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Increased somatosensory amplification is associated with decreased pressure pain thresholds at both trigeminal and extra-trigeminal locations in healthy individuals

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Abstract

Background: The diagnosis of temporomandibular disorders (TMD) is based on patient history and physical examination, and may require medical imaging. Masticatory muscle palpation is essential to make a diagnosis of TMD. However, the response of masticatory muscles to mechanical pressure stimuli depends on many physical and psychological factors.

Objective: This study aimed at determining the impact of somatosensory amplification (SSA)—an estimate of somatic awareness and bodily hypervigilance—on pressure pain thresholds (PPTs) measured at both trigeminal and extra-trigeminal locations in healthy individuals.

Methods: PPTs were measured at the right anterior temporalis and superficial masseter, and the thenar eminence of the right hand in one hundred healhty individuals (69F, 31M), divided in three groups based on their SSA scores: low (N = 32), intermediate (N = 34) and high (N = 34). General linear models were used to test between-group differences in PPTs including sex as a covariate. The level of significance was set at $P < .05$.

Results: Individuals with high SSA had lower PPTs at the anterior temporalis than individuals with low ($P = .006$) and intermediate ($P = .001$) SSA. No significant between-group differences were found in PPTs measured at the masseter ($P = .372$). PPTs measured at the thenar eminence were significantly lower in the high than the low SSA group ($P = .009$). Females had lower PPTs at the masseter than males ($P = .021$) but not at other muscle locations (all $P > .05$).

Conclusion: Increased somatosensory amplification is associated with decreased pressure pain thresholds at both trigeminal and extra-trigeminal locations in healthy individuals. SSA could be a potential confounder while diagnosing TMD and evaluating treatment outcomes.

KEYWORDS
somatosensory amplification, masseter, temporalis, temporomandibular joint disorders, quantitative sensory testing

1 | BACKGROUND

Temporomandibular disorders (TMD), a set of pathological conditions affecting the muscles of mastication and/or the temporomandibular joint, have a complex multifactorial aetiology[1] and are the most common cause of chronic oro-facial pain.[2,3] Painful TMD of the muscles of mastication, that is TMD myalgia (mTMD), is the most common form of TMD and affects more than 45% of patients with a TMD.[4] According to the Diagnostic Criteria for TMD (DC/TMD),[5] the diagnosis of mTMD relies on patient pain history and clinical examination which includes palpation of the muscles of mastication, which should evoke a familiar pain in the muscle palpated.[5] During the clinical examination, masticatory muscle palpation helps the clinician understand whether the muscles are painful, whether peripheral sensitisation has occurred, and whether treatment is effective in alleviating pain and muscle tenderness.

Pressure algometry—a quantitative sensory testing (QST) method—quantifies the sensitivity to mechanical stimuli applied at a specific site.[6] It is generally used to measure the pressure pain threshold (PPT, the smallest pressure perceived by the patient as painful) at muscle locations in both clinical and research settings to monitor the effect of experimental and/or clinical interventions.[6,7] Decreased PPTs may suggest the presence of peripheral and/or central sensitisation. PPTs measured at the masseter and temporalis of individuals with mTMD are lower than those of healthy controls and decrease when TMD develop.[7-9] Several physical and psychological factors have been reported to affect thresholds to painful stimuli and PPTs of the masseter and temporalis muscles. Among those are sex,[10-12] hormones,[13] age,[14,15] ethnicity,[16,17] stress[18] and anxiety.[19]

It is well known that individuals with increased sensitivity to nociceptive stimuli delivered at extra-trigeminal locations are also more sensitive to nociceptive stimuli at trigeminal locations. For instance, heat pain thresholds measured at the arm and foot were found to correlate with PPTs measured at the masseter and the temporalis.[20] As well, individuals with increased sensitivity to cold stimuli in their hand experienced increased tooth pain when orthodontic separators were placed between their teeth.[21]

