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Original article

Sensory characteristics of tender points in the lower back

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ABSTRACT

Palpation of tender points in superficial tissue is commonly undertaken in the management of musculoskeletal pain. The sensory characteristics of digitally tender points (DTPs) have not been defined. This study had two major aims: 1) to characterise 'Strain–Counterstrain' DTPs, using quantitative sensory testing (QST) in participants with low back pain (LBP); 2) to compare corresponding points at lumbar sites in participants with LBP to those without LBP. Fifteen participants with LBP (9 females), mean (SD) Oswestry scores 20.8 (10.1)) and 15 participants without LBP (6 females) were included. QST was undertaken by a single examiner blind to the location of DTPs and included measurement of electrical detection and electrical pain threshold, thermal (hot/cold) detection and thermal pain threshold, vibration detection threshold and pressure-pain threshold. In participants with LBP, DTPs demonstrated significantly lower electrical detection and electrical pain thresholds compared to contralateral non-tender points ($p < 0.0001$). These findings may be indicative of altered central processing of Aβ afferents with terminal receptors at DTPs. Participants with LBP demonstrated elevated cold pain thresholds at lower back sites and at the peripheral shoulder site compared to participants without LBP ($p < 0.001$). This may also indicate augmented central pain processing in participants with LBP.

1. Introduction

In assessment of musculoskeletal conditions, clinicians often identify digitally tender points (DTPs) in superficial tissue (Jones et al., 1995; Simons et al., 1999; Henriksson, 2003; McLean and Clauw, 2005) although the significance of these points for management of musculoskeletal conditions is controversial (Lewis et al., 2008). The sensory characteristics of DTPs have not been comprehensively examined, however lower electrical pain thresholds compared to contralateral control points (Vecchiet et al., 1990) and spontaneous electrical activity (Couppe et al., 2001) have been found at DTPs identified using 'myofascial pain syndrome' (MPS) procedures, although the latter findings were not confirmed in a recent, more rigorous study (Couppe et al., 2007).

Strain–Counterstrain (SCS) is a form of spinal manipulative therapy involving passive body positioning claimed to reduce tenderness at DTPs ('SCS tender points') and elicit reductions in pain and dysfunction (Kusunose, 1993; Jones et al., 1995). In the SCS paradigm (Kusunose and Wendorff, 1990; Jones et al., 1995), as for other paradigms (Simons et al., 1999; Henriksson, 2003), DTPs are identified by examination of defined anatomical sites and it has been proposed that dysfunctional joints in the spine are commonly associated with DTPs found at the adjacent spinous processes or paravertebral musculature (Kusunose, 1993). Further, it has been proposed that DTPs at anterior body sites may be associated with posterior spinal pain (Jones et al., 1995). DTPs in this paradigm are described as '…small zones of intense, tender, edematous muscle and fascial tissue about a centimeter in diameter' (Kusunose, 1993) and have been estimated to be four times more sensitive to palpatory pressure than 'normal' tissue (Jones et al., 1995) although these observations have not been confirmed in controlled studies.

Quantitative sensory testing (QST) may be useful to determine whether DTPs are in fact distinguishable from 'normal' tissue and if they are, to characterise these differences. This may illuminate possible processes underlying DTPs and facilitate investigation into the mechanisms by which treatments, might, as proposed, reduce tenderness at DTPs and effect clinical reductions in pain and dysfunction for painful musculoskeletal conditions (Jones et al., 1995; Simons et al., 1999).

The aims of this study were threefold. Primarily, to characterise DTPs using QST, in the lower back region of participants with low back pain (LBP), by comparing them with contralateral non-tender points (CNTPs). Secondly, to use QST to compare common sites, in the lower back region, for participants with and without LBP.

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Thirdly, QST was also undertaken at a peripheral site (away from the site of pain) in the deltoid muscle, to examine for the possibility of differences in central modulation of sensation between participants with and without LBP. SCS assessment procedures were used to identify DTPs and so the nomenclature of this paradigm (Kusunose and Wendorff, 1990; Jones et al., 1995) is used in the paper.

2. Methods

The study gained ethical clearance by the institutional Medical Research Ethics Committee. A controlled, within-subjects study design was used to compare QST measures taken at DTPs to CNTPs in the low back region for participants with LBP. A cross-sectional design was used to allow comparison between QST taken at the low back region and at a peripheral shoulder point in participants with and without LBP.

