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# Relationship of active trigger points with related disability and anxiety in people with tension-type headache

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**Abstract**

To investigate the differences in the presence of trigger points (TrPs) and their association with headache-related disability and mood disorders in people with frequent episodic tension-type headache (TTH) (FETTH) and chronic TTH (CTTH). One hundred twenty-two individuals with TTH participated. Clinical features of headache (i.e., intensity, duration, and frequency) were recorded on a headache diary. Headache-related disability was assessed with the Headache Disability Inventory, trait and state anxiety levels with State-Trait Anxiety Inventory, and depression with the Hospital Anxiety and Depression Scale. TrPs were bilaterally explored in the temporalis, masseter, suboccipital, upper trapezius, splenius capitis, and sternocleidomastoid muscles. Sixty-two (51%) patients were classified as FETTH, whereas 60 (49%) were classified as CTTH. Individuals with CTTH showed higher burden of headache and depression than FETTH ($P < 0.001$). Subjects with FETTH showed similar number of TrPs (total number: $5.9 \pm 3.1$, active TrPs: $4.7 \pm 2.5$, and latent TrPs: $1.2 \pm 1.9$) than those with CTTH (total number: $5.7 \pm 3.2$, active TrPs: $4.2 \pm 3.0$, and latent TrPs: $1.5 \pm 1.8$). The number of active TrPs was significantly associated with the burden of headache ($r = 0.189$; $P = 0.037$) and trait anxiety ($r = 0.273$; $P = 0.005$): the higher the number of active TrPs, the greater the physical burden of headache or the more the trait anxiety level. No association with the depression was observed. The presence of active TrPs in head and neck/shoulder muscles was similar between individuals with FETTH and CTTH and associated with the physical burden of headache and trait anxiety levels independently of the subgroup of TTH.

**Abbreviations:** CTTH = chronic tension-type headache, ETTH = episodic tension-type headache, FETTH = frequent episodic tension-type headache, HADS = Hospital Anxiety and Depression Scale, HDI = Headache Disability Inventory, ICHD-III = International Classification of Headache Disorders, third edition, NPRS = Numerical Pain Rate Scale, STAI = State-Trait Anxiety Inventory, TrP = trigger point, TTH = tension-type headache.

**Keywords:** anxiety, burden, depression, tension-type headache, trigger points

## 1. Introduction

Tension-type headache (TTH) is a common headache disorder and is an important condition in terms of socioeconomic impact.[1] The mean global prevalence of TTH in an adult population is around 42%.[2] The general costs in Europe in 2010 were €13.8 billion for headache, including migraine and TTH.[3] Current research into the pathogenesis of TTH focuses on the role of muscle and facilitation of nociceptive pain processing.[4] In fact, it is becoming clear that TTH has a muscle component, and peripheral and central sensitization mechanisms may play a role on its development and chronification.[5]

Some studies have previously reported the association between TTH and impairments in the musculoskeletal system.[6] Among these impairments, myofascial trigger points (TrPs) have received particular attention in the headache literature. There is evidence supporting a potential association between TTH and TrPs[7]; although treatment studies on inhibiting the TrPs are not conclusive.[4] TrPs are defines as "hypersensitive spots in taut bands of skeletal muscles which elicit referred pain, autonomic, and motor symptoms when stimulated."[8] TrPs are considered clinically active when the elicited referred pain reproduces symptoms similar to those the patients suffer from.[8] Previous studies have reported that the referred pain elicited by active TrPs in head, neck, and shoulder musculature mimics the headache pain pattern in individuals with chronic TTH (CTTH)[9–11] and episodic TTH (ETTH)[12,13]; nevertheless, the number of active TrPs seems to be lower in the episodic form. This hypothesis was partly supported by the only study investigating directly the differences in the presence of active TrPs between subjects with CTTH and ETTH.[14] Nevertheless, it should be recognized that the sample sizes of these studies were small and did not differentiate between frequent or infrequent episodic TTH. New studies including larger sample sizes and using the most updated diagnostic criteria of TTH are needed.

