and pain outcomes differs substantially across studies. A study of patients with Complex Regional Pain Syndrome found that the previous day's level of depressed mood, but not anxiety, predicted self-reported clinical pain[22] and a recent systematic review found that depression but not anxiety was related to knee pain[55]. Conversely, preoperative anxiety predicts postoperative pain[71] and experimentally induced anxiety produces pronounced increases in pain reports[62]. Positive affect has also been linked to lower levels of self-reported pain[75] and increased tolerance when induced by pleasant images[16]. Despite its potential relevance, many pain studies do not attempt to differentiate the impact of affect and SA on measures of pain sensitivity. This could be important for researchers interested in the neurobiological basis of pain disorders, given recent neuroimaging investigations suggest that the neural underpinnings of SA may differ from those associated with general depressive and anxious symptoms[21, 35]. For instance, one functional connectivity study found that higher levels of SA were associated with greater functional connectivity between elements of the so-called pain matrix[53], and SA also has been shown to be characterized in part by cognitive biases such as greater attention to and recall of bodily symptoms.[72]

Tender points are discrete areas of the body where moderate palpation produces pain in some individuals. Tender points have been used to classify individuals with FM for research purposes for many years[74], though it has been known for some time that the number of positive tender points demonstrates a linear relationship with measures of distress and disability.[14, 19, 73] These findings suggest that tender points measure a continuum of the "widespreadness" of pain sensitivity and possibly a vulnerability to develop clinical pain disorders.

Disentangling the unique contributions of SA and affective states on pain outcomes has the potential to improve the measurement of clinical pain and address potential risk factors for developing pain disorders. To determine the relationship between SA, affect, and the

diffuseness of pain sensitivity we used established subscales for SA and affective constructs to predict tender points in a community sample. We hypothesized that variance in the number of tender points would be most strongly explained by SA followed by negative and positive affect. We further hypothesized that part of the association between affective symptoms and the spatial distribution of pain sensitivity would be mediated by comorbid levels of SA, indicating a more proximal role for SA in predicting tender points..

## 2. Methods

### 2.1. Sample

We performed a retrospective analysis of the Midlife in the United States (MIDUS) biomarker study. Between 1995 and 1996, 7189 non-institutionalized adults were recruited by random-digit dial to take part in a study of health and aging (MIDUS I)[5]. Of these, 4963 were re-contacted between 2004 and 2005 to take part in a follow-up study. The Biomarker Project[17] represented a subset of these participants who underwent a physical examination, additional questionnaires, and provided blood and urine for analysis of a variety of physiological measures, including markers of inflammation and sympathetic nervous system (SNS) activity. All participants in the second wave (MIDUS II) who completed the phone interview were eligible for this biomarker study. 1255 agreed to participate and were provided compensation to cover travel expenses to one of the three sites (the University of Wisconsin, Madison; the University of California, Los Angeles; Georgetown University, Washington, D.C.). At the University of Wisconsin a long form physical evaluation was conducted that included a tender point examination on 522 participants. Of these, 15 were missing data on a variable of interest and were excluded. Therefore, the final sample consisted of 507 participants. All participants provided informed consent and all procedures were approved by the respective institutional review boards.

### 2.2. Measures

#### 2.2.1. Mood and Somatic Awareness—The Mood and Anxiety Symptom Questionnaire (MASQ) is a self-report measure of symptoms of anxiety and depression[68, 69]. In the MIDUS sample a 64-item version was administered. Participants are asked how much they experienced each item in the last week on a 5-point scale (1= not at all, 5 = extremely). These items were used in a principal components analysis to identify mood and SA constructs. A three-part (tripartite) structure of the MASQ has been observed in multiple samples consisting of a) general psychological distress, b) positive affect, and c) somatic awareness factors[32, 68] primarily through the use of Principal Components Analysis (PCA). The resulting subscales demonstrate excellent convergent validity when compared to other measures of depression and SA.[66] This tripartite structure has also been observed in at least one sample of chronic pain patients[25]. Others have questioned this model via the use of confirmatory factor analysis[4] or by proposing alternative models that employ first and second order factors of depression and anxiety[8]. To differentiate the items Bedford (1997) has argued for a two part approach to item reduction, retaining items only if they load on an individual factor at .30 or greater *and* show a .20 higher loading than on any other factor[2]. Using this approach the tripartite structure of the MASQ provides a good fit to the data in

large samples of men and women, healthy controls, patients in primary care, and patients in mental health care settings[66]. Supplementary Table 1 shows each of the 64 MASQ items.