2.1. Participants

Fifteen participants with LBP (9 females and 6 males) and 15 (6 females and 9 males) without LBP were recruited. LBP was defined as per the International Association for the Study of Pain (Merskey and Bogduk, 1994) and participants were included regardless of whether symptoms were unilateral or bilateral, the chronicity of symptoms, presence of leg symptoms, or medications taken. Additional inclusion criteria were: between 18 and 65 years of age; able to lie prone; having two or more DTPs (SCS tender points) identified at lower back sites. Volunteers were excluded if they had a history of spinal fractures or surgery, had been diagnosed with an inflammatory disorder or with fibromyalgia syndrome or if their LBP was of traumatic onset. Participants without LBP had not required treatment for LBP in the last 12 months and otherwise met the same inclusion criteria as those with LBP but were not examined for DTPs.

Participants signed informed consent forms before they completed the 'General Health Questionnaire-28' (GHQ-28) (Goldberg, 1978). Participants with LBP illustrated their pain regions on a body chart, completed the 'Oswestry Disability Questionnaire' (Fritz and Irrgang, 2001) and provided visual analogue scores (VAS) for current pain and maximum and minimum pain experienced in the preceding 48 h.

2.2. Procedures

2.2.1. Determining test sites

DTPs were identified via palpation with either the thumb or index finger with pressure directed in the prescribed direction (Kusunose and Wendorff, 1990; Jones et al., 1995) at potential sites (Table 1) (Fig. 1). For participants with LBP, the two DTPs considered most tender by the assessor, based on verbal feedback with palpation, and which had CNTPs, were marked using a skin-marking pen. CNTPs were similarly marked. Additionally, unless already marked, test points were also marked at 'posterior lumbar 4' (PL4) and 'lower pole lumbar 5' (LPL5) sites bilaterally (Fig. 1) and also at a peripheral point located in the middle head of the deltoid muscle of the non-dominant arm. These latter 5 test sites, that is, bilateral PL4 and LPL5 sites and the peripheral deltoid site were also marked for the participants without LBP to allow comparison between participants with and without LBP.

2.2.2. QST

A single examiner, blind to the location of DTPs performed QST. The order of QST was kept constant to avoid changing the influence of one test on subsequent tests across participants. The tests were performed in the following order: electrical detection threshold, electrical pain threshold, warmth detection threshold, cold detection threshold, warmth pain threshold, cold pain threshold, vibration threshold (VT) and pressure-pain threshold (PPT). The sequence in which DTPs and CNTPs were measured was kept constant and as follows: non-dominant shoulder followed by points in the lower back from superior to inferior and left to right. Standardised instructions were used for all QST procedures and measures were repeated three times with mean scores used for analysis.

Electrical detection thresholds and electrical pain thresholds were measured with a Neurometer CPT/C device (Neurotron., Baltimore, USA) using the ascending method of limits. Current with a sinusoidal frequency of 250 Hz was delivered to the skin through a pair of 1 cm diameter gold electrodes coated with a thin layer of conductive gel and held firmly to the test point. The current was increased from zero at a rate of 1 mA/s up to 10 mA and thereafter at 10 mA/s. The participant was instructed to indicate when they first detected a 'sensation'.

Similarly, to measure electrical pain thresholds, the current was increased as above and the participant was instructed to indicate when the 'sensation' became one of 'discomfort'.

Thermal (hot/cold) detection and pain thresholds were measured with a Thermotest (Somedic AB, Sweden) using a 2 cm × 1 cm portion of the 2 cm × 8 cm thermode. This was

Table 1
Anatomical locations of potential digital tender point (DTP) sites assessed; labelled according to Strain–Counterstrain (SCS) nomenclature (Kusunose and Wendorff, 1990).

	Anatomical location
PL1–PL4	Lateral to respective spinous process or in immediate adjacent paraspinal musculature
PL5	Lateral to spinous process
LPL5	Between PSIS and PIIS
UPL5	Superior, medial surface of the PSIS
PL3 (iliac)	Approximately 3 cm below margin of ilium and 7 cm lateral to PSIS
PL4 (iliac)	Approximately 4 cm below margin of ilium and just posterior to the border of TFL

PSIS: posterior superior iliac spine; PIIS: posterior inferior liac spine; TFL: tensor fascia lata.