Somatosensory amplification (SSA)—the tendency to perceive normal somatic and visceral sensations as being relatively intense, noxious, and disturbing[22]—contributes to increasing pain sensitivity.[19,23] SSA can be measured in the clinical setting using the somatosensory amplification scale (SSAS),[24] which investigates how much an individual is bothered by various uncomfortable visceral and/or somatic sensations, including pain. As masticatory muscle palpation is routinely used to make diagnosis of TMD and to monitor the effects of treatment, understanding whether increased SSA is associated with decreased PPTs in the muscles of mastication could allow to increase the precision of diagnosis and evaluate more accurately patient response to therapeutic interventions. Also, as SSA has been reported to be increased in individuals with painful TMD,[25,26] it would be important to understand what it the impact of SSA on PPTs.

This study aimed at determining the impact of SSA on PPTs measured at trigeminal and extra-trigeminal locations in healthy individuals. Specifically, this study tested whether high SSA is associated with decreased PPTs at two major muscles of mastication, that is anterior temporalis and superficial masseter, and an extra-trigeminal location, that is the thenar eminence of the right hand. It was hypothesised that individuals with high SSA had lower PPTs at all muscle locations compared to those with low SSA.

2 | METHODS

2.1 | Participants

Healthy participants were recruited in the local community. The TMD pain screener was preliminary used to detect individuals with TMD symptoms (TMD pain screener score ≥ 3).[27] Those with a score < 3 were invited to the research laboratoy and submitted to a clinical examination including a TMD assessment according to the DC/TMD.[5] Exclusion criteria were wearing extended dental fixed prostheses (3 teeth or more), ongoing orthodontic and/or dental treatment, neurological disorders and/or depression, painful TMD and/or other orofacial pains (eg migraine, headache and tooth pain), report of chronic pain conditions, intaking drugs affecting the central nervous system and muscle relaxants, or intaking analgesics within 48 hours preceding the measurements. The clinical examination was performed by licensed dentists enrolled in the graduate programme in orthodontics at the University of Toronto under the senior investigator's supervision (IC), or by himself. All operators attended clinical sessions and frontal lectures about TMD clinical examination provided by the senior investigator (IC).

One hundred healthy individuals (69 females, 31 males, mean age ± SD: 25.6 ± 5.4 years) were recruited in the study.

2.2 | Psychophysical measurements

2.2.1 | Questionnaires

All participants completed a demographic questionnaire, the Somatosensory Amplification Scale (SSAS),[22,24] the Oral Behavior Checklist (OBC)[5,28] and the State-Trait Anxiety Inventory (STAI).[29]

The SSAS includes 10 statements investigating participants' sensitivity to bodily sensations using a 5-point scale (range of scores: 10-50). The respondent is asked to rate how much he/she is bothered by various uncomfortable visceral and/or somatic sensations, and includes statements like '*I hate to be too hot or too cold*', '*I am often aware of various things happening within my body*', *'Even something minor, like an insect bite or a splinter really bothers me'*, etc Participants chose among the following options: *'not at all'*, *'a little'*, '*moderately*', *'quite a bit'* or *'extremely'*, which corresponded to a score from 0 to 4.

TABLE 1 Demographic characteristics

	Age	SSA	OBC6	OBC	STAI (Y2)
Low SSA (N = 32)	27.5 ± 7.3[a] 27 [5]	19.5 ± 2.2[b] 20 [3]	5.0 ± 3.1 4 [4]	19.6 ± 7.9 18.5 [11]	38.0 ± 11.08 34.5 [11]
Intermediate SSA (N = 34)	25.6 ± 4.7 25.6 [9]	24.7 ± 1.7[b] 24.5 [3]	5.1 ± 3.6 4 [5]	19.6 ± 9.9 19 [12]	43.6 ± 14.9 39.5 [20]
High SSA (N = 34)	24.0 ± 2.9 [a] 24 [4]	33.7 ± 3.4[b] 33 [5]	7.1 ± 4.9 6 [7]	24.2 ± 11.5 21 [13]	46.7 ± 17.3 42 [25]
	$P = 0.025$[A]	$P < 0.001$[B]	$P = .160$	$P = .157$	$P = .062$

Note: Mean ± SD and median [IQR] scores for SSA (somatosensory amplification), OBC (oral behaviour checklist), OBC6 (oral behaviour checklist 6-item subscale) and STAI (Y2): trait anxiety. [A] [B]Between-group and [a] [b]post hoc statistically significant differences at $P < .05$.

