

Original article

Tactile acuity is disrupted in osteoarthritis but is unrelated to disruptions in motor imagery performance

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Abstract

Objective. To determine whether tactile acuity is disrupted in people with knee OA and to determine whether tactile acuity, a clinical signature of primary sensory cortex representation, is related to motor imagery performance (MIP; evaluates working body schema) and pain.

Methods. Experiment 1: two-point discrimination (TPD) threshold at the knee was compared between 20 participants with painful knee OA, 20 participants with arm pain and 20 healthy controls. Experiment 2: TPD threshold, MIP (left/right judgements of body parts) and usual pain were assessed in 20 people with painful knee OA, 17 people with back pain and 38 healthy controls (20 knee TPD; 18 back TPD).

Results. People with painful knee OA had larger TPD thresholds than those with arm pain and healthy controls ($P < 0.05$). TPD and MIP were not related in people with knee OA ($P = 0.88$) but were related in people with back pain and in healthy controls ($P < 0.001$). Pain did not relate to TPD threshold or to MIP ($P > 0.15$ for all).

Conclusion. In painful knee OA, tactile acuity at the knee is decreased, implying disrupted representation of the knee in primary sensory cortex. That TPD and MIP were unrelated in knee OA, but related in back pain, suggests that the relationship between them may vary between chronic pain conditions. That pain was not related to TPD threshold nor MIP suggests against the idea that disrupted cortical representations contribute to the pain of either condition.

Key words: osteoarthritis, tactile acuity, two-point discrimination, motor imagery, left/right judgements, sensory-motor incongruence.

Introduction

OA is a degenerative, inflammatory condition that affects the joints and surrounding tissues [1], but many people with radiographic evidence of structural changes report no symptoms or disability [2]. The tenuous relationship between structural pathology and symptoms in OA

strongly suggests that other processes contribute to pain, a suggestion reflected in recent investigations of brain function in arthritis-related pain [1, 3–5].

Other chronic pain states, such as complex regional pain syndrome (CRPS) and back pain, are also associated with changes in brain function [6–10], most notably altered response profiles of cells in the primary somatosensory cortex (S1) [6, 7, 11, 12], often termed cortical reorganization. Importantly, the extent of S1 reorganization is positively related with pain intensity [9] and duration [6] and negatively with tactile acuity [9]. This relationship is preserved with successful intervention—all three variables improve together [10, 13, 14]. In fact, tactile acuity, measured by two-point discrimination (TPD) threshold [15], is considered a clinical signature of S1 representation [16]. Therefore an increase in TPD threshold (loss of tactile acuity) is considered suggestive of disruptions to S1 cortical maps of that specific body part [17].

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It is unclear whether S1 maps may be affected in people with painful knee OA and whether or not tactile acuity deficits are, in fact, specific to the location of pain. More precisely, is it knee pain, or perhaps pain in general, that is associated with tactile acuity deficits at the knee? The first aim of this study was to investigate this question by comparing knee TPD thresholds between people with painful knee OA, people with upper limb pain and people without pain (healthy controls). Because people with OA often have numerous joints affected (i.e. in the upper body as well as lower body), we recruited people with pain only in the upper limb so as to truly delineate the relationship between pain location and tactile acuity. We hypothesized that TPD threshold at the knee would be larger in people with knee OA than in both healthy controls and people with upper limb pain (i.e. tactile acuity is disrupted and specific to the location of pain).

Another unresolved issue is how S1 reorganization might contribute to pain. One proposal is that it disrupts working body schema—the cortical maps that are used to plan, coordinate and execute movement (also known as cortical proprioceptive representation)—causing discordance between motor intent and motor output [18, 19]. If this is indeed the case, one would expect a relationship to be present between tactile acuity, working body schema function and pain. Extensive research shows that working body schema can be interrogated by evaluating motor imagery performance (MIP; commonly assessed using a left/right judgement task) [20–28]. In this task, participants judge whether photographs of a body part correspond to the left or right side of the body. To make this decision, people mentally manoeuvre their own body part to match that shown in the picture [28]. Accurate left/right judgements depend on an intact working body schema [28]. Thus, we might expect a relationship between TPD threshold, left/right judgement accuracy and pain.