**2.2.2. Health Information and Current Medication Data**—The following were collected by self-report: engaging in regular exercise (20 minutes or more at least 3 times/ week), smoking status (current/former, never) age, gender, and presence of chronic conditions/symptoms (heart disease, high blood pressure, circulation problems, blood clots, heart murmur, transient ischemic attack or stroke, anemia or other blood disease, cholesterol problems, diabetes, asthma, emphysema/chronic obstructive pulmonary disease, tuberculosis (TB), positive TB skin test, thyroid disease, peptic ulcer disease, cancer, colon polyp, arthritis, glaucoma, cirrhosis/liver disease, alcoholism, depression, blood transfusion before 1993). Sleep efficiency was calculated from participants (n=409) who wore an activity monitor for seven consecutive days. Sleep efficiency was calculated from the percentage of scored total sleep time (from the device) divided by the interval duration and averaged over the seven sleep periods.

Participants were instructed to bring all medication, in original bottles to the University of Wisconsin-Madison site at the time of the evaluation. Codes were applied to each medication based on medication name, route of administration, and purpose, following the American Hospital Formulary System (AHFS) Pharmacologic-Therapeutic classification system. For the current analyses, medications were coded into categories based on common pharmacologic effects that might impact pain and/or mood. These were antidepressants (e.g., selective serotonin/norepinephrine reuptake inhibitors, tricyclic antidepressants), corticosteroids, opiates, nonsteroidal anti-inflammatory drugs, and anxiolytics/sedatives (e.g. benzodiazepines).

**2.2.3 Physical Examination**—The physical examination was conducted by a credentialed clinician (i.e., advanced practice nurse, nurse practitioner, physician assistant, medical doctor). Height and weight were measured for calculation of body mass index. Joints were examined for deformities, crepitation, limited range of motion, swelling, heat, and redness. Muscles were examined for tremor, atrophy, and fasciculation. Participants were then coded as having either normal or abnormal joint/musculature findings. A neurological sensation examination was conducted on the right and left upper and lower extremities (light touch, pinprick, temperature, vibration, and limb position) and participants were coded as having either normal (i.e., sensation detected) or abnormal (i.e., sensation not detected) responses to each stimulus.

A tender point examination was conducted on eighteen distinct areas of the body using the tender point examination portion of the American College of Rheumatology 1990 criteria for fibromyalgia.[74] Examiners were initially trained by the same experienced clinician to ensure consistent and proper technique. Further training was conducted by the most experienced clinician available. These were tested bilaterally at the occiput: suboccipital muscle insertions, trapezius: midpoint of the upper border, supraspinatus: above the medial border of the scapular spine, gluteal: upper and outer quadrants of the buttocks, greater trochanter: posterior to the trochanteric prominence, low cervical: anterior aspects of the intertransverse spaces at C5–C7, second rib: second costochondral junction, lateral epicondyle: 2 cm distal

to the epicondyles, and knee: medial fat pad proximal to the joint line. A tender point was determined by applying either the thumb or first two fingers at a pressure of approximately 4 kg.

**2.2.4 C-Reactive Protein (CRP)—**Fasting blood samples were obtained from participants prior to breakfast. Samples were stored at −80°C. CRP was assayed with an immunonepholometric assay using a BNII nephelometer (Dade Behring Inc). The inter-assay coefficient of variation (CV) is 2.1 to 5.7%.

**2.2.5. Norepinephrine & Creatinine—**A 12-hour overnight (7:00 pm – 7:00 am) urine sample was obtained from each participant in a container with 25 mL of 50% acetic acid. These were stored at −80°C. High-Pressure Liquid Chromatography (HPLC) was used to measure norepinephrine[31]. The inter-assay CV is 6.7–6.9%. Creatinine was measured using an Enzymatic Colorimetric Assay. The inter-assay CV is 0.85%. Norepinephrine levels were then adjusted to levels of creatinine.

