Oral Sciences

Effect of somatosensory amplification and trait anxiety on experimentally induced orthodontic pain

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Cioffi I, Michelotti A, Perrotta S, Chiodini P, Ohrbach R. Effect of somatosensory amplification and trait anxiety on experimentally induced orthodontic pain.
Eur J Oral Sci 2016; 124: 127–134. © 2016 Eur J Oral Sci

The perception of pain varies considerably across individuals and is affected by psychological traits. This study aimed to investigate the combined effects of somatosensory amplification and trait anxiety on orthodontic pain. Five-hundred and five adults completed the State Trait Anxiety Inventory (STAI) and the Somatosensory Amplification Scale (SSAS). Individuals with combined STAI and SSAS scores below the 20th percentile (LASA group: five men and 12 women; mean age $\pm$ SD = 22.4 $\pm$ 1.3 yr) or above the 80th percentile (HASA group: 13 men and seven women; mean age $\pm$ SD = 23.7 $\pm$ 1.0 yr) were selected and filled in the Oral Behaviors Checklist (OBC). Orthodontic separators were placed for 5 d in order to induce experimental pain. Visual analog scales (VAS) were administered to collect ratings for occlusal discomfort, pain, and perceived stress. Pressure pain thresholds (PPT) were measured. A mixed regression model was used to evaluate pain and discomfort ratings over the 5-d duration of the study. At baseline, the LASA group had statistically significantly higher PPT values for the masseter muscle than did the HASA group. During the experimental procedure, the HASA group had statistically significantly higher discomfort and pain. A significant difference in pain ratings during the 5 d of the study was found for subjects in the HASA group. Higher OBC values were statistically significantly positively associated with pain. Somatosensory amplification and trait anxiety substantially affect experimentally induced orthodontic pain.

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Pain is an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage (1). In clinical realms where procedural pain is common and expected, it is easy to assume that all such pain is a direct consequence of, and in direct proportion to, the nociception activated by the clinical procedure (e.g. placement of a new orthodontic archwire) and to assume that the complexity of the just-stated definition applies only to more complex pain problems. However, when given the same stimulus, such as an initial archwire, it is clinically obvious that the perception of pain varies considerably across individuals – as predicted by the pain definition. For example, although the expected procedural pain of a new archwire is generally believed to be both relatively minor and self-limiting, some patients will report a different experience.

It is generally accepted that certain affective and cognitive behavioral factors contribute to these differences in individual pain perception (2, 3). For instance, and specifically relevant to the medical and dental settings, pain perception is influenced by factors such as somatosensory amplification and anxiety (4, 5). Somatosensory amplification refers to the tendency to perceive a given somatic sensation as intense, noxious, and disturbing (6), and such bodily attention is highly relevant in medical/dental settings. Trait anxiety refers to a general pattern of physical dysregulation and worry that is characteristic of an individual (7). An early observation was that trait anxiety might contribute to somatosensory amplification (6). And indeed, a number of studies have demonstrated that somatosensory amplification is correlated with several indices of general distress, including anxious and depressive symptoms (8–10). These observed correlations range between 0.28 and 0.54, indicating a potential relationship between these two constructs that is both theoretically and clinically important.

A common source of acute, but self-limiting, discomfort or pain is routine orthodontic procedures (5, 11, 12). The extent of pain associated with these procedures varies considerably across individuals, just as any pain varies. Yet, the reason why certain individuals are more pain sensitive than others to the same orthodontic stimulus remains relatively poorly understood, although progress has been made with respect to specific variables in specific contexts (5, 13, 14). For example, anxiety appears to influence the perception of

orthodontic pain (11), and individuals with prolonged pain during orthodontic treatment exhibit higher anxiety scores than do individuals with pain of short duration (15). In contrast, the role of somatosensory amplification in affecting pain perception within individuals receiving orthodontic treatment has not, to our knowledge, been investigated. And, following the early observations of BARSKY (6), somatosensory amplification and anxiety would appear to have a reciprocal effect on each other, and consequently the combined effects of somatosensory amplification and anxiety appear to be a sensible domain to investigate factors that affect the discomfort or pain associated with orthodontic tooth movement.