In addition to the discordance theory, two lines of enquiry suggest that TPD might relate to left/right judgement accuracy. First, cutaneous (i.e. tactile) inputs have been shown to contribute to proprioception [29]. Second, the working body schema is thought to depend in part on S1 data [30]. S1 disruption does not seem to impair motor performance or cause pain in healthy people [31], but it has been argued that S1 disruption might influence motor performance in a compromised system such as in chronic pain [32].

We interrogated this idea by measuring both TPD and left/right judgement accuracy in the affected body region in people with chronic pain. We included knee OA participants (knee TPD, left/right judgements of pictures of feet/lower limbs), back pain participants (back TPD, left/right judgements of pictures of the back) and also evaluated healthy controls undergoing the same task-matching. We predicted that if S1 maps disrupt motor performance, then TPD threshold would be negatively related to left/right judgement accuracy. Furthermore, if this disruption in cortical body maps contributes to pain in knee OA or

back pain, then pain intensity/duration would be positively related to TPD threshold and negatively related to left/right judgement accuracy.

Experiment 1: is tactile acuity disrupted in painful knee OA?

Methods

Full details regarding participant characteristics, recruitment and assessments are available from previously published work [4]. A brief description is given below.

Participants

Three groups of participants were recruited: (i) 20 consecutive patients with confirmed knee OA [33]; (ii) 20 age-matched patient controls with upper limb pain only and (iii) 20 healthy controls (further participant information for experiment 1 is available as supplementary data, available at *Rheumatology* Online). Written consent was obtained from all participants. This study was approved by ethics committees (Northern Sydney Central Coast Area Health Service and University of Sydney) and conformed to the Helsinki Declaration.

Questionnaires

Participants completed a questionnaire that collected demographic information (age, gender, height/weight and dominant hand/foot) and other condition-specific information (history of knee pain). Knee pain (current/average pain over last 48 h; 100-mm visual analogue scale with 0=no pain and 100=worst pain imaginable) and Oxford knee score (0–48; higher scores represent lesser disability [34]) data were also collected.

TPD threshold at the knee

Mechanical/digital callipers were used to evaluate TPD in the vertical direction on both knees using suprathreshold (but non-noxious stimuli) and following an established protocol [15]. TPD threshold was defined as the shortest distance between calliper points at which the participant could clearly detect two points instead of one. Sensory testing ensured that there were no areas of hypoaesthesia. With the participant's eyes closed, the callipers were placed on various locations of the knee (corresponding to TPD measurement locations) using the same force as used in formal testing. All participants were able to feel and distinguish these stimuli in all areas of the knee. TPD was measured 2 cm medial of the medial border of the patella and 2 cm lateral of the lateral border of the patella on each knee (using the tibiofemoral joint line as a reference point). At each location, an ascending and a descending run was completed (using 5 mm increments) with an average of these two runs used to calculate the TPD threshold. This resulted in a total of two TPD threshold measurements per knee. The side (left/right), location (medial/lateral) and sequence (ascending/descending) were randomized.

Statistics

Data were analysed using SPSS 19.0 (SPSS, Chicago, IL, USA), under the guidance of a specialist biostatistician (see Acknowledgements). Visual inspection and Shapiro–Wilk statistics ($P < 0.01$) revealed that TPD data were not normally distributed. Using visual inspection of box and whisker plots, three outliers were identified (one knee OA participant and two healthy controls) and removed from the analysis. Data were then \log_{10} -transformed. Visual inspection and Shapiro–Wilk statistic confirmed normality ($P > 0.05$ for all comparisons).