### 2.3 Statistical Analyses

Analyses were conducted in SPSS version 22.0 and R version 3.2.2 (Package 'MASS').

**2.3.1 Affect and Somatic Awareness – Mood and Anxiety Symptom Questionnaire—**We used the available items from a three factor solution using the full 90-item MASQ questionnaire in a large sample (n=534) that resulted from this item reduction approach (Keogh and Reidy, 2000; sixteen items for general psychological distress, thirteen items for positive mood, sixteen items for SA).[32] These subscales were correlated with other measures of mood/affect administered in the MIDUS Biomarker project (Perceived Stress Scale [PSS][13], Center for Epidemiological Studies – Depression [CES-D][56], Spielberger Trait Anxiety Inventory [STAI][60]). Because the MASQ version administered in the MIDUS Biomarker subsample used 64 items rather than the 90 items frequently used in other samples, we also opted to confirm the tripartite structure in this sample via PCA (Supplementary Table 1).

**2.3.2. Bivariate Analyses—**Associations between potential covariates and number of tender points were examined using non-parametric methods: Spearman's rank correlations for continuous variables and Mann-Whitney U tests for categorical variables. Covariates that were significantly associated with the number of tender points ($p < .05$) were retained in multivariate models. Additionally, associations between the measures of interest (general psychological distress, positive affect, SA) and number of tender points were examined by Spearman's rank correlation.

**2.3.3. Multivariate Model—**To determine which measures of SA and mood were most strongly associated with the number of tender points, we used negative binomial regression in a model including each of the measures (general psychological distress, positive mood, SA) and significant covariates. Negative binomial regression is a similar approach to Poisson regression with one additional parameter to account for the overdispersion of the data – relevant here because of the large number of participants without any tender points (76%).

This model was then compared to Poisson regression, and zero-inflated negative binomial models using the same data by Bayesian Information Criteria (BIC). By these metrics the negative binomial model provided the best fit to the data (negative binomial model: 981 < zero-inflated Poisson model: 997 < Poisson regression model: 1256). Fixed effects from negative binomial models are interpreted in the same way as results from Poisson regression models.

**2.3.4 Secondary Analysis—**To determine if observed relationships between SA and tender points were driven by those participants with the highest levels of SA, we conducted a secondary analysis identical to the multivariate model but excluding participants whose SA levels were one standard deviation above the mean (SA    28; n = 63).

**2.3.5. Mediation Models—**To determine if the relationship between general psychological distress and positive affect with tender points is mediated by somatic awareness, we conducted causal mediation analyses using the framework advocated by Imai, Keele and Tingley (2010)[29], implemented in Python Statsmodels. The mediation is between: a) positive affect and SA, and b) general psychological distress and SA. These models provide estimates of average mediated and direct effects while accounting for covariates, and also estimate both types of effects for different levels of the independent variable. Standardized values of general psychological distress, positive affect, and SA were used for these models.

# 3. Results

## 3.1. Sample Demographic and Health Characteristics

Participants were approximately 53 years old on average. A majority were female (59%) and married or living with a partner (56%). On average, participants were using approximately three prescription medications and had approximately four chronic conditions. See Table 1 for demographic and health characteristics of the complete sample including more detailed information regarding medication use, and comparisons of participants with and without tender points.

## 3.2 MASQ subscales

The subscales derived from Keogh & Reidy's three factor solution[32] were correlated with other measures of emotionality administered in the MIDUS Biomarker subsample in the manner expected (e.g., MASQ-general psychological distress was associated with measures of negative mood, MASQ-positive mood was associated with positive affect). See Table 2. The PCA conducted on the 64 items resulted in a three factor solution very similar to those previously reported (See Supplementary Table 1 for methods and results.) The subscales used in subsequent analyses were highly correlated with the subscales derived from the PCA (General Psychological Distress subscales, r = .96; Positive Mood subscales, r = .994; SA subscales, r = .98; all p < .001). These results are unsurprising as eleven items were common to both general psychological distress subscales, thirteen items were common to both positive mood subscales, and thirteen were common to both SA subscales.