The OBC includes 21 items assessing awareness and self-reported frequency of oral behaviours using a 5-point Likert scale (range of scores: 0-84). The STAI includes 20 items for assessing state anxiety (Y1) and 20 statements for assessing trait anxiety (Y2) using a 4-point Likert scale. The Y1 scale includes 20 statements evaluating feelings immediately prior to the experiment (range of scores: 20-80). The Y2 scale evaluates generalised anxious feelings and proneness to anxiety with 20 statements. Since correlations have been reported between SSAS and OBC, and SSAS and STAI,[19,25,30] for the purpose of this study, OBC and STAI (Y2) data were collected to account for a potential confounding effect on PPT outcomes in the statistical analysis.

2.2.2 | Pressure Pain Thresholds (PPTs)

Participants' PPTs were measured at both trigeminal (right superficial masseter and anterior temporalis muscles) and extra-trigeminal regions (thenar muscle on the palmar side of the right hand) using an electronic pressure algometer (Algomed, Medoc, Israel) with a rubber tip measuring 1 cm^2 with real-time visual feedback. The right side was chosen for convenience. All participants set on a chair with their head unsupported. The measurements were performed in a quiet room with controlled temperature. Only the research participant and the operator were in the room.

PPTs of the superficial masseter muscle were measured halfway between the origin and the insertion of the muscle and 1 cm posterior to its anterior boundary. PPTs of the anterior temporalis muscle were measured on the line from the top edge of the eyebrow to the highest point of the pinna of the ear and 2 cm behind the anterior margin of the muscle. PPTs of the thenar muscle were measured on the thenar eminence located on the palmar side of the hand, as previously done.[8,9,31] For all measurements, the algometer was positioned perpendicular to the skin. The operator placed the algometer tip on the respective site and applied pressure at a constant rate of 20 kPa/s using visual feedback on a computer monitor that the participant could not visualise. Participants were asked to press a button the moment the pressure stimulus applied to their muscles changed from a pressure sensation to a painful sensation (PPT). Each measurement was serially repeated at each muscle site four times, with a 1-minute interval between each measurement. The order of measurements was randomised.

All participants received financial compensation for their time in the form of a gift card or cash. The procedures were approved by the Research Ethics Board at the University of Toronto (#32797).

2.3 | Statistical analysis

Continuous variables were reported as means ± standard deviations (SD) or as medians and interquartile ranges [IQR] if not normally distributed. The SSA scores were used to construct three study groups based on tertiles: low SSA (SSA < 23), intermediate SSA (23 ≤ SSA ≤ 29) and high SSA (SSA > 29). PPT data were aggregated by computing the mean of the trials obtained at each location, after having discarded the first trial.

An a priori power analysis was conducted using G × Power (Heinrich-Heine-Universität Düsseldorf, Germany)[32] to test the difference between the three independent groups (SSA groups), after having included sex and the interaction sex by group in the model. The analysis showed that a total sample of 80 participants was required to achieve a power of 0.80 using a medium effect size ($d = 0.4$) and an alpha of 0.05.

Two separate general linear models were used to test between-group differences in PPTs measured at trigeminal (right superficial masseter muscle and anterior temporalis muscle) and extra-trigeminal (thenar eminence of the right hand) locations respectively, using sex as a covariate. The interaction sex by SSA group was preliminary tested and retained only in the first model because it was statistically significant. A sub-score (OBC6) was computed by summing six parafunctions listed in the OBC involving tooth-to-tooth contact and clenching as done previously.[19,25,33,34] Between-group differences in age, OBC, OBC6, STAI(Y2) were tested using Mann-Whitney tests. Post hoc analyses were adjusted using Bonferroni's method.