First, selecting only those with unilateral knee OA, a paired t -test was completed to determine whether differences in TPD were present between the affected and the unaffected knee. Because no differences were present (see results below), both the unaffected and the affected knee were included in the knee OA data. Second, a three (group: knee OA, patient controls and healthy controls) by four (TPD location: left knee medial, left knee lateral, right knee medial and right knee lateral) repeated measures analysis of variance (covariate age and gender) was used to determine if TPD threshold was different between groups. Least significant difference *post hoc* tests were used to determine pairwise differences. An anti-log of the \log_{10} -transformed TPD thresholds from the above analysis was undertaken to present the results as mean \pm s.d.

Results

All results are presented as mean \pm s.d. Table 1 presents participant characteristics and Table 2 presents raw data (unadjusted) for TPD thresholds. There was no difference between TPD thresholds of the affected and unaffected knee in people with painful knee OA, regardless of location of TPD measurement ($t_{1,15} = -0.36$, $P = 0.73$ for medial; $t_{1,15} = 1.674$, $P = 0.12$ for lateral).

After controlling for age and gender, TPD threshold at the knee was larger in those with painful knee OA (40.6 ± 4.7 mm) than it was in those with arm pain (33.3 ± 4.7 mm; *post hoc* test $P = 0.02$) or in pain-free controls (30.1 ± 4.6 mm; $P = 0.02$). TPD threshold was no different between those with arm pain and controls ($P = 0.41$). TPD threshold was also larger on the lateral knee locations than on the medial locations and was larger in females than in males. Specifically, there was a main effect of group ($F_{2,52} = 4.2$, $P = 0.02$; Fig. 1), age ($F_{1,52} = 7.2$, $P = 0.02$), gender ($F_{1,52} = 7.2$, $P = 0.01$; supplementary Table S1, available at *Rheumatology* Online) and a main effect of TPD location ($F_{3,159} = 3.2$, $P = 0.03$; Fig. 1 and supplementary Tables S1 and S2, available at *Rheumatology* Online). These results show that although age and gender affect TPD, group had an effect over and above that of age and gender. There was no TPD location \times group interaction ($P = 0.15$), TPD location \times gender interaction ($P = 0.58$) or TPD location \times age interaction ($P = 0.07$).

Experiment 2: is there a relationship between tactile acuity and MIP in people with painful knee OA or back pain?

Methods

Participants

Combined data from two cross-sectional studies were used.

Study A. Data from the 20 participants with painful knee OA and 20 healthy controls, as described in Experiment 1, were used for analysis. Participants with arm pain were not included due to mismatch between site of pain and TPD measurement location (i.e. TPD was measured only at the knee).

Study B. To evaluate the relationship between TPD threshold and left/right judgement accuracy in another chronic pain condition, we used data from a convenience sample of 17 participants with non-specific low back pain (LBP) who took part in a study on disruption of working body schema [20]. We also used the data from the 18 healthy pain-free controls [20] (see supplementary Table S3 for eligibility criteria, available at *Rheumatology* Online). These pain-free controls were additional to the 20 healthy controls recruited for Study A. Informed consent was obtained from all subjects. This study was approved by institutional ethics committees (University College London and National Health Services Central Ethics Committee) and conformed to the Helsinki Declaration.

Procedure

Study A. As described in Experiment 1.

Study B. Demographic and clinical data were collected in an interview and via standard full physical examination by a physiotherapist. These assessments determined the location and characteristics of participants' back pain (average pain intensity over the last 48 h; pain duration). Participants also completed a 36-item Short-Form Health Survey Questionnaire (SF-36) [35].

TPD threshold

Study A. TPD threshold was measured at the knee as described in Experiment 1.

Study B. Using a plastic calliper ruler, TPD threshold was measured horizontally on both sides of the back in the area between the first lumbar vertebra and iliac crest according to established protocol [15]. Sensory testing with monofilaments and then the callipers ensured that all experimental stimuli were suprathreshold. TPD threshold was defined identical to Study A (and Experiment 1) and it was calculated as the average of a descending run (5 mm increments from 100 mm) and an ascending run (5 mm increments from 10 mm), taking an average of the left measure and the right measure.