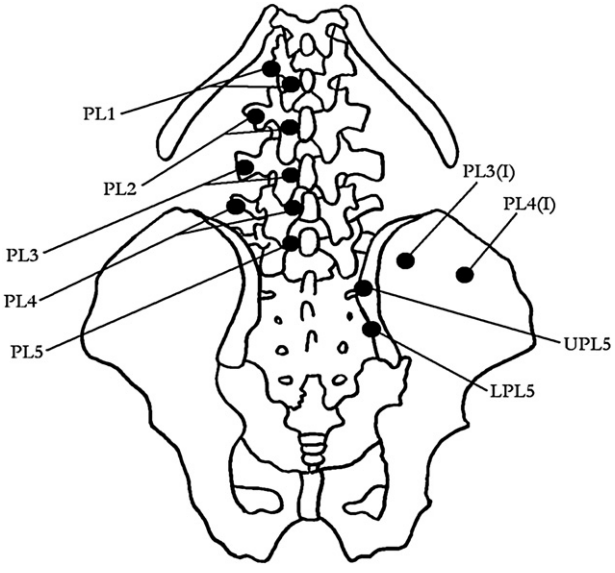

Fig. 1. Diagrammatic representation of potential digital tender point (DTP) sites labelled according to Strain–Counterstrain (SCS) nomenclature.

achieved by covering most of the thermode prior to application to the skin in order to localise skin heating and cooling at the test points. The device was preset so that temperature either increased or decreased at a rate of 1 °C/s from a baseline of 32 °C. Participants were asked to 'press the button as soon as you first detect the sensation of warmth (or cold)'.

To measure thermal (hot/cold) pain thresholds, the Thermotest was preset to either increase or decrease at a rate of 1 °C/s from a baseline of 32 °C. Participants were asked to 'press the button when the hot or cold sensation first becomes painful'. The Thermotest was set to maximum and minimum cut-out temperatures of 50 °C and 4.5 °C respectively. If, for a given trial, the hot or cold pain thresholds were not reached before the maximum or minimum cut-out temperatures, the maximum or minimum cut-out temperatures were recorded for that trial.

Vibration detection thresholds were measured with a Vibrametre (Somedic AB, Sweden), with tissue displacement range of $0.1 \pm 400\,\mu m$ and a constant frequency of 120 Hz, using the ascending and descending method of limits. Vibration was increased and decreased at 0.1 μm increments. For each test point, participants were asked to indicate when the vibration first appeared (the perception threshold: VPT) and when it disappeared (the disappearance threshold: VDT). The VT was then noted as the average of VPT and VDT.

A digital electronic pressure algometer (Somedic AB, Sweden) with a 1 cm^2 rubber footplate applied at 40 kPa/s was used to measure PPT. Participants were asked to 'push the button' (which activated the recorder marker) when the sensation of 'pressure' became one of 'pressure and pain'. A minimum of 10 s was allowed between the three repeated measures.

2.3. Analysis

Student t-tests were used to compare the means of age, weight, height and GHQ-28 scores between participants with and without LBP. Since measurements were taken at two DTPs for each participant with LBP, data was obtained from thirty DTPs (six LPL5 sites, one UPL5 site, five PL5 sites, eleven PL4I sites, six PL3I sites and one PL2 site) (Fig. 1) and their thirty CNTPs. Since there was only one participant with a DTP at UPL5, the data from this site was not included in analysis. ANOVA was used to compare QST data at DTPs and CNTPs. Planned comparisons were made to contrast DTPs and CNTPs at individual sites.

QST data, excluding that for cold pain detection, for thirty PL4 sites, thirty LPL5 sites and 15 deltoid sites for participants with LBP were compared to corresponding points in participants without LBP using ANOVA. Because cold pain detection data for both participants with and without LBP did not conform to a normal distribution, a non-parametric test (Wilcoxon) was necessary for group comparison for this measure. Statistical significance was set at $p < 0.05$ for all analyses.