The level of significance was set at < 0.05. SPSS version 24 (IBM Corp. Released 2016, Armonk, NY: IBM Corp) was used for the analyses.

TABLE 2 Results from the general linear models

	PPT-RM	PPT-RT	PPT-RH
Group	0.999 (0.372)	4.898 (0.010)[A]	4.567 (0.013)
Group × sex	0.840 (0.435)	0.147 (0.863)	—
sex	5.556 (0.021)[A]	0.910 (0.434)	0.188 (0.665)

Note: F values and (*P* values) are reported. PPT (pressure pain threshold), RM (right superficial masseter), RT (right anterior temporalis) and RH (thenar eminence of the right hand). Two separate models were constructed, one for trigeminal locations (PPT-RM and PPT-RT) and one for the extra-trigeminal location (PPT-RH). The interaction Group -sex was not included in the statistical model using PPT-RH as dependent variable. [A]Statistically significant at $P < .05$.

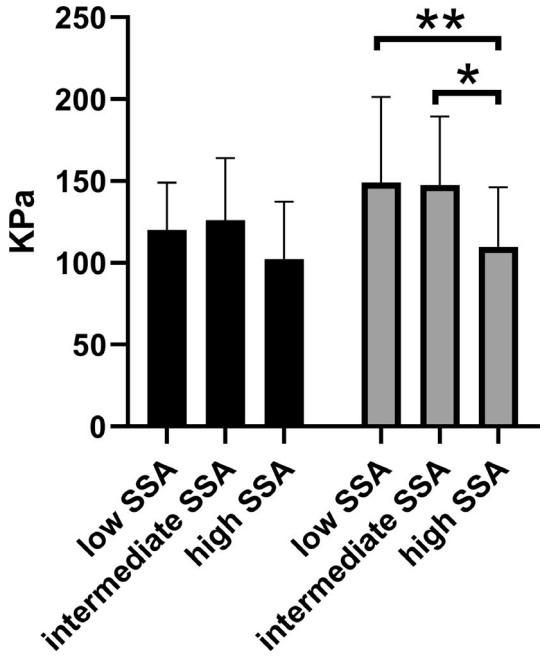

FIGURE 1 Pressure pain thresholds (PPTs) measured at trigeminal locations. Mean PPTs measured at the right superficial masseter (black) and anterior temporalis (grey) in the low, intermediate, and high somatosensory amplification (SSA) groups. The error bars indicate standard deviations. Significant difference at $^*P < .05$ and $^{**}P < .05$

3 | RESULTS

No participant withdrew from the study. The low, intermediate and high SSA groups included 32 (23 F, 9 M), 34 (19 F, 15 M) and 34 (27 F, 7 M) subjects, respectively. The mean age ± SD was 27.5 ± 7.3, 25.6 ± 4.7 and 24.0 ± 2.9 years respectively. Demographic characteristics, SSA, OBC and STAI (Y2) data, and between-group comparisons for these measures are reported in Table 1. There were no between-groups differences in OBC6 ($P = .160$), OBC ($P = .157$) and STAI (Y2) ($P = .062$) scores.

PPTs measured at the right superficial masseter, anterior temporalis and thenar eminence are reported in Figures 1 and 2. Results from the general linear models are reported in Table 2. A significant effect of SSA on PPTs measured at the anterior temporalis ($F = 4.898$, $P = .010$) but not on those measured at the superficial masseter ($F = 0.999$, $P = .372$) was found. PPTs measured at the anterior temporalis muscles were significantly lower in the high SSA (109.9 ± 36.1 KPa) than the low (149.1 ± 52.2 KPa; $P = .006$) and the intermediate SSA (147.6 ± 41.8 KPa; $P = .001$) groups. No differences were found between the intermediate and low SSA groups ($P = .999$). PPTs of the superficial masseter were 102.3 ± 35.1 KPa, 126.0 ± 38.0 KPa, and 120.1 ± 28.9 KPa in the high, intermediate and low SSA group, respectively.