In addition, it seems clear that individuals with TTH usually suffer from mood disorders, particularly anxiety and depres-

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sion.[15] In fact, emotional stress is one of the most common trigger and aggravating factor of TTH episodes.[16] It is purported that active TrPs can precipitate or promote TTH by transmitting nociceptive input to the trigeminocervical nucleus caudalis.[17] Similarly, stress can also trigger TTH through hyperalgesic effects by reducing the thresholds to noxious input from pericranial muscles.[18] Therefore, it is possible that comorbid anxiety and depression frequently observed in people with TTH are related to the presence of active TrPs. No previous study has investigated the relationship between mood disorders and TrPs in primary headaches. Similarly, the relationship between active TrPs and headache-related disability has not been yet investigated in TTH. It would be important to determine if the presence of TrPs is associated with headache-related disability.

Therefore, the objectives of the present study were to investigate the differences in the presence of TrPs between individuals with frequent episodic TTH (FETTH) and CTTH using the most updated diagnostic criteria, to determine the association between headache-related disability and active TrPs in individuals with TTH, and to determine the association between mood disorders (anxiety and depression) and active TrPs in TTH. Our hypothesis was that people with CTTH would exhibit greater number of active TrPs, headache-related disability, and mood disorders than those with FETTH and that active TrPs would be associated with higher headache-related disability and higher mood disorders.

## 2. Methods

### 2.1. Participants

Subjects with TTH were recruited from different university-based hospitals between January 2015 and January 2016. All diagnoses were made according to the criteria of the International Classification of Headache Disorders, third edition (ICHD-III beta, 2013) down to third-digit level (codes 2.2 and 2.3) by neurologist experts in headache field.[19] They performed a face-to-face interview followed by a general and neurological examination. To be included, patients had to describe all the pain features of TTH: bilateral location, pressing and tightening pain, moderate intensity ($\leq$6 on a 10-point Numerical Pain Rate Scale, NPRS), and no aggravation of pain during physical activity. Patients should report neither more than one of photophobia, phonophobia, or mild nausea and neither moderate nor severe nausea nor vomiting as requested by the ICHD-III diagnostic criteria.[19]

Key elements of the clinical history, including headache-family history, headache features, temporal pattern, and intake medication, were assessed. A headache diary for 4 weeks was used to substantiate the diagnosis and to record the headache clinical features.[20,21] On this diary, patients registered the number of days with headache (d/wk), the duration of each headache attack (h/d), and the headache intensity on an 11-point NPRS[22] (NPRS; 0: no pain, 10: maximum pain).

Participants were excluded if presented other primary/secondary headache; medication overuse headache as defined by the ICHD-III; history of cervical or head trauma (i.e., whiplash); pregnancy; history of cervical herniated disk or cervical osteoarthritis on medical records; any systemic degenerative disease, for example, rheumatoid arthritis and lupus erythematous; comorbid diagnosis of fibromyalgia; receiving anesthetic block in the previous 6 months; receiving previous physical treatment in the neck/head the previous 6 months; or abuse of caffeine or other stimulating substances. A careful clinical examination of each patient was conducted to determine inclusion and exclusion criteria.

All participants read and signed a consent form before their participation. The study was approved by local Ethics Committees (URJC 23/2014 [Spain], N20140063 [Denmark], and CESU 5/2015 [Italy]).

### 2.2. Trigger point examination

Participants were evaluated pain-free or, in those with high frequency of headache, when the intensity of pain was $\leq$3 points. TrPs were explored bilaterally in the temporalis, masseter, suboccipital, sternocleidomastoid, upper trapezius, and splenius capitis musculature by a clinician with more than 10 years of experience in TrP diagnosis and blinded to TTH diagnosis (FETTH or CTTH). The order of evaluation was randomized between individuals with a 1-minute rest period between muscles. TrPs diagnosis was performed, in general, according to the following criteria: presence of a palpable taut band in the muscle, presence of a painful tender spot in the taut band, local twitch response on palpation of the taut band, and reproduction of referred pain.[8] TrP diagnosis was conducted using snapping palpation (first to locate the taut band, and then moving the thumb tip back and forth to roll the underlying muscle fibers) to induce a local twitch response and flat palpation (placing the padded aspect of the thumb on the spot and applying pressure against the underlying tissue or bone) to induce the referred pain. For the suboccipital muscles, the protocol described by Fernández-de-las-Peñas et al[9] was used. TrP diagnosis in these muscles was conducted when a pressure applied on the suboccipital region for 10 seconds elicited referred pain and contraction of the muscles in the head extension increased the referred pain.[9] A TrP was considered active when the referred pain elicited during examination reproduced at least part of the TTH pain pattern that the subject usually suffered from, and, therefore, the pain was recognized as a usual and/or familiar pain. A TrP was considered latent when the pain elicited during examination did not reproduce any TTH pain feature, and, therefore, the elicited pain was not recognized as a usual/familiar pain symptom.[8]