3. Results

There were no significant differences for the variables of age, weight, height and GHQ-28 scores between participants with and without LBP. The mean (SD) data for GHQ-28, Oswestry Disability Index and VAS are illustrated in Table 2. Of the participants with LBP, 4 had symptoms of less than 3 months duration, 8 had symptoms of greater than 3 months duration and 2 had experienced an acute exacerbation of persistent LBP. The duration of symptoms for one subject was not ascertained. Eight participants with LBP experienced pain in the lumbar region alone, 3 in the lumbar and buttock regions, 2 in the lumbar, buttock and leg regions, one in the lumbar and groin regions and one in the lumbar, buttock, leg and groin regions. Eight participants with LBP had bilateral symptoms and seven unilateral symptoms. Two participants with LBP were taking non-opioid analgesics, one participant both a non-opioid and opioid analgesic and two participants were taking non-steroidal anti-inflammatory medications.

Table 2
Summary of descriptive data for participants with and without LBP (low back pain) (mean (standard deviation)).

Descriptive data	Participants with LBP	Participants without LBP
Age (years)	40.9 (11.3)	38.7 (12.3)
Height (cm)	169.9 (8.1)	175.5 (8.3)
Weight (kg)	74.8 (13.7)	75.9 (16.6)
Sex	6M 9F	9M 6F
GHQ	14.3 (5.1)	14 (7.5)
Oswestry	20.8 (10.1)	
VAS pain (current, minimum and maximum [preceding 48 h])	2 (1.9) 0.7 (1.1) 4.9 (2.7)	

3.1. DPTs compared to CNTPs in participants with LBP

ANOVA revealed significantly lower electrical detection threshold at DTPs than at CNTPs ($p < 0.0001$) (Fig. 2), although comparisons for individual sites indicated no significant differences at PL5 sites and the single PL2 site. For the LPL5 site, the estimate of difference in electrical detection between means of DTPs and CNTPs at the 95% confidence interval was 12.5 mA (4.71–20.3); at the PL3I site 12.4 mA (4.0–20.9) and at the PL4I site 9.7 mA (3.5–15.9) (Fig. 2).

Significantly lower electrical pain threshold at DTPs than at CNTPs ($p < 0.0001$) (Fig. 3) were revealed by ANOVA, although again, no significant difference was found for the single PL2 site. For PL3I, PL4I and LPL5 sites the estimate of difference in electrical pain threshold means between DTPs and CNTPs at the 95% confidence interval was 58.8 mA (2.4–115.2) and at the PL5 site the estimate of difference was 163.6 mA (58.8–268.4).

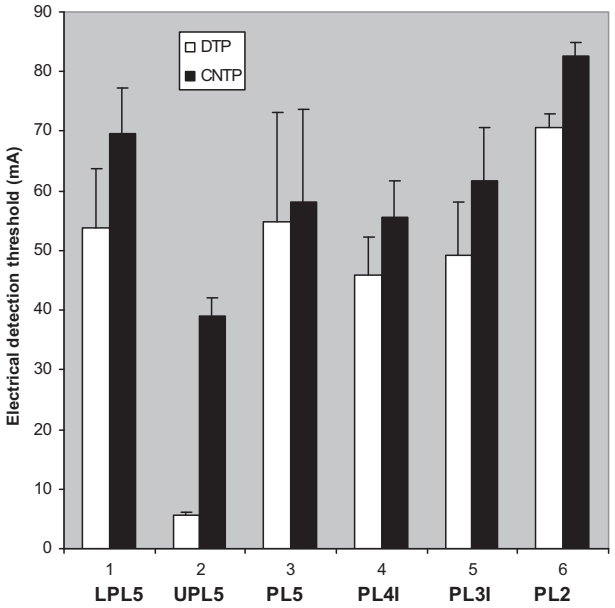

Fig. 2. Bar graphs showing electrical detection thresholds (mA) (with standard error bars) for DTPs (digitally tender points) and CNTPs (contralateral non-tender points) at lumbar Strain–Counterstrain (SCS) tender point sites (LPL5, UPL5, PL5, PL4I, PL3I, PL2) for participants with low back pain (LBP). Significant differences were found at all sites except PL5 and PL2.