Left/right judgement task

Left/right judgements were performed using Recognise, a commercially available online software program

TABLE 1 Participant characteristics

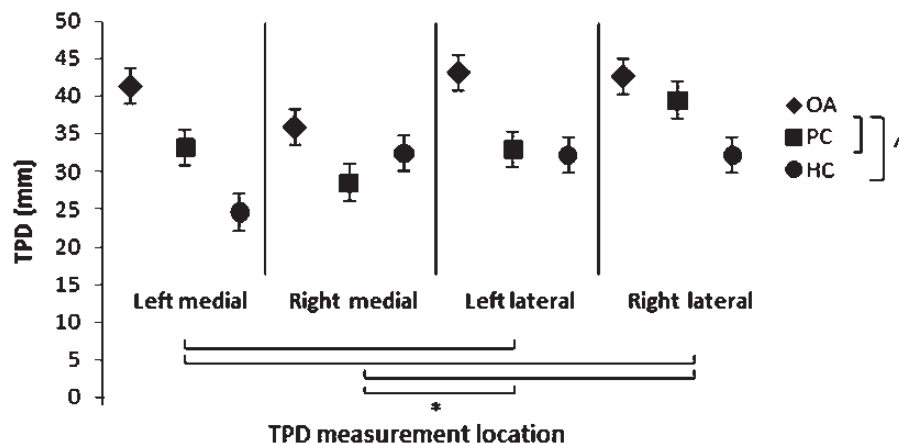
| | Knee OA (<i>n</i> = 20) | PC (<i>n</i> = 20) | HC (knee) (<i>n</i> = 20) | Back pain (<i>n</i> = 17) | HC (back) (<i>n</i> = 18) |
|-------------------------------------|--|---|----------------------------|----------------------------|----------------------------|
| Age (years) | 68 (9) | 65 (8) | 37 (16) | 45 (14) | 41 (11) |
| Number of females | 14 | 9 | 12 | 14 | 11 |
| Right hand dominant, <i>n</i> | 18 (2 missing) | 18 (1 missing) | 20 | 13 | 18 |
| Right leg dominant, <i>n</i> | 18 (2 missing) | 19 (1 missing) | 20 | Not collected | Not collected |
| BMI (kg/m ²) | 29.4 (5.0) | 26.1 (3.7) | 23.4 (3.0) | Not collected | Not collected |
| Pain location, <i>n</i> | | | | | |
| Left side | 7 | 5 | N/A | 4 | N/A |
| Right side | 9 | 10 | N/A | 7 | N/A |
| Bilateral | 4 | 2 | N/A | 4 | N/A |
| Central | N/A | N/A | N/A | 2 | N/A |
| No pain | 0 | 1 ^a | N/A | 0 | N/A |
| Missing | 0 | 2 | N/A | 0 | N/A |
| Pain intensity ^b | | | | | |
| Current pain | Knee 21.9 (25.5) | Knee 1.5 (6.5) | Knee 0.95 (2.3) | Back Not collected | Back N/A |
| Pain over last 48 h | 51.9 (21.6) | 2.2 (7.1) | 1.2 (3.0) | 3.4 (2.2) | N/A |
| History of pain (years) | Knee: affected: 8.3 (7.4); unaffected: 2.6 (16.2); bilateral: 6.0 (3.2) | Knee: N/A Arm: not collected 47 (2.1) | Knee: N/A 48 (1.4) | Back: 9.2 (11.4) | Back: N/A |
| Duration of current episode (years) | Knee: N/A | Arm: not collected | Knee: N/A | Back: 2.6 (6.0) | Back: N/A |
| Oxford knee score (0–48) | 22 (8.3) | 47 (2.1) | 48 (1.4) | N/A | N/A |
| Physical Component Score (SF-36) | N/A | N/A | N/A | 19.7 (7.4) | N/A |

All values are mean (s.d.) unless otherwise specified. *n*: number of participants; missing: missing data; N/A: not applicable. ^aReported upper limb soreness, but scored pain as 0. ^bKnee pain intensity measured on a 0–100 mm numerical rating scale, back pain intensity measured on a 0–10 visual analogue scale.