### 2.3. Headache Disability Inventory

The Headache Disability Inventory (HDI) was designed to assess the burden of headache using 25 items that inquire about the perceived impact of headache on emotional functioning (e.g., "Because of my headaches I feel handicapped") and daily activities (e.g., "Because of my headaches I feel restricted in performing my routine daily activities").[23] Possible answers for each item are YES (4 points), SOMETIMES (2 points), and NO (0 point). Thirteen items asses the emotional component of headache (HDI-E, maximum score: 52), and the remaining 12 items assess the physical component (HDI-P, maximum score: 48). A greater score suggests a greater burden/disability of headache. The HDI has exhibited good stability at short ($r$=0.93–0.05) and long term ($r$=0.76–0.83).[24]

### 2.4. State-Trait Anxiety Inventory

The State-Trait Anxiety Inventory (STAI) is a 40-item scale assessing separate dimensions of state anxiety (items l–20, STAI-S) as well as trait anxiety (items 21–40, STAI-T).[25,26]

The STAI-S items assess relatively enduring symptoms of anxiety. Sample items include "I am worried" and "I am jittery." Participants use a 4-point response scale ranging from "not at all" to "very much," to indicate the extent to which they experience each emotion. The STAI-S scale had exhibited good internal consistency (α ranging from 0.83 to 0.92). The STAI-T scale measures a stable propensity to experience anxiety and tendencies to perceive stressful situations as threatening. It consists of 20 statements requiring individuals to rate how they generally feel on a 4-point scale. The STAI scales have shown a mean internal consistency score of 0.89 and mean test–retest reliability of 0.88.[27] In both scales, higher scores indicate greater state or trait anxiety.

### 2.5. Hospital Anxiety and Depression Scale

The Hospital Anxiety and Depression Scale (HADS) is a 14-item self-report screening scale, 7 items for anxiety (HADS-A) and 7 for depression (HADS-D), developed to indicate the presence of anxiety and depression states.[28] Each item scores on a 4-point Likert scale (0–3) giving a maximum subscale score of 21 for each scale.[29] The HADS has shown good validity and reliability with values ranging from 0.60 to 0.80.[30] In patients with TTH, the HADS has also shown good internal consistency (Cronbach α: 0.84).[31]

### 2.6. Statistical analysis

Data were analyzed with the SPSS statistical package (22.0 Version, SPSS Inc, Chicago, IL, USA). Descriptive data were collected on all patients. The Kolmogorov–Smirnov test revealed that quantitative data exhibited a normal distribution ($P > 0.05$). Differences in clinical data (i.e., frequency, intensity, or duration), depression, anxiety, disability, and the number of TrPs (active or latent TrP) between patients with FETTH or CTTH were assessed with the unpaired Student $t$ test. The chi-square ($\chi^2$) test was used to analyze the differences in the distribution of TrPs (active or latent) for each muscle within both groups. The Pearson correlation test ($r$) was used to determine the association between the number of active TrPs, depression (HADS-D), anxiety (HADS-A, STAI-S, and STAI-T), burden (HDI-P and HDI-E), and clinical variables relating to headache pain (frequency, intensity, and duration). Correlations were considered weak when $r < 0.3$, moderate when $0.3 < r < 0.7$, and strong when $r > 0.7$.[32] The statistical analysis was conducted at a 95% confidence level, and a $P$ value less than 0.05 was considered statistically significant.