### 3.3. Bivariate Analyses

Using non-parametric correlations, age, BMI, number of chronic conditions, CRP, and norepinephrine were associated with the number of tender points (all $p < .05$) while sleep efficiency was not ($p = .10$). Using Mann-Whitney U tests, female gender, abnormal joints/musculature, use of NSAIDs, use of opioids/analgesics, use of sedatives/anxiolytics, and use of antidepressants were associated with more tender points (all $p < .05$) while use of corticosteroids ($p = .40$), regular exercise ($p = .07$), smoking status ($p = .65$), and abnormal neurological examination results ($p = .40$) were not. General psychological distress (Spearman's rho = .189, $p < .01$), positive affect (Spearman's rho = $-$ .101, $p = .019$) and SA (Spearman's rho = .295, $p < .01$) were each associated with the number of tender points in bivariate analyses.

### 3.4. Multivariate Model

Higher levels of SA were associated with a greater number of tender points on physical examination ($p = .019$) while levels of general negative mood ($p = .13$) and positive affect ($p = .50$) were not, controlling for age, gender, BMI, use of antidepressants, use of sedatives/anxiolytics, use of opioids, CRP and norepinephrine, physical examination results and the number of chronic conditions present. Older age ($p = .014$), female gender ($p < .01$), use of sedatives/anxiolytics ($p = .035$), use of opioids ($p = .040$), and abnormal joints/musculature on physical examination ($p = .020$) were also associated with a greater number of tender points. Each one point increase on the SA subscale (range 16–51) was associated with an approximately 5% higher estimated likelihood of finding an additional tender point during the examination (Estimated likelihood=1.051, 95% CI = 1.008, 1.098. See Table 3 for full parameters of the model including estimates for each covariate and Figure 1 showing the distribution of tender points by low, medium and high levels of SA (for illustrative purposes).

### 3.5. Secondary Analysis

Excluding high SA participants resulted in no substantial differences from the results of the multivariate model including all participants. SA was associated with the number of tender points found on physical examination (IR= 1.096, 95% CI =1.001, 1.202, p = .047) while neither general distress (p = .20) nor positive affect (p = .76) were associated with the number of tender points.

### 3.6. Mediation Analyses

SA was a significant mediator of the effect of general psychological distress on tender points, accounting for approximately 55% of the total effect (Est. = .561, 95% CI = .076, 1.46, p = .016). In this model the direct effect of general psychological distress on tender points was not significant (Est. = .170, 95% CI = −.090, .499, p = .21) while the indirect (mediated) effect was significantly associated with tender points (Est. = .202, 95% CI = .038, .388, p = .014).

Conversely, SA did not mediate the effect of positive affect on tender points (mediated effect Est. = −.044, 95% CI = −.151, .027, p = .21) nor was there a significant direct effect of positive affect on tender points (Est. = −.068, 95% CI = −.239, .115, p = .41).

## 4. Discussion

The primary finding of this study is that a higher tender point count is most strongly associated with SA, controlling for measures of general psychological distress and positive mood. This is the first study to our knowledge to attempt to disentangle the association of SA from affective states in relation to the spatial distribution of pain sensitivity in a large community sample. These findings are similar to well-established associations between SA and other pain-related outcomes[18, 46] and extend previous work by demonstrating the primacy of SA even when accounting for affective processes frequently found to be comorbid with more tender points. While both general psychological distress and positive mood were associated with tender points in bivariate analyses, these associations were no longer significant in multivariate models including SA. The results of the mediation analyses confirm that some of the association between general psychological distress and the diffuseness of pain sensitivity on physical examination is due to levels of SA.

Evaluations of the association of a variety of pain outcomes with measures of affect and SA have revealed inconsistent results. Self-reported pain was not associated with SA scores in one study of 280 chronic pain patients when affective measures were also used in the analyses[52]; this may be because negative affective measures are more strongly associated with clinical pain reports than the distribution of pain sensitivity as measured by tender points, or because that study used raw scores, rather than extracted components. In contrast, a mediational analysis evaluating the association of SA and negative affect measures with clinical abdominal pain in schoolchildren outcomes found that SA was strongly associated with this measure, and that it mediated the association of depression with painful abdominal symptoms[36]; this finding is echoed by a study of adults with functional gastrointestinal symptoms in which SA was associated with all types of GI symptoms while depression was only moderately associated with non-painful symptoms[12]. A recent study of healthy individuals employing a factor analytic approach found that SA, but not negative or positive mood, was related to the qualitative evaluation of evoked pain[37]. The most comparable study to date examined patients with psychological distress and found that SA was associated with the likelihood of having a high number of tender points (>5),[45] though this study did not attempt to psychometrically isolate components of SA and psychological distress. Prospective analyses have demonstrated that SA is a strong predictor of the development of painful disorders, including TMD and widespread fibromyalgia-like pain/tenderness[23, 46]; these are critical findings as they suggest that SA is a risk factor for, rather than a consequence of, the development of chronic pain conditions.