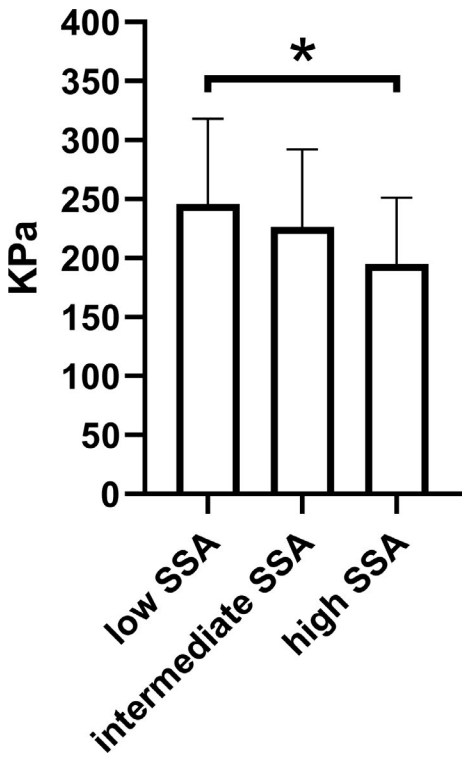

FIGURE 2 Pressure pain thresholds (PPTs) measured at extra-trigeminal locations. Mean PPTs measured at the thenar eminence of the right hand in the low, intermediate, and high somatosensory amplification (SSA) groups. The error bars indicate standard deviations. Significant difference at $^* P < .05$

Females had lower PPTs at the masseter (109.0 ± 35.1 KPa) than males (131.8 ± 31.8 KPa; $F = 5.556$, $P = .021$) but not at the temporalis (females: 131.8 ± 49.7 KPa; males: 142.8 ± 39.8 KPa; $F = 0.910$, $P = .343$).

PPTs measured at thenar eminence were affected by SSA ($F = 4.567$, $P = .013$). They were lower in the high (195.2 ± 56.1 KPa) than the low SSA group (245.9 ± 72.3 KPa, $P = .009$). No differences were found between the low and the intermediate (226.5 ± 65.8 KPa, $P = .769$) SSA groups. PPTs measured at the thenar eminence were not affected by sex (females: 219.2 ± 69.1 KPa; males: 230.7 226.5 ± 64.8 KPa; $F = 0.188$, $P = .665$).

4 | DISCUSSION

This study aimed to determine the impact of Somatosensory Amplification on PPTs measured at both trigeminal (anterior

temporalis and superficial masseter) and extra-trigeminal (thenar eminence) locations in healthy pain-free individuals. PPTs were collected in a large research sample including male and female participants, using a previously published protocol.[8,9,19] Since PPTs have been reported to be different between males and females,[11,12] sex was included as a covariate in the statistical models. The results of this study demonstrate that high SSA is associated with decreased PPTs measured at the anterior temporalis muscle and thenar eminence. PPTs in the participants with high SSA were approximately 35% lower than those measured in the other groups.

PPTs of the superficial masseter, anterior temporalis and thenar eminence were within normal ranges.[6,16] Although a significant between-group difference was found on PPTs measured at the anterior temporalis and thenar eminence, SSA did not influence significantly PPTs of the superficial masseter. This could be in part explained by the greater variation in PPT measurements at the superficial masseter than the anterior temporalis. Indeed, the coefficients of variation of PPTs measured at the superficial masseter of both the high and intermediate SSA groups were slightly greater than those measured at the anterior temporalis (high SSA: 34% vs 32%; intermediate SSA: 30% vs 28%).