Associations between oral parafunctional behaviors and orofacial pain, and between oral parafunctional behaviors and anxiety, have also been observed (16, 17), but whether oral parafunction as a trait behavior contributes to the pain or anxiety, whether oral parafunction is a mediating variable, whether oral parafunction is a consequence (such as of pain or of anxiety), or whether oral parafunction is tied into a complex process (such as fear-avoidance), are presently unknown (18).

Finally, little is known regarding whether anxiety and somatosensory amplification might be responsible for differences in pain sensitivity between the trigeminal and extra-trigeminal sites and whether orthodontic pain may contribute to affect pain sensitivity in these locations.

Hence, the aim of this study was to evaluate the perception of orthodontic pain and occlusal discomfort induced experimentally in individuals with high or low combined scores of somatosensory amplification and trait anxiety, to examine the combined role of these variables in acute pain perception in an orthodontic treatment context, and to test whether orthodontic pain may affect pain sensitivity at trigeminal and extra-trigeminal locations.

It was hypothesized that individuals with high vs. low scores of somatosensory amplification and trait anxiety perceive experimentally induced orthodontic pain differently. A secondary hypothesis was that higher levels of oral parafunctional behaviors would occur in individuals with high combined scores of somatosensory amplification and trait anxiety and might react more strongly to nociception (17).

Material and methods

The Somatosensory Amplification Scale (SSAS) (6) and the State-Trait Anxiety Inventory (STAI; form Y) (7) were provided to 505 students at the University of Naples, Federico II, Italy. The distributions of each of the SSAS and trait anxiety scores of the STAI were assessed in order to select subgroups with extreme scores. Individuals ($n = 122$) who scored below the 20th percentile (cut-off values: SSAS $\leq$ 12; STAI $\leq$ 19) or above the 80th percentile (cut-off values: SSAS $\geq$ 23; STAI $\geq$ 28) of each respective instrument were selected. Four categories of score distributions were then identified and individuals were assigned as follows: 49 subjects in the High SSAS/High STAI group; 53 subjects in the Low SSAS/Low STAI group; 11 subjects in the High SSAS/Low STAI group; and nine subjects in the Low SSAS/High STAI group. These individuals were then contacted regarding study participation and screened. Exclusion criteria included: previous or current orthodontic treatment; presence of fixed or removable dentures; one or more missing teeth (with the exception of third molars); active psychiatric disorders; or using medications acting on the central nervous system (Fig. 1). After the screening procedure, exclusion from the study and refusal to participate resulted
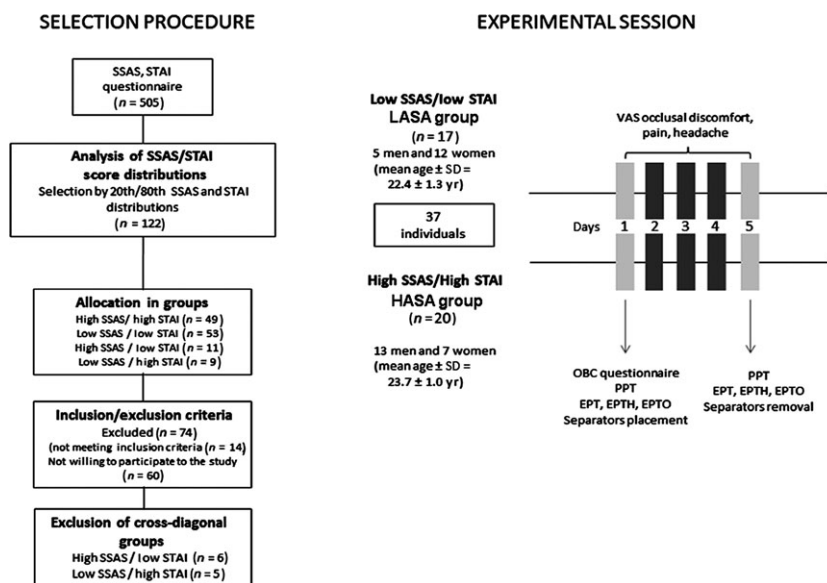