TABLE 2 TPD threshold (mm) and left/right judgement accuracy (% correct) raw data for Experiments 1 and 2

| | Knee OA | Upper limb pain | Healthy controls (knee) | Back pain | Healthy controls (back) |
|---|------------------|-------------------|-------------------------|-------------|-------------------------|
| Experiment 1 | | | | | |
| Median TPD thresholds (interquartile range) | | | | | |
| Left knee medial | 43.8 (38.1–53.8) | 33.3 (23.8–49.3) | 23.0 (19.9–33.3) | — | — |
| Left knee lateral | 52.5 (35.1–62.4) | 37.3 (24.6–49.3) | 27.5 (20.8–42.3) | — | — |
| Right knee medial | 44.3 (35.8–50.8) | 33.0 (21.25–43.3) | 25.8 (19.1–33.6) | — | — |
| Right knee lateral | 44.8 (35.9–55.8) | 38.8 (34.6–52.5) | 29.3 (24.1–37.8) | — | — |
| Experiment 2 | | | | | |
| Mean TPD thresholds (s.d.) ^a | | | | | |
| Knee (total) | 45.1 (11.8) | b | 29.4 (8.8) | — | — |
| Back | — | — | — | 59.8 (11.7) | 45.3 (5.1) |
| Mean accuracy of left/right judgements (s.d.) | | | | | |
| Feet/lower limb | 60.7 (20.1) | b | 88.5 (21.9) | — | — |
| Trunk rotation | — | — | — | 61.4 (17.6) | 80.5 (8.7) |

^aUnadjusted TPD thresholds. ^bData collected but not used, because site of pain (upper limb) did not match TPD measurement location (knee) nor body part used in left/right judgement task (feet/lower limb). —: data not collected.

FIG. 1 Knee TPD thresholds specific to group and measurement location.

Mean TPD thresholds (mm) specific to group status (knee OA, arm pain and healthy controls) and location of measurement on the knee, adjusting for age and gender. **Post hoc* differences between locations $P < 0.05$ (after a main effect of location, $P = 0.03$). ^*Post hoc* differences between groups $P < 0.05$ (after a main effect of group, $P = 0.02$). There were no significant interactions ($P = 0.07$ – 0.58).

(<http://recognise.noigroup.com/recognise>). Participants were instructed to perform the task as quickly and as accurately as possible. Accuracy (percentage correct) was the primary outcome variable for this experiment. See supplementary Fig. S1, available at *Rheumatology* Online, for representative images used in left/right judgement tasks.

Study A. This task involved performing left/right judgements for photographs of left and right feet/lower limbs in a variety of postures. Images of feet have been used previously, with judgement accuracy found to be reduced in people with knee OA (vs healthy controls) [4]. Ten

photographs of feet were randomly selected from a pool of 20 and were presented in a random order. Participants responded by pressing either the A key (left index finger; left foot images) or the D key (right index finger; right foot images) on the computer keyboard. One practice trial was completed and participants then undertook a second trial of 10 images for which data were analysed.

Study B. This task involved performing left/right judgements for photographs of the back in postures of left or right trunk rotation. The photographs were randomly selected from a pool of 56 photographs and were displayed in a random order. The index and middle finger of the

dominant hand were placed on A and D key of the computer keyboard. Participants responded by pressing A if the photograph showed the model in left trunk rotation and D if it showed the model in right trunk rotation. The left/right judgement task involved two trials of 40 images. Each trial was preceded by a practice trial of 80 images.