Pressure algometry has been largely used to determine pain sensitivity to mechanical stimuli in clinical and research settings. Specifically, pressure pain thresholds (PPTs) have been commonly used to detect muscle tenderness and to study TMD.[6,35] Therefore, an accurate assessment of PPTs is of crucial importance in making a correct diagnosis and evaluating the response to treatment. To the best of our knowledge, this is the first study that has evaluated the effect of SSA on the PPTs of the muscles of mastication of healthy individuals. Studies have investigated the effect of an increased somatic awareness on PPTs measured at other body locations. Herbert et al found a significant correlation between pain vigilance,[36] which they measured using the Pain Vigilance and Awareness Questionnaire (PVAQ),[37] and PPTs measured at the knee and quadriceps of patients with knee osteoarthritis. Furthermore, they reported that pain hypervigilance was a predictor of temporal summation of heat pain, a marker of central sensitisation.[36] Grundtrom et al reported a significant correlation between pain sensitivity measured using the Pain Sensitivity Questionnaire (PSQ)[38] and PTTs measured at pelvic regions in patients with pelvic pain.[39]

Estimates of somatic awareness, such as SSA, are not commonly included as potential confounders in TMD research. Yet, individuals with self-reports of TMD pain had greater SSA scores than individuals without oro-facial pain complaints.[25] Similarly, Raphael et al reported higher SSA scores in individuals with chronic myofascial pain compared to controls.[26] Therefore, SSA may play an important role on TMD pain. However, the retrospective nature of these studies could not help in determining whether increased levels of SSA precede TMD pain or are the consequence of it. In a recent study, the potential role of SSA on pain sensitivity has also been examined with a longitudinal investigation. Individuals with high SSA scores submitted to a tonic painful stimulus lasting 5 days (orthodontic pain evoked by elastic separators) reported greater pain scores than individuals with low SSA.[19] Of interest, Sharma et al showed that individuals with enhanced sensitivity to heat pain had elevated injury-associated risk of TMD pain compared to people with low sensitivity to heat pain.[40] Therefore, measures of sensitivity to pain and SSA could help identify those individuals who may develop chronic pain.

For the purpose of this study, OBC and trait anxiety data were collected to account for potential confounders on PPT outcomes in the statistical analysis. In a previous investigation with a significant larger research sample (208 healthy individuals), a significant mild to moderate positive correlation was found between SSAS scores and each of OBC, OBC6 and trait anxiety.[25] However, in this study, significant between-groups differences in these measures were not found. Therefore, it is likely that SSA severity did not contribute to significant differences in the frequency of oral behaviours and trait anxiety in the current study.

The neurobiological mechanisms underlying SSA are minimally known. Self-reported SSA have been reported to be associated with long-latency potentials of auditory evoked related potentials, which suggests that SSA is related to cognitive processing.[41] Perez et al reported that SSA may be related to abnormal interactions across neural circuits mediating visceral-somatic perception, emotional processing/awareness and cognitive control.[42] They proposed a theoretical neural framework for SSA in somatoform illness. Specifically, key brain areas related to SSA may be the same brain regions related to negative expectation bias (anterior cingulate cortex, orbitofrontal cortex, insula, hippocampal formation and brainstem), negative attentional bias (anterior cingulate cortex, amygdala and dorsolateral prefrontal cortex) and pain catastrophising (anterior cingulate cortex, dorsolateral prefrontal cortex and insula).[42] However, the authors based their report on studies investigating somatoform disorders and it cannot be inferred whether this theoretical framework would apply to individuals without these conditions. Therefore, further studies should be designed to understand the neurobiological mechanisms of SSA.