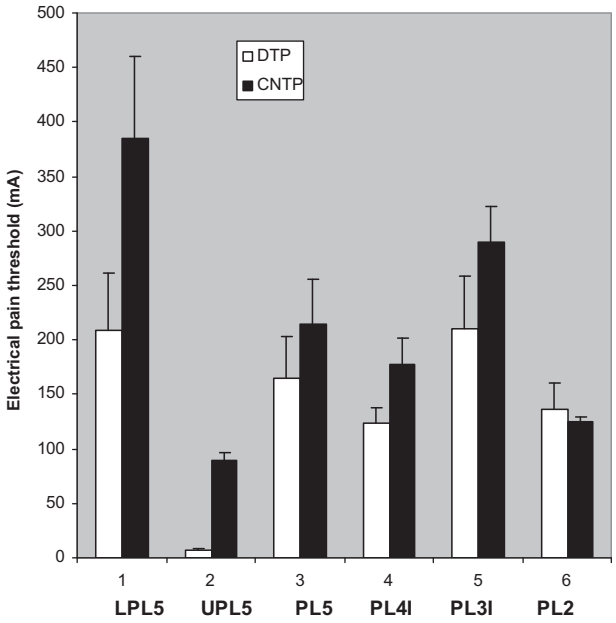

Fig. 3. Bar graphs showing electrical pain thresholds (mA) (with standard error bars) for DTPs (Digitally tender points) and CNTPs (contralateral non-tender points) at lumbar Strain–Counterstrain (SCS) tender point sites (LPL5, UPL5, PL5, PL4I, PL3I, PL2) for participants with low back pain (LBP). Significant differences were found for all SCS tender point sites except PL2.

No other differences between DTPs and CNTPs for other QST measures were revealed by ANOVA, although specific contrasts did demonstrate differences at individual sites. Heat detection thresholds were significantly lower at DTPs compared to CNTPs for the PL5 ($p < 0.05$) site and cold pain thresholds were significantly lower at DTPs at the LPL5 site ($p < 0.05$) (Table 3).

3.2. Comparison between participants with and without LBP

The Wilcoxon matched pairs test, indicated a significant difference for means of cold pain threshold between participants with LBP and those without LBP, with decreased thresholds (increased sensitivity) in participants with LBP at all common sites: bilateral LPL5 ($W = 2736$, $p = 0.0001$), bilateral PL4 ($W = 2966$, $p = 0.0009$), non-dominant shoulder ($W = 519.5$, $p = 0.0002$). The mean (95% confidence interval) values for cold pain threshold for participants with and without LBP, at shared lumbar sites, were 13.1 °C (10.8–15.4) and 7.8 °C (6.4–9.2) respectively and for the shoulder site 14.6 °C (10.2–19) and 7.6 °C (5–10.2) respectively. None of the other QST measures showed differences between participants with and without LBP (Table 4).

Table 3
Mean (95% confidence interval) for QST (quantitative sensory testing) measures at DTPs (digitally tender points) and CNTPs (contralateral non-tender points) for participants with low back pain.

QST	DTP	CNTP
Electrical detection (mA)	47.3 (32–62.6)*	58.0 (44.4–71.6)
Electrical pain (mA)	153.2 (100.4–206)*	237 (170.7–303.3)
Heat detection (°C)	37.7 (37–38.4)	38.2 (37–39.4)
Heat pain (°C)	46.7 (45.4–48)	46.8 (45.5–48.1)
Cold detection (°C)	29.4 (28.7–30.1)	29.5 (28.6–30.4)
Cold pain (°C)	11.0 (8.1–13.9)	13.0 (9.7–16.3)
Vibration detection (μm)	21.4 (14.8–28)	21.0 (14.6–27.4)
Pressure-pain threshold (kPa)	323.1 (251.4–394.8)	331 (257–405.6)

*Denotes significant difference between DTP and CNTP.

4. Discussion

The results of this study indicated that participants with LBP demonstrated reduced electrical detection (hyperaesthesia) and electrical pain threshold (hyperalgesia) at DTPs. Participants with LBP demonstrated decreased cold pain thresholds (cold hyperalgesia) compared to subjects without LBP at both the lumbar and the peripheral shoulder site.