Fig. 1. Selection procedure and experimental design. EPT, electrical perception threshold; EPTH, electrical pain threshold; EPTO, electrical pain tolerance; OBC, Oral Behaviors Checklist; PPT, pressure pain thresholds; SSAS, Somatosensory Amplification Scale scores; STAI, State Trait Anxiety Inventory (Trait anxiety scores); VAS, visual analog scale scores.

in 60% subject loss, and only 48 individuals of the initial 122 identified on the basis of extreme SSAS or STAI scores were eligible for the experimental procedure. The Low SSAS/Low STAI (LASA) group included 17 people (five men and 12 women; mean age ± SD = 22.4 ± 1.3 yr), and the High SSAS/High STAI (HASA) group included 20 people (13 men and seven women; mean age ± SD = 23.7 ± 1.0 yr). As the remaining number of people belonging to each of the Low SSAS/High STAI ($n = 6$) and High SSAS/Low STAI ($n = 5$) groups was very low, these groups were excluded from further participation in the study.

The study was carried out as a single-blind longitudinal design (Fig. 1). For classification purposes of the study sample, a clinical examination, according to Axis I of the Research Diagnostic Criteria for Temporomandibular Disorders (RDC/TMD) (19), was performed by a single examiner (A.M.) who was calibrated for RDC/TMD diagnosis in a previous study (20) and who was blind to the allocation of subjects in the study group.

After psychophysical measurements were obtained (see below), permanent maxillary and opposing mandibular first molars, each with firm mesial and distal proximal contacts, were identified; if the first molars on both sides exhibited these characteristics, then the experimental side was selected randomly by using a custom-made Java Script. Orthodontic elastic separators (American Orthodontics, Sheboygan, WI, USA) were applied mesial and distal to the selected teeth. Each participant was asked to keep the orthodontic elastic separators in place for 5 d, was provided with a symptom diary, and was asked to report the intensity of pain and occlusal discomfort (that is the occlusal disturbance induced by the separators) induced by the orthodontic elastic separators at five specified time-points during each day (8.00 h, 12.00 h, 16.00 h, 20.00 h, and before going to sleep). At each time-point, the participant was also asked to report perceived daily stress independently from the positioning of the orthodontic elastic separators; stress ratings were a planned covariate for this study. Each of the three constructs was rated on a 100-mm visual analog scale (VAS) with construct-relevant end points (21); for example, the end point for pain at 0 mm was 'no pain' and at 100 mm was 'the worst pain I can imagine'. Similar end-point VAS anchors were used for discomfort and stress. Instructions regarding the symptom log were provided, and the participant was dismissed. After 5 d, participants returned to the laboratory, and the examination and the psychophysical measurements were repeated. Thereafter, the orthodontic elastic separators were removed and participants were debriefed.

Psychophysical measurements were collected before and after the experimental session at different trigeminal and extra-trigeminal locations. Pressure pain thresholds (PPT) were assessed using an electronic algometer (Somedic, Hörby, Sweden) equipped with a rubber tip (of surface area 1 cm^2). The device was positioned perpendicular to the skin at the selected site and the pressure was increased at approximately 30 kPa s^{-1}. The PPT was determined as the point at which the pressure stimulus changed from a sensation of pressure into a sensation of pain (22). The participant indicated the PPT by pressing a button, which froze the current pressure value on the digital display. The procedure was explained to the participant, who was asked to keep their muscles relaxed during the measurements. Pressure pain thresholds were assessed by a single blind examiner (S.P.). All measurements were taken at three locations on the same side of the face that the orthodontic elastic separators had been placed. The measurement sites were selected on each muscle as follows. For the masseter muscle, the site was located midway between the origin and the insertion, 1 cm posterior to its anterior boundary. For the temporalis muscle, the site was located on the line from the top edge of the eyebrow to the highest point of the pinna of the ear, 2 cm behind the anterior margin of the muscle, as determined by palpating the muscle during voluntary contraction. For the thenar muscle, measurements were made on the skin of the palmar side, on the thenar eminence. The measurements were repeated for a total of three trials at each muscle, with a 1-min interval between trials. The order of measurements was randomized across participants.