## 3. Results

### 3.1. Clinical data of the sample

From 160 eligible subjects with headache who accepted to participated, 38 were excluded for the following reasons: comorbid migraine (n = 22), reporting previous neck trauma (n = 8), and fibromyalgia diagnosis (n = 8). A total of 122 individuals with TTH were finally included. Sixty-two (51%) were classified as FETTH, whereas the remaining 60 (49%) were classified as CTTH accordingly to the ICHD-III. Fifty-five (45%) patients were taking prophylactic drugs (i.e., amitriptyline) on a regular basis. No changes were conducted on preventive medication intake. Individuals with CTTH exhibited higher frequency and longer duration, but similar intensity, of headache attacks than those with FETTH (all, $P < 0.05$). Further, CTTH also showed higher burden of headache and depression than FETTH (all $P < 0.001$). No differences in anxiety levels were observed ($P > 0.351$). Table 1 shows the clinical data of each group of people with TTH.

The HDI-P showed moderate-to-weak positive associations with the intensity ($r = 0.345$; $P < 0.001$) and the frequency ($r = 0.293$; $P < 0.001$) of headache: the higher the intensity or the higher the frequency of TTH attacks, the higher the physical burden of headache.

### 3.2. Trigger points in tension-type headache

The mean ± SD number of TrPs in the selected and examined muscles for each patient with FETTH was 5.9 ± 3.1 of which 4.7 ± 2.5 were active TrPs and the remaining 1.2 ± 1.9 latent TrPs. Similarly, each subject with CTTH showed a total number of 5.7 ± 3.2 (active: 4.2 ± 3.0, latent: 1.5 ± 1.8) TrPs. No significant differences existed in the total number (t = 1.294; $P = 0.198$),

**Table 1**

**Clinical and demographic characteristics of patients with frequent episodic and CTTH.**

| | Frequent episodic tension-type headache (n = 62) | | Chronic tension-type headache (n = 60) | |
|---|---|---|---|---|
| | Mean | 95% CI | Mean | 95% CI |
| Clinical data | | | | |
| Gender (male/female) | 18/46 | | 17/43 | |
| Age, y | 44 | 40–48 | 46 | 42–50 |
| Headache pain characteristics | | | | |
| Time of onset, y | 10.3 | 7.3–10.3 | 8.4 | 5.5–11.3 |
| Frequency, d/mo* | 9.8 | 8.8–10.9 | 26.5 | 25.8–27.3 |
| Intensity (NPRS, 0–10) | 5.8 | 5.3–6.3 | 5.9 | 5.5–6.4 |
| Duration, h/d† | 6.6 | 5.6–7.7 | 8.5 | 7.4–9.6 |
| Psychological outcomes | | | | |
| HDI-E (emotional, 0–52)* | 14.3 | 11.8–16.9 | 25.7 | 22.0–29.4 |
| HDI-P (physical, 0–48)* | 19.1 | 16.4–21.7 | 26.9 | 23.5–30.4 |
| HADS-D (depression, 0–21)* | 7.4 | 6.3–8.3 | 9.9 | 8.6–11.1 |
| HADS-A (anxiety, 0–21) | 11.0 | 9.9–12.2 | 10.3 | 9.0–11.5 |
| STAI-S (state, 0–60) | 21.5 | 20.3–22.7 | 21.7 | 20.4–23.1 |
| STAI-T (trait, 0–60) | 23.8 | 22.2–25.4 | 23.5 | 21.7–25.3 |

CI = confidence interval, HADS = Hospital Anxiety and Depression Scale, HDI = Headache Disability Inventory, STAI = State-Trait Anxiety Inventory.

* Significant differences between patients with frequent episodic and chronic tension-type headache ($P < 0.001$).

† Significant differences between patients with frequent episodic and chronic tension-type headache ($P < 0.05$).