Altered Aβ fibre activation may explain reduced electrical detection and electrical pain thresholds at DTPs compared to CNTPs. Electrical stimulation is proposed to be non-physiological and therefore to bypass receptor transducers (Arendt-Nielsen et al., 2001; Graven-Nielsen and Mense, 2001). At detection level it directly activates Aβ fibres, which have the largest diameter and the lowest threshold of afferent fibres (Collins et al., 1960; Sang et al., 2003). Possible mechanisms underlying both reduced electrical detection and electrical pain threshold at DTPs are not clear but may occur as a result of altered central processing of Aβ afferents with receptor terminals at DTPs. It has been shown that electrical threshold stimulation evoked pain rather than an innocuous tactile Aβ fibre sensation in patients with complex regional pain syndrome and mechanical allodynia (Price et al., 1989; Eliav and Gracely, 1998). Initial pain from electrical stimulation would normally be expected to be due to activation of smaller Aϑ fibres, without unmyelinated C fibre activation, at stimulation intensities greater than electrical threshold stimulation (Collins et al., 1960; Sang et al., 2003). Price et al. (1989) hypothesised that in patients with mechanical allodynia, the intraspinal circuitry which inhibits Aβ activity is deficient or absent, causing an altered response of the wide dynamic range neurons in the dorsal horn. In our study, DTPs demonstrated hyperalgesia rather than allodynia since electrical pain thresholds were higher than electrical detection thresholds. In the case of allodynia, pain thresholds and detection thresholds would be expected to occur at the same stimulus intensity. Thus, it is likely that Aϑ fibres contributed to electrical pain sensation at DTPs (Collins et al., 1960), although, it has been suggested that Aβ fibre sensitisation and hyperalgesia lie on a continuum between the extremes of normal tactile sensation and Aβ mediated allodynia due to altered central processing resulting from inflammation (Eliav and Gracely, 1998). This suggestion is supported by findings that reduced electrical detection threshold is present in the receptive fields of oral nerves inflamed by tooth extraction (Eliav and Gracely, 1998) and malignancy (Eliav et al., 2002) and reduced electrical pain threshold is present in receptive fields of oral nerves injured by mechanical trauma during tooth extraction (Eliav and Gracely, 1998). Therefore, it is possible that the results of our study indicate altered central processing due to inflammation of Aβ afferent nerves that have terminal receptors at DTPs (either adjacent to their axons or in the region of their terminal receptors), but this remains hypothetical pending further study.

We found no difference in thermal detection thresholds between DTPs and CNTPs suggesting no altered activity in either nociceptive Aϑ (cold detection) (Collins et al., 1960; Siao et al., 2004) or C fibres (heat detection) (Collins et al., 1960; Siao et al., 2004) at DTPs. Similarly, no difference in VT, primarily mediated by Aα afferents innervating joints and skeletal muscle (Vinik et al., 1995), was found between DTPs and CNTPs.

Since DTPs were identified using digital palpation, it was anticipated that they would be found to be mechanically hyperalgesic, that is, demonstrate lower PPTs. However, this was not the case and the explanation for this is unclear. Although several studies have supported algometry, particularly electronic algometry as a reliable procedure for measurement of muscle tenderness (Fisher, 1987; Vatine et al., 1993; Nussbaum and Downes, 1998), it could be argued that algometry does not adequately

Table 4
Mean (95% confidence interval) for QST (quantitative sensory testing) measures at common measurement sites (PL4, LPL5 and deltoid) for participants with and without LBP (low back pain).

QST	PL4 site		LPL5 site		Deltoid site	
	Participants with LBP	Participants without LBP	Participants with LBP	Participants without LBP	Participants with LBP	Participants without LBP
Electrical detection (mA)	52.2 (37.3–67.1)	44.1 (32.5–55.7)	49.2 (36.5–61.9)	66.5 (50.9–82.1)	47.1 (30.5–63.7)	57.2 (27.7–86.7)
Electrical pain (mA)	211.9 (137.8–286)	156.8 (101.8–211.8)	195.7 (136.5–254.9)	220.8 (141.7–299.9)	217.2 (129.5–304.9)	204.9 (133.3–276.5)
Heat detection (°C)	36.8 (36.3–37.3)	38.5 (37.3–39.7)	37.5 (37–38)	38.5 (37.5–39.5)	40 (37.8–42.2)	42.7 (40.7–44.7)
Heat pain (°C)	46.0 (44.7–47.3)	46.5 (41.1–47.9)	45.8 (43.7–47.9)	47.1 (45.9–48.3)	47.6 (46.2–49)	49 (48.3–49.7)
Cold detection (°C)	30.5 (30.1–30.9)	29.2 (28.2–30.2)	29.9 (29.3–30.5)	28.9 (27.7–30.1)	29.6 (28.7–30.5)	28.9 (27.1–30.7)
Cold pain (°C)	13.0 (9.8–16.2)*	9.3 (6.6–11)	13.2 (12.9–16.5)*	9.0 (6.4–11.6)	14.6 (10.2–19)*	7.6 (5–10.2)
Vibration detection (μm)	10.9 (6.2–15.6)	7.4 (3.5–11.3)	18.6 (12.4–24.8)	12.1 (4.1–20.3)	3.8 (2.4–5.2)	3.5 (2.2–4.8)
Pressure-pain threshold (kPa)	462.1 (371.1–553.1)	634.4 (534.5–734.3)	380.9 (299.8–462)	535.9 (441.9–629.9)	296.2 (227.4–365)	401.9 (283.7–685.6)