The same examiner recorded the electrical perception threshold (EPT), the electrical pain threshold (EPTH), and the electrical pain tolerance (EPTO) to cutaneous stimulation using the Pain Matcher device (Cefar Medical, Lund, Sweden). This microprocessor device provides constant current (15 mA) in monophasic rectangular pulses at random velocity with a frequency of 10 Hz to the electrodes. The EPT is defined as the electrical stimulus perceived by the participant as pulses in the thumb and the index finger. The EPTH is defined as the electrical stimulus perceived by the participant as painful. The EPTO is defined as the most painful electrical stimulus that the participant can tolerate. The participant was instructed regarding the procedure and to then hold the device in their left hand and press the electrodes with the thumb and index finger of the right hand. Three trials were made for each of EPT, EPTH, and EPTO. To minimize any sensitization phenomenon, there was a 30-s interval between each trial during the measurements of EPT and EPTH, and a 60-s interval between each trial during the measurements of EPTO.

The extent of oral parafunctions was evaluated using the Oral Behaviors Checklist (OBC) (23). The OBC was scored using only items 3, 4, 5, 10, 12, and 13, which assessed, respectively, during waking hours, the following: grinding; clenching; pressing; biting or playing with soft tissue; holding objects between the teeth; and use of chewing gum. The rationale for using only these items was that these oral behaviors are characterized by pressing attitudes against soft tissues, objects, or teeth, whereas the others behaviors included in the OBC do not. A mean score of the six items was computed.

The local Ethics Committee approved the study protocol, and each participant signed an informed consent. The study participants received no financial compensation for participation and were assured that they could withdraw from the study at any time.

Statistical analysis

Continuous variables were reported as mean ± SD. The interaction between gender and study group was tested in all of the statistical models because gender may significantly influence pain perception (24).

The psychophysical measurements were reduced at each time point by computing the mean of the three trials obtained at each PPT location and by computing the mean of the three trials for each electrical stimulation construct. To evaluate baseline associations between psychophysical measurements and participant psychologi-

cal characteristics (as defined by study group) before the experimental intervention, a set of separate multiple regression models was used. Baseline PPT, EPT, EPTH, and EPTO values were considered as separate dependent variables for the respective models, and study group, gender, and perceived stress (from the symptom diary) were used as independent variables. First-order interactions between study group and gender were retained in the final model when evaluating PPT at thenar eminence because it was statistically significant.

A second set of multiple regression analyses was used to evaluate changes from baseline recordings for each of the dependent variables examined (PPT, EPT, EPTH, and EPTO); the same independent variables reported above were used in the models to control for individual differences at baseline.

Mixed-effects regression models were used to evaluate the trajectories of discomfort and pain (main outcome variables) during the five recording days in the two study groups. These models used all available VAS data collected over the 5 d, and they properly account for inter-correlations between repeated measurements (25). Recording day, daily time point, gender, study group, perceived stress, and the interaction between day and study group were used as independent variables in each of the examined models. Only the interaction between day and group was statistically significant for all the outcome measurements examined (all $P < 0.05$), and consequently it was the only interaction retained in the model. A second set of models was computed as a sensitivity analysis in which the OBC score was included as a covariate. This was not the case for the main analysis to avoid possible over-adjustment effects.

A similar mixed-effect regression model was used to evaluate the trajectories of stress during the five recording days in the two study groups.

The sample-size determination was based on a study by KROPMANS *et al.* (26), who reported that the smallest detectable clinical difference in pain perception, as measured on a VAS, was 28 mm for current pain intensity in the temporomandibular joint area. We used a value deriving from temporomandibular pain studies because, to our knowledge, the smallest detectable difference for orthodontic pain on a VAS has never been reported. We had assumed that a difference of 20 mm might be used for the sample size determination and that 14 participants per group were sufficient to obtain 80% power in our study (mean difference between groups = 20 mm; SD = 20 mm; $\alpha = 0.05$).

Data were analyzed using SAS version 9.2 (SAS Institute, Cary, NC, USA).

Results

All individuals were TMD-free at baseline according to the RDC/TMD. No individual developed signs or symptoms of TMD during the experimental phase, thus ruling out TMD symptoms as contributing to the pain reported across the experimental period.