**Table 2**

**Individuals with frequent episodic and CTTH with TrPs.**

| | Temporalis FETTH (n = 62) | | Masseter FETTH (n = 62) | | Sternocleidomastoid FETTH (n = 62) | |
|---|---|---|---|---|---|---|
| | Right side | Left side | Right side | Left side | Right side | Left side |
| Active TrPs (n, %) | 45 (72.5%) | 41 (66%) | 14 (23%) | 11 (18%) | 23 (36.5%) | 19 (30.5%) |
| Latent TrPs (n, %) | 7 (11%) | 7 (11%) | 12 (19%) | 14 (23%) | 13 (22%) | 16 (26%) |
| No TrPs (n, %) | 10 (16.5%) | 14 (23%) | 36 (58%) | 37 (59%) | 26 (41.5%) | 27 (43.5%) |
| | Temporalis CTTH (n = 60) | | Masseter CTTH (n = 60) | | Sternocleidomastoid CTTH (n = 60) | |
| | Right side | Left side | Right side | Left side | Right side | Left side |
| Active TrPs (n, %) | 38 (63%) | 38 (63%) | 10 (17%) | 13 (22%) | 17 (28%) | 15 (25%) |
| Latent TrPs (n, %) | 9 (15%) | 7 (12%) | 12 (20%) | 7 (12%) | 11 (18.5%) | 11 (18.5%) |
| No TrPs (n, %) | 13 (22%) | 15 (25%) | 38 (63%) | 40 (66%) | 32 (53.5%) | 34 (56.5%) |
| | Upper trapezius FETTH (n = 62) | | Splenius capitis FETTH (n = 62) | | Suboccipitalis FETTH (n = 62) | |
| | Right side | Left side | Right side | Left side | Bilateral | |
| Active TrPs (n, %) | 30 (48%) | 25 (40%) | 31 (50%) | 33 (53%) | 14 (22.5%) | |
| Latent TrPs (n, %) | 9 (15.5%) | 5 (8%) | 4 (6.5%) | 1 (1.5%) | 12 (19%) | |
| No TrPs (n, %) | 23 (36.5%) | 32 (52%) | 27 (43.5%) | 28 (45.5%) | 36 (58.5%) | |
| | Upper trapezius CTTH (n = 60) | | Splenius capitis CTTH (n = 60) | | Suboccipitalis CTTH (n = 60) | |
| | Right side | Left side | Right side | Left side | Bilateral | |
| Active TrPs (n, %) | 26 (43%) | 20 (33%) | 24 (40%) | 27 (45%) | 14 (23.5%) | |
| Latent TrPs (n, %) | 7 (12%) | 5 (8.5%) | 5 (8.5%) | 4 (6.5%) | 8 (13.5%) | |
| No TrPs (n, %) | 28 (45%) | 35 (58.5%) | 32 (51.5%) | 29 (48.5%) | 38 (63%) | |

CTTH = chronic tension-type headache, FETTH = frequent episodic tension-type headache, TrPs = trigger points.

active (t = 1.010; $P = 0.311$), or latent (t = 0.662; $P = 0.509$) TrPs between individual with FETTH or CTTH. The distribution of TrPs in the analyzed muscles was not significantly different for any muscle between patients with FETTH and CTTH (all, $P > 0.414$, Table 2). Active TrPs within the temporalis, splenius capitis, and upper trapezius muscles were the most prevalent in either individuals with FETTH or CTTH.

No associations were observed between the number of active TrPs and any clinical pain feature, that is, intensity, frequency, or duration of the headache (all, $P > 447$). The number of active TrPs was significantly and positively associated with burden of headache (HDI-P: $r = 0.189$; $P = 0.037$; Fig. 1A) and trait anxiety (STAI-T: $r = 0.273$; $P = 0.005$; Fig. 1B): the higher the number of active TrPs, the greater the physical burden of headache, or the more trait anxiety level independently of the TTH group. No other association was observed (Table 3).

## 4. Discussion

The present study found that the presence of active TrPs in the selected head and neck/shoulder muscles was similar between individuals with FETTH and CTTH. Patients with CTTH exhibited higher burden of headache and depression than those with FETTH. The number of active TrPs was associated with the physical burden of headache and trait anxiety levels.