The breadth of the MIDUS Biomarker study allowed for a variety of important controls that might contribute to peripheral nociceptive input. The physical examination and medical history allowed for assessment of chronic conditions that are characterized by musculoskeletal pain (e.g., arthritis) and abnormal joints or musculature that could might result in increased numbers of tender points. Elevated peripheral inflammation has previously been associated with increased pain sensitivity both under basal conditions[38], and during experimentally induced inflammation with immunogenic challenges[70] an effect that may be mediated by increases in negative affective processes[34]. Sympathetic nervous system activation measured by urinary catecholamine (as in the MIDUS study) has also been linked

to increased reports of musculoskeletal pain.[20] Various medications can also modulate pain responses. For instance, long-term use of opioids is suspected of promoting hyperalgesia in some patients,[67] antidepressants may be effective in pain relief for some conditions but not others[57, 63] and the chronic use of benzodiazepines is associated with chronic pain, though the association is not yet well understood.[40, 51] In these analyses the use of opioids, sedatives and antidepressants were all associated with a greater number of tender points, perhaps indicating neural modulation of pain or psychopathology, though determining the nature of the association is not possible in a cross-sectional study. However, controlling for all of the above factors *did not* eliminate the association of SA with tender points.

That SA was still strongly associated with tender point counts in the presence of a comprehensive set of covariates suggests that it is a construct of primary importance when evaluating widespread pain sensitivity by physical examination. One possibility is that SA is related to abnormal pain-evoked brain activity. While few neuroimaging studies have used SA as a construct in relation to imaging outcomes those that have seem to reveal distinct activity in pain networks associated with the construct. In patients with mood disorders, SA scores derived from the MASQ were associated particularly with resting state functional connectivity between the rostral anterior cingulate cortex (rACC) and the ventral striatum[53]; this is of interest to pain researchers because the rACC plays a central role in descending inhibitory pain networks,[3] and such networks have been found to be dysfunctional in chronic pain patients[50]. In diverticular disease patients, high SA is associated with less deactivation of ascending pain structures such as the ventral posterolateral thalamus and posterior insula, and less deactivation in pain affect structures such as the hippocampus and amygdala in anticipation of painful heat[59]. Taken together, these results suggest that SA may be associated with impaired inhibition of pain.

SA has also been associated with abnormal responses to experimental pain testing and sensory tasks. Higher SA is associated with lower heat pain tolerance in women with provoked vestibulodynia[61], greater soreness in the trapezius after rapid needle insertion withdrawal[44], a greater number of masticatory sites rated as painful by TMD patients[58], and lower orofacial pressure pain thresholds[11]. These evoked-pain outcomes are echoed by studies of sensation and interoceptive processes. SA is associated with increases in the false-alarm rate on the somatic signal detection task -- an experience of illusory touch – in healthy individuals, as well as those with both medically explained and medically unexplained chronic abdominal pain[6]. Similarly, SA is associated with a worse performance on a heartbeat detection ability task[42] and longer ERP latency following auditory cues[49]. These findings suggest that SA is not simply an increased awareness of or sensitivity to somatic sensations, but involves a distortion of attentional processes as well. There is also evidence that SA is associated with the gain control for other sensory modalities, as it has been shown to correlate with perceived unpleasantness of auditory tones.[28] It is worth noting that most of these studies used populations with clinical pain or mood disorders. Our findings suggest that SA is an important construct in relation to the distribution of pain sensitivity found on palpation in a community sample, and our secondary analysis indicated that this is so even when those with the highest levels of SA were removed.