Descriptive statistics and between-group comparisons, after adjusting for gender, PPT (temporalis, masseter, and thenar), EPT, EPTH, and EPTO at baseline (before orthodontic separator placement), are reported in Table 1. No significant between-group differences or

interactions were found for the temporalis muscle. The LASA group had higher PPT values than the HASA group ($P = 0.011$) for the masseter muscle; gender did not affect this relationship. A significant interaction between gender and group ($P = 0.023$) was found for PPT measured at thenar eminence, with the PPT being higher in men ($P = 0.003$). No significant between-group differences were found for EPT, EPTH, and EPTO at baseline (Table 1).

Changes in PPT, EPT, EPTH, and EPTO from baseline to the fifth day (as calculated as difference scores between day 5 and baseline) did not differ between groups (all $P > 0.05$).

The trajectories of the daily diary-based reports of pain and occlusal discomfort, from baseline to the fifth day, are shown in Fig. 2. For each of the scores, a significant interaction between day and group was found (Table 2). Specifically, during the 5 d of the study, the HASA group had higher VAS scores for discomfort and pain than the LASA group, and the patterns differed with time (Fig. 2). The interaction group*day was significant for both discomfort ($P = 0.001$) and pain ($P < 0.0001$). Post hoc analyses for the variations across the 5 d of the study showed no differences between days in the LASA group for pain and occlusal discomfort, whereas a significant variation was found in the HASA group for pain ($P = 0.0007$) but not for discomfort ($P = 0.07$, Fig. 2, Table 2). In particular, for the HASA group pain was higher at day 1 and decreased subsequently. In a sensitivity analysis, when

Table 1

Descriptive statistics and between-group comparisons for pressure pain thresholds (PPT) (masseter, temporalis, and thenar), electrical perception threshold (EPT), electrical pain threshold (EPTH), and electrical pain tolerance (EPTO) at baseline, before orthodontic separator placement (day 1)

Measurement	LASA	HASA	P
PPT masseter (kPa)	189.1 ± 45.2	146.5 ± 51.5	**0.011**
PPT temporalis (kPa)	185.5 ± 50.2	163.1 ± 42.5	0.154
PPT thenar* (kPa)	242.5 ± 76.6	193.4 ± 70.9	0.228
Male subjects	280.9 ± 56.7	197.3 ± 73.2	**0.003**
Female subjects	158.1 ± 31.4	185.6 ± 71.2	0.470
EPT	4.4 ± 0.3	4.1 ± 0.3	0.165
EPTH	11.2 ± 5.8	9.4 ± 5.1	0.323
EPTO	22.9 ± 13.6	18.5 ± 13.5	0.315

HASA, individuals with combined STAI and SSAS scores above the 80th percentile; LASA, individuals with combined STAI and SSAS scores below the 20th percentile; SSAS, Somatosensory Amplification Scale; STAI, State Trait Anxiety Inventory.
Values are given as mean ± SD. Baseline PPT, EPT, EPTH, and EPTO values were considered as separate dependent variables for the respective statistical models; study group, gender, and perceived stress (from the symptom diary) were used as independent variables. First-order interactions between study group and gender were retained in the final model when evaluating PPT at thenar eminence because it was statistically significant. Values given in bold type are statistically significant.
*Owing to a significant interaction between gender and group ($P = 0.023$), comparisons are reported separately for male and female subjects.
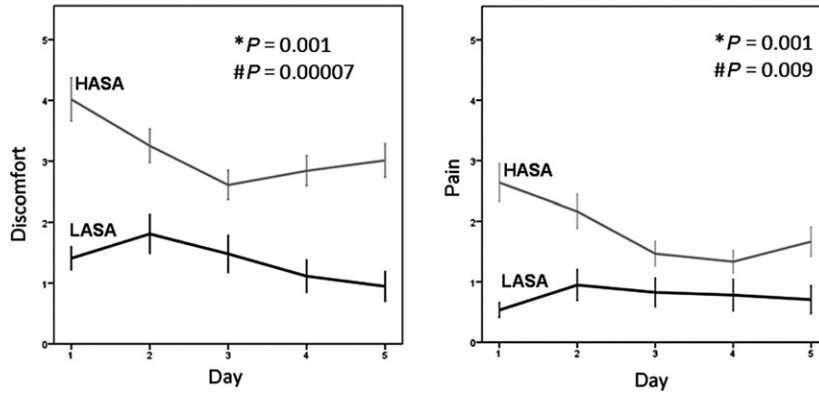
Fig. 2. Occlusal discomfort and pain during the experimental session. Visual analog scale (VAS) scores (cm) over the 5 d of the experimental phase of the study are reported in individuals with combined State Trait Anxiety Inventory (STAI) and Somatosensory Amplification Scale (SSAS) scores below the 20th percentile (the LASA group) (black line) and individuals with combined STAI and SSAS scores above the 80th percentile (the HASA group) (gray line). The error bars indicate the standard error of the mean. *Overall significant difference between groups. #Overall significant difference between days within the HASA group.