The fact that individuals with CTTH exhibited higher burden of headache than those with FETTH agrees with a recent study showing that headache impact was higher in individuals with higher headache frequency of headaches.[33] In fact, we also observed a moderate association between the physical burden of headache and the frequency of TTH attacks. Similarly, CTTH was also associated with higher depressive symptoms than FETTH. It seems that 70% of subjects with headache experience, at sometime, depressive episodes[15] being more pronounced in those patients with higher frequency of headaches.[34,35] An unexpected finding of our study was that CTTH and FETTH individuals exhibited similar levels of anxiety. It is possible that this lack of difference is related to the high frequency of attacks that exhibited our group of FETTH patients.

### 4.1. Trigger points in tension-type headache

The fact that the referred pain elicited by active TrPs reproduces TTH pain is not new since previous studies also observed the presence of active TrPs in this population.[9–14] Nevertheless,

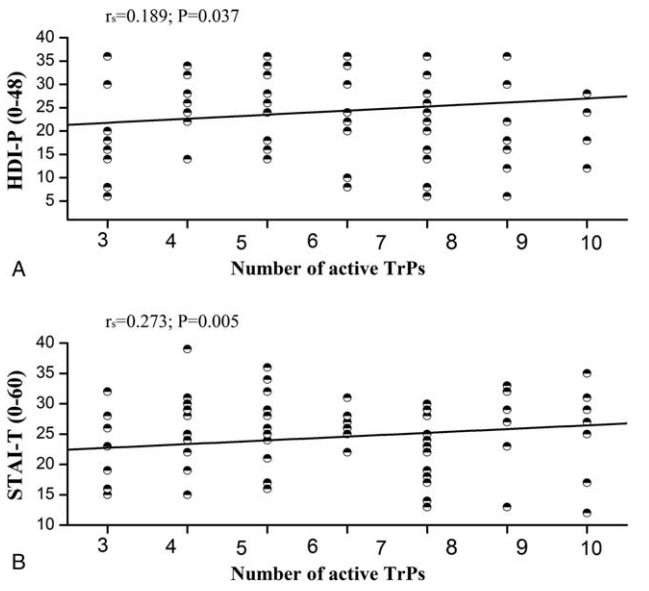


**Figure 1.** Scatter plots of relationships between the number of active trigger points with physical burden of headache (Headache Disability Inventory-P) (A) and trait anxiety levels (State-Trait Anxiety Inventory-T) (B) (n = 122). Note that several points are overlapping. A positive linear regression line is fitted to the data.

**Table 3**

**Pearson correlation coefficients between the number of active or latent TrPs and the remaining variables.**

| | HDI-P | HDI-E | STAI-T | STAI-S | HADS-A | HADS-D |
|---|---|---|---|---|---|---|
| Active TrPs | $r=0.189$; $P=0.037$* | $r=0.075$; $P=0.412$ | $r=0.273$; $P=0.005$* | $r=0.021$; $P=0.830$ | $r=0.006$ $P=0.950$ | $r=0.084$; $P=0.357$ |
| Latent TrPs | $r=0.008$; $P=0.928$ | $r=0.010$; $P=0.911$ | $r=0.114$; $P=0.250$ | $r=0.002$; $P=0.987$ | $r=0.043$; $P=0.685$ | $r=0.111$; $P=0.225$ |

| | Age | Time of onset | Intensity | Frequency | Duration |
|---|---|---|---|---|---|
| Active TrPs | $r=0.093$; $P=0.385$ | $r=0.044$; $P=0.635$ | $r=0.069$; $P=0.447$ | $r=0.055$; $P=0.550$ | $r=0.006$; $P=0.946$ |
| Latent TrPs | $r=0.097$; $P=0.387$ | $r=0.011$; $P=0.903$ | $r=0.124$; $P=0.234$ | $r=0.062$; $P=0.496$ | $r=0.001$; $P=0.994$ |

HDI = Headache Disability Inventory, STAI = State-Trait Anxiety Inventory, TrP = trigger point.
* Statistically significant ($P<0.05$).

previous studies included small sample sizes, and only 1 compared directly subjects with ETTH and CTTH.[14] Our study is the first 1 investigating differences between FETTH and CTTH using the updated diagnostic criteria (ICHD-III) and including a considerable sample of participant. We did not find differences in either the number or distribution of active TrPs between subjects with FETTH or CTTH, in contrast which was observed in a previous study.[14] A possible explanation for discrepancy between our results and those reported by Sohn et al[14] is that individuals with FETTH included in their study exhibited lower frequency of headaches than our sample of FETTH. It would be interesting to determine the presence of active TrPs in subgroups of individuals with FETTH suffering from different frequencies of headache.