### 4.1. Limitations

A significant limitation of this study is the absence of clinical pain measures. Previous research has revealed moderate associations between clinical pain measures and tender point counts (i.e., Pearson correlations between .4 and .6).[54, 73] These findings suggest that tender point counts, while related to clinical pain, are not a proxy for self-report measures. Our findings relate to the diffuseness of pain-sensitivity on manual palpation only – tender points – and it is possible that other measures of experimental pain evaluations might reveal substantial differences in the relationship between affective symptoms, SA, and other evoked-pain outcomes. For instance, the so-called "medial" pain system has been differentially associated with pain affect rather than sensory qualities of pain[64], and it is possible that general psychological distress or positive affect are associated with affective qualities of pain including clinical pain reports, while SA is associated more with lower thresholds or tolerance in experimental pain paradigms. The overall low prevalence of tender points in this sample limits inferences about those at the extreme end of this spectrum -- those with tenderness and pain across the body characteristic of fibromyalgia or related chronic pain conditions. Other important psychological constructs such as pain catastrophizing were not measured in the MIDUS Biomarker study, and we therefore cannot draw conclusions about their association with pain sensitivity on physical examination in this sample.

### 4.2. Conclusions and Future Directions

The present results indicate that somatic awareness is significantly associated with the distribution of pain sensitivity in the general community and suggest that this psychological construct mediates some of the association between affect and pain. SA should be more frequently assessed in both research and clinical settings; the availability of validated short forms of the MASQ should be helpful toward this end.[39, 66] SA's robust association with evoked pain on palpation, in a manner independent of the influence of mood, could make it particularly useful for gleaning information about the distribution of a patient's pain sensitivity without requiring attendance at the clinic. Psychosocial interventions in samples characterized by high levels of SA have demonstrated some success in reducing clinical pain levels;[24] whether these interventions would also reduce the spatial distribution of pain sensitivity revealed during clinical examinations is an open question. Future studies of pain in community samples might employ measures of SA and affect and relate them to a variety of experimental and self-report pain outcomes. For instance, pain tolerance or pain affect might have different relationships with SA and negative/positive mood than pain threshold and sensory pain ratings. Recent research suggests that simple evaluations of the diffuseness of pain are prospectively useful for predicting pain related outcomes and determining pain treatment responses following common procedures[7, 30]; therefore, measuring SA may contribute to better prediction of these outcomes and treatment responses.

## Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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## Acknowledgments