*Denotes significant difference between participants with and without LBP.

measure tenderness identified with digital palpation. The PPT findings may have been confounded by the QST procedures that preceded them. The sequence of QST procedures was kept constant; with PPT measures performed last to avoid an order effect masking possible differences between DTPs and CNTPs. Evidence has been provided of increased PPT measures (hypoalgesia) at referred pain areas (posterolateral neck muscles) taken 7.5 and 15 min following bilateral, but not unilateral, injections of hypertonic saline into the trapezii (Ge et al., 2003, 2006). It was suggested that hypoalgesia at referred pain areas was induced by descending inhibition triggered by spatial summation (Ge et al., 2003). It is conceivable, that in our study, the preceding bilateral measures for electrical, heat and cold pain thresholds may have induced hypoalgesia through spatial summation of muscle pain in the lower back.

Decreased cold pain threshold (cold hyperalgesia) was the only QST measure found to be different between participants with LBP and those without LBP. Our finding of no significant difference in PPT for participants with and without LBP is consistent with that of a previous study where PPTs at anatomically identified points in the low back of participants with recurrent bouts of LBP were not found to be different from those without LBP (Schenk et al., 2007). Mean (95% confidence interval) for cold pain measures, at common sites was, 13.1 °C (10.8–15.4) for participants with LBP compared to 7.8 °C (6.4–9.2) for those without LBP. It has been suggested that cold hyperalgesia at temperatures of between 15 °C and 20 °C rather than in the normal range of 0–10 °C, is a feature of neuropathic pain (Bennett, 2006; Hofbauer et al., 2006). Cold hyperalgesia has also been demonstrated to be a feature of other painful musculoskeletal conditions such as whiplash where it may represent augmented central pain processing mechanisms (Sterling et al., 2003). However we found no group difference in other QST measures and this is at odds with findings in whiplash where individuals with cold hyperalgesia also demonstrated mechanical hyperalgesia and sympathetic disturbances (Sterling et al., 2003). In other conditions, there does appear to be some relationship between the degree of sensory disturbance and pain and disability levels (Sterling et al., 2003; Chien et al., 2008). The participants with LBP in our study reported mild to moderate levels of pain and disability and this may account for the inconsistent changes in sensory sensitivity seen. Interestingly, at the remote peripheral shoulder site, subjects with LBP also demonstrated cold hyperalgesia compared to control subjects (Table 4). This may suggest that changes in cold pain thresholds occur as a result of augmented central nociceptive processing.

It should be noted that 10 of the 15 participants with LBP had experienced their symptoms for greater than 3 months. The duration of symptoms for these participants may be a factor in our finding that they displayed signs indicative of altered central processing since it has been shown that participants with chronic idiopathic LBP demonstrate signs of augmented central pain processing (Giesecke et al., 2004). Few of our participants with LBP were on any medication; therefore it is unlikely that medication usage would have influenced our results.

Considering our findings of reduced electrical detection and electrical pain thresholds at DTPs future investigations might examine the immediate effect of passive body positioning (SCS treatment) on QST measures prior to clinical studies investigating the efficacy of such an approach for LBP. Given previous findings of lowered electrical pain thresholds at DTPs identified using MPS procedures (Vecchiet et al., 1990, 1991), investigation is warranted to compare DTPs identified using the SCS and MPS paradigms.

5. Conclusion

DTPs in the SCS paradigm are characterised by reduced electrical detection and electrical pain thresholds which may indicate disturbed Aβ fibre function as a consequence of altered central pain processing. Cold hyperalgesia was found to be a feature of participants with LBP and may also indicate augmented central pain processes but no other evidence of sensory hypersensitivity was found.

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