Statistics

Data were analysed using SPSS 19.0 (SPSS, Chicago, IL, USA), under the guidance of a specialist biostatistician (see Acknowledgements). In Experiment 1, TPD was not different between affected and unaffected knees, so an average value was taken for Study A. Visual inspection and Shapiro–Wilk statistics ($P > 0.05$ for all) revealed that TPD data were normally distributed for all groups in Studies A and B.

Linear regression related TPD threshold (dependent variable) to left/right judgement accuracy with group (knee OA, back pain and healthy controls), task (knee TPD/foot images vs back TPD/back images), gender and age also included as independent variables. Univariate regression analyses were completed. All independent variables with $P < 0.25$ were included in the multiple regression model. Effect modification analyses were completed using a backward elimination method. Any significant/influential independent variables were retained to control for possible confounding.

Finally, in people with knee OA and back pain, Spearman's correlation coefficients were used to determine the relationship between pain (intensity and duration) and TPD thresholds as well as between pain and left/right judgement accuracy.

Results

Table 1 presents participant characteristics and Table 2 presents the raw (unadjusted) TPD and accuracy data. Once controlling for group, task and accuracy \times group interactions, left/right judgement accuracy was no longer related to TPD threshold ($P = 0.30$). However, significant accuracy \times group interactions (both $P < 0.001$) suggested that a group-specific analysis was warranted (see supplementary data for the overall relationship between TPD and left/right judgement accuracy, available at *Rheumatology* Online).

Relationship between TPD threshold and left/right judgement accuracy specific to group and task

Painful knee OA. Accuracy was not related to TPD threshold ($F_{1,19} = 0.024$, $P = 0.88$; Fig. 2A), nor was age ($P = 0.83$) or gender ($P = 0.28$).

Back pain. Accuracy was negatively related to TPD threshold ($F_{2,16} = 58.9$, $P < 0.001$; Fig. 2B): increase in TPD threshold by 1 mm was associated with a decrease in accuracy of 0.6% ($\beta = -0.6$, 95% CI -0.80 , -0.43), adjusting for age. Gender was unrelated to TPD threshold ($F_{1,16} = 0.03$, $P = 0.86$), and the interaction between age \times accuracy was nonsignificant ($P = 0.44$).

Healthy controls. Accuracy was negatively related to TPD threshold ($F_{4,37} = 21.5$, $P < 0.001$; Fig. 2C): increase in TPD

threshold by 1 mm was associated with a decrease in accuracy of 0.14% ($\beta = -0.14$, 95% CI -0.27 , -0.013), adjusting for age, task and accuracy \times task interaction. Gender was unrelated to TPD threshold ($F_{1,37} = 0.62$, $P = 0.44$), and the interaction between age \times accuracy was nonsignificant ($P = 0.31$).

Relationship between pain and TPD/MIP

Pain intensity and duration were not related to TPD thresholds or left/right judgement accuracy in people with knee OA or back pain (Table 3).