The mechanism associating active TrPs with TTH has been postulated by Fernández-de-las-Peñas et al[17] in an updated pain model. In this model, sustained local contraction observed in the TrP can promote hypoxia and ischemia, elevating the concentrations of algogenic substances and chemical mediators, for example, calcitonin gene-related peptide, substance P, bradykinin and serotonin, and consequently, and may lead to a peripheral sensitization.[17] In fact, a microdialysis study supports the hypothesis that active TrPs are associated with higher levels of these and other chemical mediators near the TrP area.[36] Therefore, when the craniocervical musculature has active TrP, the nociceptive input driving to the trigeminocervical nucleus caudalis may contribute to sensitization mechanisms observed in TTH.[17] The present study supports that active TrPs are present in FETTH in a similar manner as that in CTTH supporting that active TrPs may contribute to central sensitization observed also in the episodic form.[37] Current and previous evidence would support that muscular factors may be responsible not only for the acute headache episode but also for chronification of the pain.[38] In fact, the potential relevance of active TrPs in TTH is supported by the association of the number of active TrPs with the physical burden of headache. Nevertheless, this association was weak ($r<0.2$), and future clinical studies should investigate its clinical relevance. This can be related to the fact that studies have not been conclusive in terms of how blocking the nociception from the TrPs affects the headache pain.[39]

### 4.2. Trigger points and anxiety

This is the first study investigating the association of active TrPs with mood disorders in TTH. An interesting finding was that the number of active TrPs showed a small association with trait anxiety. Previous studies have found that subjects with temporomandibular myofascial pain report higher levels of anxiety than healthy people[40,41] and that anxiety increases the likelihood of muscle tenderness.[42] In fact, general distress has been identified as a risk factor for acute local muscle pain and referred muscle pain using a standardized nociceptive input.[43] This finding would support the fact that subjects with higher trait anxiety levels exhibited a greater number of active TrPs. This hypothesis maybe also related to the fact that physiological muscle recovery depends on trait anxiety levels.[44] Since absence or delayed muscle relaxation is a potential mechanism for TrP activation,[45] it is possible that higher trait anxiety levels represent a risk factor for active TrPs. Finally, we do not currently know the effect of TrP management in mood disorders; future studies should investigate these hypotheses.

### 4.3. Limitations

Although strengths of the present study include a large sample size and the inclusion of both FETTH and CTTH patients according to the most updated diagnostic criteria, we should recognize some potential limitations. First, we did not conduct a sample size power analysis; so it is possible that some analyses were underpowered. However, since we included the biggest sample of patients with TTH investigating the role of TrPs in this population, we believe that the inclusion of more individuals would not alter the direction of our findings. Second, patients were recruited from tertiary care hospitals; therefore, it is possible that they represent a specific subgroup of the general population with TTH. Third, the cross-sectional nature of the study does not permit to establish a cause and effect relationship between active TrPs and physical burden of headache and trait anxiety levels in this population. Finally, the reliability of TrP diagnostic criteria is questioned since some systematic reviews concluded that the reliability varied widely depending, not only on the diagnostic criteria or on the muscle evaluated, but also across various studies.[46,47] However, it is important to consider that previous reviews did not include any study specifically reporting the reliability of TrP identification, only the reliability of each diagnostic criteria in isolation.[46,47] In fact, recent studies have reported moderate-to-excellent reliability for TrP diagnosis based on the presence of an experienced assessor[48,49] and the combination of the diagnostic criteria used in the present study.[50,51]

## 5. Conclusion

The presence of active TrPs in selected head and neck/shoulder muscles was similar between people with FETTH or CTTH. Patients with CTTH exhibited higher burden of headache and depression than those with FETTH. The number of active TrPs was positively associated with the physical burden of headache and trait anxiety in both individuals with CTTH and FETTH.

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