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## Highlights

- Somatic awareness is an emerging concept in pain research

- Somatic awareness is associated with higher tender point count

- Somatic awareness mediates the relationship between distress and tender point count
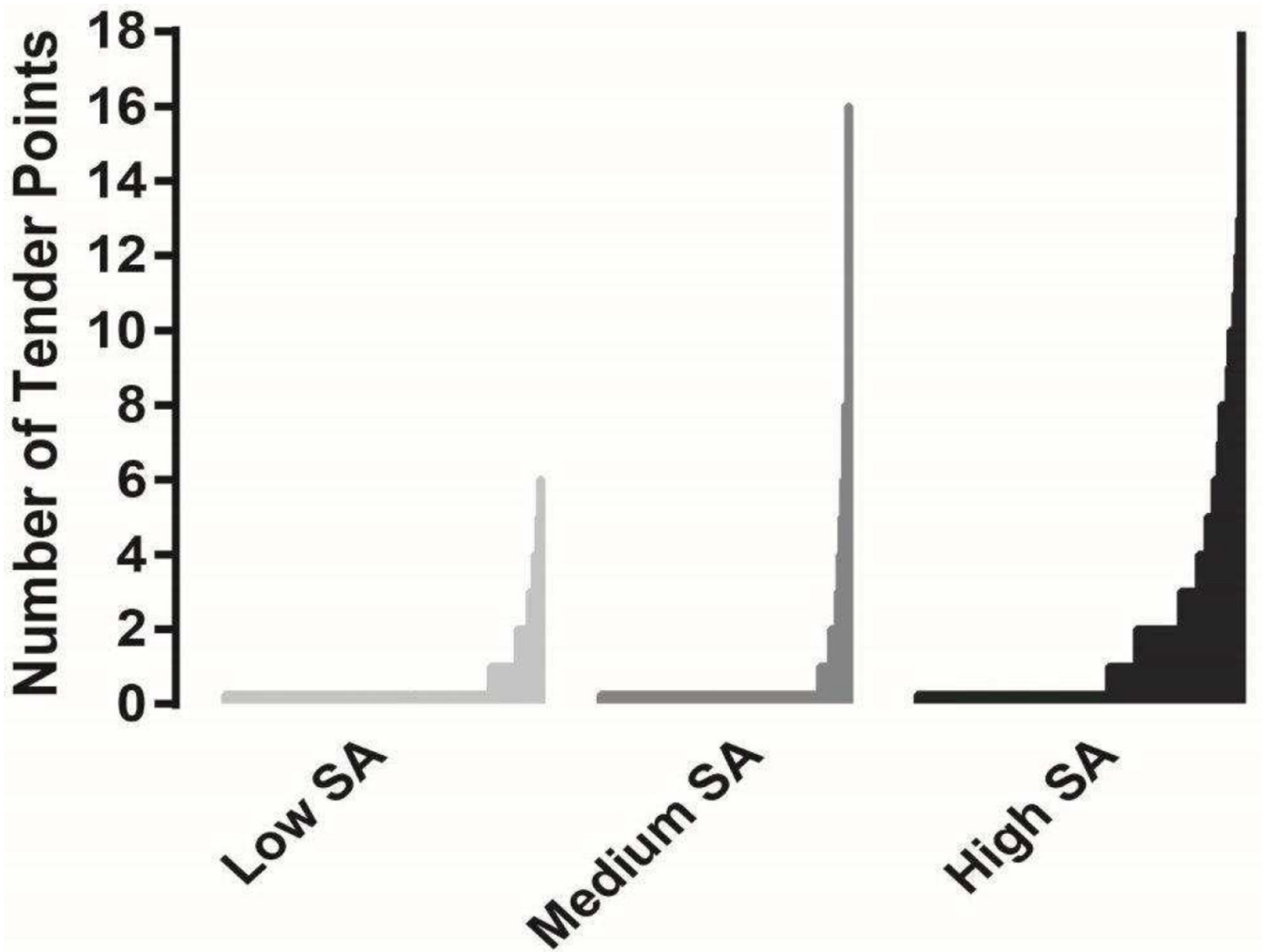
**Figure 1.**
Distribution of tender points by low, medium and high levels of somatic awareness (divided into tertiles by SA rank). Low group SA mean (SD) = 16.98 (.83), medium group SA = 19.85 (.78), high group SA= 27.56 (6.30).. Low group SA mean (SD) = 16.98 (.83), medium group SA = 19.85 (.78), high group SA= 27.56 (6.30).

**Table 1**

Participant characteristics and a comparison of those with and without tender points.

| Characteristic Mean (SD) | All (n=507) | 0 TPts (n=385) | 1 TPts (n =122) | $F_{1,505}$ | p |
|---|---|---|---|---|---|
| Age | 53.38 (11.74) | 52.71 (11.60) | 55.48 (11.99) | 5.20 | .023 |
| Somatic Awareness | 21.64 (6.02) | 20.46 (4.51) | 25.37 (8.28) | 69.86 | <.01 |
| Positive Affect | 40.72 (9.95) | 41.17 (9.62) | 39.30 (10.84) | 3.31 | .069 |
| General Psychological Distress | 24.41 (8.39) | 23.39 (7.12) | 27.62 (10.75) | 25.01 | <.01 |
| Body Mass Index | 30.70 (7.20) | 30.06 (6.90) | 32.70 (7.76) | 12.79 | <.01 |
| Number of Chronic Conditions | 4.17 (2.97) | 3.72 (2.73) | 5.58 (3.26) | 38.87 | <.01 |
| Sleep Efficiency (%) | 79.48 (10.37) | 79.73 (10.63) | 78.71 (9.55) | .74 | .39 |
| Blood C-Reactive Protein, µg/mL[a] | 0.25 (.51) | .20 (.50) | .40 (.50) | 14.35 | <.01 |
| Urine Norepinephrine, µg/g[b] | 1.37 (.20) | 1.35 (.19) | 1.42 (.19) | 11.14 | <.01 |
| Number of Rx Medications | 2.62 (2.87) | 2.24 (2.64) | 3.84 (3.22) | 30.49 | <.01 |
| Number of Tender Points | 0.80 (2.12) | NA | 3.34 (3.20) | NA | NA |
| | | % | | Pearson $\chi^2$ | p |
| Gender (Female) | 59 | 52 | 81 | 32.65 | <.01 |
| Using NSAID | 42 | 40 | 49 | 3.58 | .058 |
| Using Anxiolytic/Sedative | 5 | 2 | 14 | 25.61 | <.01 |
| Using Opiate/Opioid | 8 | 5 | 16 | 18.35 | <.01 |
| Using Corticosteroids | 6 | 6 | 8 | .97 | .33 |
| Using Antidepressants | 13 | 11 | 21 | 7.42 | <.01 |
| Abnormal Joint/Musculature | 38 | 32 | 55 | 20.86 | <.01 |
| Abnormal Sensation | 58 | 59 | 57 | .18 | .68 |
| Regular Exercise | 72 | 74 | 66 | 3.50 | .061 |
| Smoker[c] | 52 | 53 | 50 | .28 | .60 |