Table 2

Results from the longitudinal analysis

Covariates	d.f.	Discomfort F value (P value)	Pain F value (P value)
Day	4	1.46 (0.2195)	**2.80 (0.0285)**
Time point (day)	20	0.31 (0.9986)	0.36 (0.9961)
Gender	1	0.36 (0.5543)	2.80 (0.1032)
Group	1	**5.99 (0.0197)**	**4.58 (0.0395)**
Group*Day	4	**4.94 (0.0010)**	**6.17 (0.0001)**
Day – LASA	4	1.97 (0.1036)	0.96 (0.4310)
Day – HASA	4	2.19 (0.0733)	**5.20 (0.0007)**

HASA, individuals with combined State Trait Anxiety Inventory (STAI) and Somatosensory Amplification Scale (SSAS) scores above the 80th percentile; LASA, individuals with combined STAI and SSAS scores below the 20th percentile; d.f., degrees of freedom.

Values given in bold type are statistically significant.

the OBC score is included as a covariate, similar results were produced (data not shown).

The perceived stress scores are shown in Fig. 3 and the longitudinal analysis on perceived stress is reported in Table 3. A significant interaction between day and group was found. The HASA group had higher VAS scores for stress than the LASA group ($P = 0.002$), and the pattern of stress differed across time only in the HASA group ($P = 0.009$).

The subscale OBC scores were higher in the HASA group (9.43 ± 3.30) than in the LASA group (5.81 ± 2.60, $P < 0.001$). An increase of OBC value was positively associated with pain ($P = 0.042$).

Discussion

To our knowledge, this is the first study that aimed to evaluate the combined effect of somatosensory amplification and trait anxiety on orthodontic pain perception. The evaluation of these constructs could be of interest possibly to identify individuals who may be more sensitive to pain and discomfort during orthodontic therapy. Indeed, different levels of anxiety and somatosensory amplification might account for the high interindividual variability of pain and/or occlusal discomfort perception observed in orthodontic patients during treatment (11, 12, 27). The results of the current study further confirm that orthodontic pain is highly variable among individuals and reveal that orthodontic pain perception is significantly greater in individuals who exhibit the combined effects of high levels of both trait anxiety and somatosensory amplification vs. low levels of both variables. This is in agreement with previous reports, which suggest that pain perception is influenced by certain affective and cognitive behaviors (2), such as anxiety (5) and somatosensory amplification (4).

We evaluated somatosensory amplification and trait anxiety in combination because mood states and psychosocial stress can influence somatosensory amplification (28). Our justification in this initial study for evaluating the joint effects of trait anxiety and somatosensory amplification was further supported by the observed correlation of 0.37, which is consistent with previous literature (8–10). Owing to this reciprocal effect between trait anxiety and somatosensory amplification, it was decided to include only individuals with extreme values in both constructs for experimental efficiency. In addition, the cross-diagonal groups (Low SSAS/High STAI and High SSAS/Low STAI) comprised, from this population sample, a relatively small number of individuals – that is, the individuals willing to participate in the study accounted for only 2% (11 of 505) of the target population.