Discussion

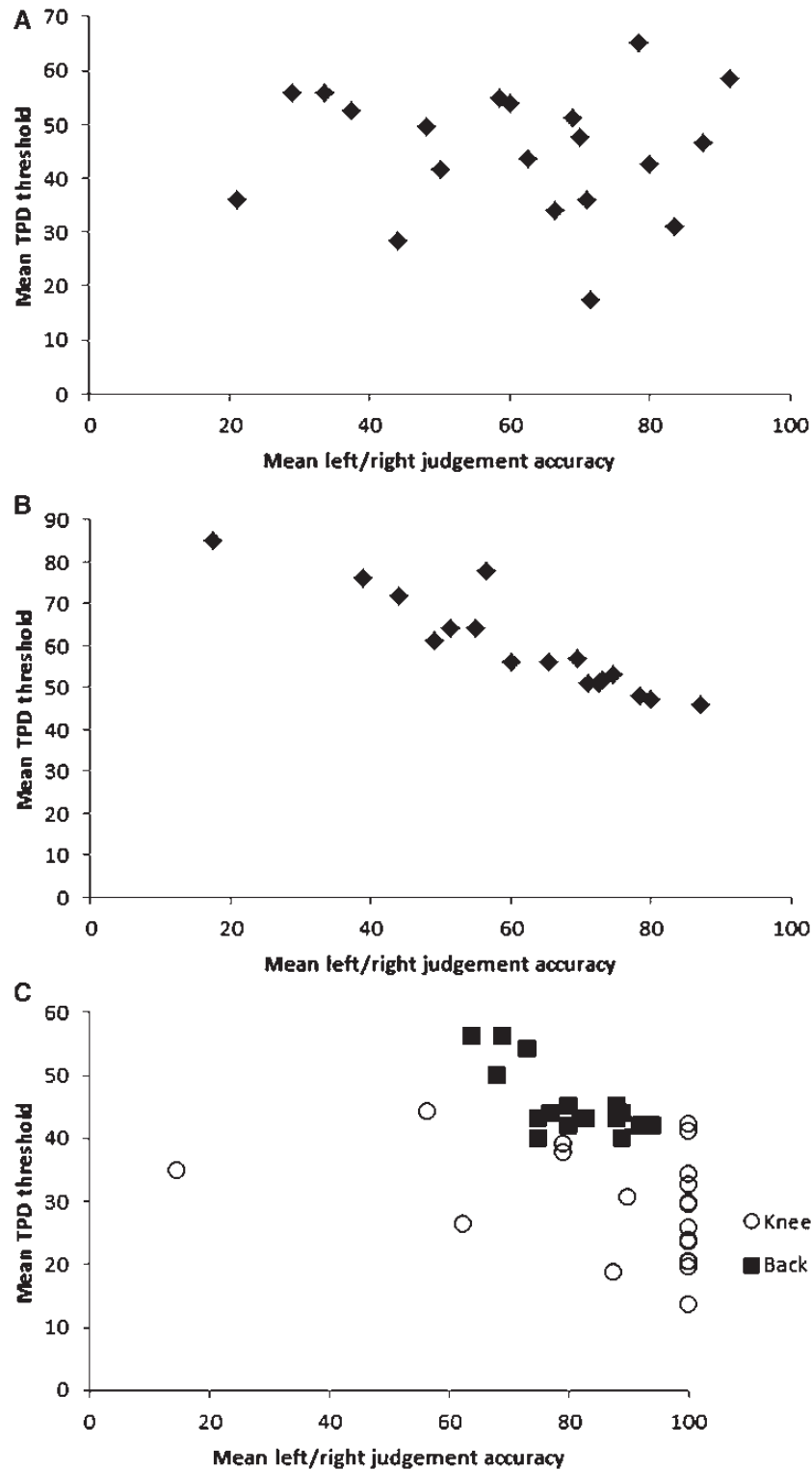
Our results support the proposal that a tactile acuity deficit is present and confirm that the location of pain is important: TPD threshold at the knee is larger in people with painful knee OA than it is in people with arm pain (and healthy controls). However, the hypothesis that, in chronic pain, tactile acuity is negatively related to left/right judgement accuracy was only partially supported—TPD threshold was related to accuracy for the back pain group and healthy controls, but not for the painful knee OA group. The direct relationship between tactile representation and working body schema in healthy controls and in people with back pain is consistent with tactile representation and working body schema colocalizing in one neural stream. However, that this relationship did not hold for people with OA suggests that tactile representation and working body schema may locate independently within the neural stream. That is, they can be independently disrupted. Finally, the hypothesis that pain intensity or duration would relate to tactile acuity and left/right judgement accuracy in chronic pain (i.e. OA or back pain) was not supported.