[a]log-transformed;

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[b] log-transformed, adjusted to urine creatinine;

[c] current or former

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**Table 2**

Correlation table showing associations between the MASQ subscales and other negative emotionality measures administered in the MIDUS biomarker study.

| | Perceived Stress Scale[13] | Spielberger Trait Anxiety[58] | Social Anxiety Scale[24] | CESD total[54] | CESD anhedonic/ vegetative subscale | CESD negative mood | CESD positive mood |
|---|---|---|---|---|---|---|---|
| MASQ Somatic Arousal | .460 | .497 | .274 | .574 | .533 | .550 | −.254 |
| MASQ General Distress | **.676** | **.697** | .353 | **.775** | .594 | **.786** | −.455 |
| MASQ Positive Affect | −.528 | −.561 | −.288 | −.587 | −.380 | −.444 | **.676** |

All correlations are significant, ($p$ <.01). Correlations above .6 are in bold.

**Table 3**

Multivariate negative binomial regression model for number of tender points.

| | Estimate | S.E. | Z value | p | exp | 95% CI, ll | ul |
|---|---|---|---|---|---|---|---|
| (Intercept) | −6.749 | 1.233 | −5.476 | <.001 | 0.001 | 0.000 | 0.014 |
| **Somatic Awareness** | 0.050 | 0.021 | 2.355 | **0.019** | **1.051** | **1.008** | **1.098** |
| General Distress | 0.025 | 0.017 | 1.519 | 0.129 | 1.026 | 0.991 | 1.062 |
| Positive Affect | −0.008 | 0.012 | −0.680 | 0.496 | 0.992 | 0.968 | 1.016 |
| **Age** | 0.028 | 0.011 | 2.455 | **0.014** | **1.028** | **1.005** | **1.052** |
| **Female** | 1.507 | 0.258 | 5.829 | **<.001** | **4.511** | **2.719** | **7.613** |
| C-reactive protein | 0.130 | 0.236 | 0.551 | 0.582 | 1.139 | 0.696 | 1.866 |
| Norepinephrine | 0.618 | 0.564 | 1.096 | 0.273 | 1.855 | 0.546 | 6.334 |
| **Using Sedative** | 0.942 | 0.448 | 2.105 | **0.035** | **2.565** | **1.068** | **6.652** |
| Using Anti-depressant | 0.567 | 0.300 | 1.890 | 0.059 | 1.764 | 0.966 | 3.296 |
| Using NSAID | −0.152 | 0.229 | −0.663 | 0.508 | 0.859 | 0.529 | 1.384 |
| **Using Opioid** | 0.731 | 0.355 | 2.059 | **0.040** | **2.077** | **1.015** | **4.492** |
| # of Chronic Conditions | 0.013 | 0.043 | 0.296 | 0.767 | 1.013 | 0.928 | 1.106 |
| **Abnormal Joint/muscle** | 0.526 | 0.227 | 2.318 | **0.020** | **1.692** | **1.070** | **2.685** |
| Body Mass Index | 0.027 | 0.015 | 1.768 | 0.077 | 1.027 | 0.995 | 1.062 |

Significant variables are in bold.