For the current research, orthodontic elastic separators were used as a model for evaluating experimental occlusal discomfort and pain, as performed previously (5), as they produce a time course of pain already described in the literature (29). It is generally known that orthodontic elastic separators cause the greatest pain on the first 2 d after insertion, and that pain starts
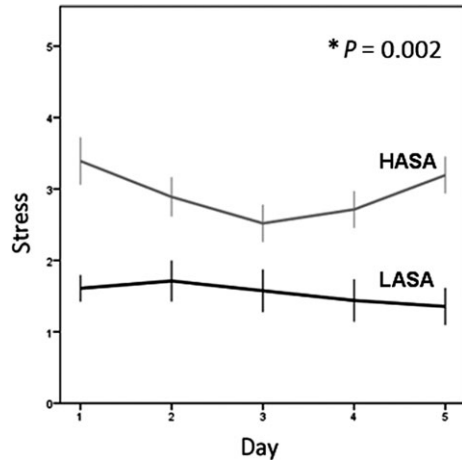
Fig. 3. Stress during the experimental session. Visual analog scale (VAS) scores (cm) over 5 d are reported in individuals with combined State Trait Anxiety Inventory (STAI) and Somatosensory Amplification Scale (SSAS) scores below the 20th percentile (the LASA group) (black line) and individuals with combined STAI and SSAS scores above the 80th percentile (the HASA group) (gray line). The error bars indicate the standard error of the mean. *Overall significant differences between groups.

Table 3

Results from the longitudinal analysis on perceived stress

Covariates	d.f.	Perceived stress F value (P value)
Day	4	0.97 (0.4254)
Time point (day)	20	0.40 (0.9918)
Gender	1	0.79 (0.3809)
Group	1	**5.47 (0.0253)**
Group*Day	4	**2.99 (0.0214)**
Day – LASA	4	0.68 (0.6060)
Day – HASA	4	**3.52 (0.0092)**

HASA, individuals with combined State Trait Anxiety Inventory (STAI) and Somatosensory Amplification Scale (SSAS) scores above the 80th percentile; LASA, individuals with combined STAI and SSAS scores below the 20th percentile. d.f., degrees of freedom.

Values given in bold type are statistically significant.

to decrease therafter, reaching minimum values between days 5 and 7.

Individuals in the HASA group had higher pain following the insertion than did those in the LASA group. This might be explained, in part, by the different perceived stress found in the two groups. Indeed, emotional stress affects the painful experience (30). Different pain VAS trajectories (Fig. 2) during the 5 d of the study were found in the two groups. Subjects in the HASA group reported peak scores for pain immediately on day 1, whereas those in the LASA group reported peak scores on day 2 (and which were, notably, only a modest increase relative to those of day 1, as commonly reported in the literature) (11, 13, 28, 29). The discrepancy in pain ratings between groups could be related to a more pronounced attentional bias in anxious individuals toward a potentially threatening stimulus represented by the insertion of the orthodontic elastic separator (31).

This process could have started as early as the informed consent procedure, during separator placement, or immediately after placement, and can be interpreted as a possible nocebo effect, that is the phenomenon in which mere suggestions actually bring about negative effects in a research participant. Indeed, it has been reported that negative verbal suggestions induce anticipatory anxious behavior about an impending pain increase, and this verbally induced anxiety triggers the activation of cholecystokinin which, in turn, facilitates pain transmission (31). This also produced characteristic U-shaped VAS trajectories for pain in the HASA group but not in the LASA group, that were paralleled by a similar pattern of stress trajectories. No effect of the five specified time points on pain and discomfort ratings was found, in accordance with a

previous study, in which orthodontic pain was examined through a longitudinal experimental design (12).

The orthodontic elastic separators caused occlusal discomfort in both groups, which was to be expected based on tooth movement, but also which was higher in the HASA group than in the LASA group, as predicted by our hypothesis. It is likely that individuals in the HASA group had a tendency to focus more and to be hypervigilant about the orthodontic elastic separators positioned between the interproximal surfaces of the teeth, the resultant sensations associated with periodontal ligament stretching and compression, and any perceived occlusal changes as a result of tooth movement. However, the statistical analysis failed to find a U-shaped profile of the VAS trajectories, as reported for pain. It is possible that this difference is related to the fact that participants were more oriented to focus on pain sensation than on discomfort while completing their daily diaries. The response of the HASA group is likely to point to occlusal hypervigilance that, as a trait, is characterized by a persistent heightened attentional focus on weak and infrequent sensations, and hypervigilance is also characterized by the additional increased focus on somatic sensations interpreted as potentially more alarming, threatening, and disturbing (32). Anxious individuals are more vigilant and have a lasting tendency to direct their attention to threat (33). Consequently, the combination of hypervigilance and anxiety appears to be additive. Whilst these assumptions probably point to a cause–effect relationship between anxiety, somatosensory amplification (cause), and increased pain perception (effect), it should be also considered that heightened body awareness (i.e. increased somatosensory amplification and hypervigilance) and anxiety could be a consequence of heightened sensitivity to pain stimuli as a result of, for example, genetic influences (34).