Mechanical nociceptive stimuli in the masseter and temporalis muscles are transmitted by Aδ and C fibres through the third branch of the trigeminal nerve via the spinothalamic tract to the primary somatosensory cortex (S1). Top-down regulation from higher brain regions could facilitate or inhibit pain.[43,44] Therefore, it is possible that individuals with high SSA may present with reduced inhibition and/or increased facilitation of peripheral nociceptive inputs possibly due to a cognitive exacerbation of pain. The function of endogenous pain inhibitory pathways can be assessed in humans by condition pain modulation (CPM) paradigms.[45] In healthy individuals, PPTs of the muscles of mastication increase after the application of conditioning painful stimuli. Differently, conditioned pain modulation is impaired in patients with chronic TMD pain, which suggests dysfunctional endogenous pain modulation in these individuals.[46,47] Of interest, individuals with TMD pain have also increased SSA[26] and present with structural abnormalities in the central nervous system (CNS) correlated with pain duration.[43,48] Yet, whether increased SSA in healthy individuals could favour the onset of maladaptive nociplastic CNS changes in

the presence of prolonged trigeminal nociceptive stimuli (such as TMD) is currently unknown. Notably, results from the Orofacial Pain Prospective Evaluation and Risk Assessment (OPPERA) Study indicate that the degree of somatic awareness is a significant predictor of chronic TMD pain.[49] Therefore, further studies using conditioned pain modulation paradigms, QST and neuroimaging should be designed to study the impact of SSA on endogenous pain modulation and CNS structural changes observed in individuals with chronic TMD.

This study presents some limitations. First, the study sample included more females than male subjects. However, the statistical model accounted for the potential effect of sex, as it was included as a covariate in the statistical models, and the interaction sex by SSA group was preliminary tested. Second, participants' ethnicity was not included as a potential confounder in the statistical analysis, although it could have influenced PPTs.[16] This study was conducted in Toronto, Ontario, Canada, which is a multi-ethnic city. More than 20 different ethnicities were identified in the study sample. Therefore, correcting the analysis for this variable may have significantly affected the power of the study. There is evidence that SSA correlates with anxiety and with the self-reported frequency of oral behaviours.[25] Therefore, it may be argued that these variables could have affected PPT measurements. However, results from the statistical analysis demonstrated that the three SSA groups did not differ for OBC, OBC6 and trait anxiety scores. Therefore, it is unlikely that the frequency of self-reported oral behaviours and trait anxiety had affected the results. Finally, the mean age of the low SSA group was slightly higher than the other groups by about 3 years. Differences in PPTs of the masticatory muscles were found between groups of individuals aged 20-30 and over 65 years old[14] or when subjects were clustered in groups of 10 years,[15] and significant correlations between PPTs and age were found only in individuals above 65 years old.[14] Therefore, it is unlikely that the slight age difference between the two study groups affected the comparison of PPTs.

5 | CONCLUSIONS

Increased somatosensory amplification is associated with decreased pressure pain thresholds at both trigeminal and extra-trigeminal locations in healthy volunteers. Specifically, individuals with high somatosensory amplification show decreased pressure pain thresholds at the anterior temporalis muscle and the thenar eminence. Therefore, the degree of somatic awareness could be a potential confounder while diagnosing TMD—as it could potentially affect the response to masticatory muscle palpation—and evaluating treatment and research outcomes.

The neurological mechanisms underlying the impact of somatosensory amplification on sensory function remain elusive. It is possible that individuals with high somatosensory amplification may present with reduced inhibition and/or increased facilitation of peripheral nociceptive inputs possibly due to a cognitive exacerbation of pain. Behavioural and neuroimaging studies should be designed to identify the neural pathways contributing to increased pain sensitivity in individuals with increased somatic awareness, and whether SSA could favour the onset of maladaptive CNS structural nociplastic changes reported in patients with chronic TMD.[43,48]

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

AUTHOR CONTRIBUTIONS

Valerie E. Spano contributed to the conception and design, data collection and analysis, interpretation of data and drafting of the paper. Tina V. Imbriglio contributed to the conception of the study, data collection, interpretation of data and revision of the manuscript. Ka Chun (Jeremy) Ho contributed to the review of the topic, data collection and revision of the manuscript. Jeffrey CF Chow contributed to the conception and design, data collection, interpretation of data and revision of the manuscript. Iacopo Cioffi contributed to conception and design, analysis and interpretation of data, and draft of the paper. All authors approved the final version of the manuscript.

PEER REVIEW

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