At baseline, individuals in the HASA group had a lower PPT at the masseter than did those in the LASA group. This could be attributed to the higher anxiety and stress levels of subjects in the HASA group (33, 35). In contrast, no between-group differences were observed

for the temporalis muscle, in agreement with the results of a previous study which showed that the temporalis is less affected by pain than is the masseter during low-level clenching experimental tasks (36). During clenching, masseter and temporalis are similarly recruited, but during incisal biting masseter muscles are dominant over the temporalis muscles (37). Incisal biting (for example, during nail biting) seems to be related to anxious and depressed personality disposition (38, 39). This, in turn, might have caused a difference in pain outcomes only for the masseter muscle. Also, similarly to a previous study (40), women had a lower PPT at thenar than men. Potential explanations for this gender difference include psychosomatic, environmental, and hormonal factors (41, 42).

Electrical pain thresholds measured at extra-trigeminal locations were within the ranges of a previous study (43) and were slightly higher in the LASA group than in the HASA group, but the difference was not statistically significant. In contrast to other studies, in which women were more sensitive to electrical pain than men (44, 45), threshold and tolerance for electrical stimuli were not affected by gender in this study, probably because of the different methodologies used to provoke pain with electrical stimuli. Also, discrepancies between the pressure and electrical responses at extra-trigeminal sites might be related to the involvement of different neuronal fibers during the sensory tests.

Finally, baseline psychophysical measurements were not significantly affected by the experimental procedure. These results suggest that anxiety and somatosensory amplification might be related to increased pain sensitivity at both trigeminal and extra-trigeminal sites, and that experimental orthodontic pain did not affect pain sensitivity in these systems.

This study has some limitations. First, it does not provide information about the cross-diagonal groups who exhibited a high value in only one of the two constructs of somatosensory amplification or trait anxiety. Consequently, the present study is unable to separate the effects of these two constructs. The findings of this study, however, lead readily to subsequent studies in which the effects can be analyzed separately. A second limitation is that the sample size was too small to provide an adequate statistical analysis of oral parafunctions as a mediating variable and how that might interact with the constructs of primary interest. The current analyses do indicate that it is likely that oral parafunctions can be part of the pathway between nociception induced by orthodontic separators and reported pain. A third limitation is that the present study does not evaluate the effects of somatosensory amplification and trait anxiety on pain reported during orthodontic treatment in the long term and whether orthodontic treatment pain contributes, in susceptible individuals, to TMD (13). Moreover, this is probably the topic of greatest importance to orthodontic clinicians and which has treatment implications, such as whether initial consultations for orthodontic treatment should include consideration of behavioral constructs and, as indicated, include behavioral medicine treatment, such as anxiety management and symptom perception management, for susceptible individuals. Fourth, the experimental design did not take into account gender-related hormonal factors and the periodicity of the menstrual cycle in women, which might have influenced pain sensitivity throughout the experimental phases (41). However, it is interesting that the HASA group included more male subjects than female subjects, whereas the LASA group included a greater number of female subjects. As a consequence of this gender disparity, it is plausible that hormonal factors did not account for a significant effect on the current differences in pain perception found between groups. Finally, the results of this study cannot provide information to understand whether increased somatosensory amplification and anxiety are causative factors for increased pain sensitivity, or for the reverse causal pathway. In conclusion, the findings of our study have revealed that individuals with joint effects of high somatosensory amplification and trait anxiety report greater occlusal discomfort and pain than do individuals with low somatosensory amplification and trait anxiety. As a consequence, orthodontic practitioners should be aware of the psychological characteristics of their patients, and possibly should recognize those individuals who may be at risk for complications during irreversible protracted dental and orthodontic treatments.

Conflicts of interest – The authors declare no conflict of interest.

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