Differences in TPD threshold between groups

Increased knee TPD threshold in knee OA patients would be predicted on the basis of research that demonstrates a strong relationship between pain and diminished tactile acuity [9, 10, 14, 36]. Increased TPD threshold has been reported for the affected arm only in unilateral CRPS [9] and for usual area of pain in chronic back pain [25, 36, 37]. Notably however, no side-to-side difference was found in those with unilateral painful knee OA. In the present study, people with knee OA often reported a previous history of pain in the unaffected knee (Table 1). Relevant to this finding is our criterion that if the other knee was pain-free at the time of study enrolment, it was considered unaffected. Some participants reported previous knee pain and it seems reasonable that this past history of pain may explain the enlarged TPD threshold of the unaffected knee. Interestingly, bilateral impairment of proprioception also occurs in people with unilateral OA [38–40].

Decreased tactile acuity is most obviously explained by disruption within S1 or its somatosensory neuraxis [17]. Because S1 receptive fields depend on intracortical inhibition [41], it is reasonable to suggest that a loss of intracortical inhibition contributes to the effect seen here. Cortical disinhibition has been reported for a range of

Fig. 2 Relationship between TPD threshold and left/right judgement accuracy.



Relationship between mean TPD threshold (mm) and mean left/right judgement accuracy (% correct) in (A) participants with knee OA; (B) participants with back pain and (C) pain-free healthy controls.

TABLE 3 Significance values for Spearman's correlations of pain (intensity and duration) with TPD thresholds (mm) and accuracy of left/right judgements (% correct)

| Pain parameter | Knee OA | | Back pain | |
|--------------------------|---------------|----------------------------------|---------------|----------------------------------|
| | TPD threshold | Accuracy of left/right judgement | TPD threshold | Accuracy of left/right judgement |
| Current pain intensity | $P = 0.17$ | $P = 0.70$ | ^a | ^a |
| Pain intensity last 48 h | $P = 0.16$ | $P = 0.82$ | $P = 0.87$ | $P = 0.89$ |
| Pain duration | $P = 0.60$ | $P = 0.85$ | $P = 0.61$ | $P = 0.30$ |

^aCurrent pain intensity data not collected in participants with back pain.

pain problems, although, to our knowledge, it has not been directly observed in S1 in people with painful knee OA. Although people with chronic pain often have hypoaesthesia or a subtle numbness [42, 43], this is unlikely to explain the current results because we used suprathreshold stimuli throughout, and previous work shows light touch perception thresholds (assessed using von Frey filaments) to be similar between people with OA and healthy controls [44]. Perhaps disruption occurs at the level of the thalamus and this is reflected in S1 reorganization—voxel-based morphometry revealed lower thalamic grey matter volume in those with painful hip OA than in healthy controls [45]. Alternatively, perhaps the problem occurs upstream from S1, where sensory data from multiple frames of reference are integrated in order to produce the sensory percept [46]. Relevant to this possibility is the recent discovery that tactile processing is disrupted in a spatially defined way in people with CRPS [47] or chronic unilateral back pain [48]. Regardless, based on both neurophysiological data demonstrating reductions in tactile acuity following plastic changes in S1 induced by a Hebbian coactivation protocol [17] and clinical data detailing the concurrent improvement in TPD threshold and normalization of S1 reorganization with functional improvements after treatment [7–9], it remains that TPD threshold can be considered a clinical signature of S1 representation, even when S1 reorganization reflects dysfunction elsewhere in the sensory processing chain.

That TPD threshold was lower on the medial knee than on the lateral knee may be related to dermatomal patterns. TPD thresholds are lower when stimulation points overlay different dermatomes than when they are within the same dermatome [41]. The medial knee has a larger overlap of dermatomes than the lateral knee and although our measurement locations were selected to minimize this effect, we cannot rule it out. Alternatively, perhaps the medial and lateral knee vary in nerve fibre density, which is known to affect TPD threshold [41].

Relationship between TPD and MIP and pain

To our knowledge, the relationship between TPD threshold and accuracy of left/right judgements has not previously been investigated. That TPD threshold was related to accuracy of left/right judgements in both healthy controls and people with back pain, but not people with

painful knee OA, was unexpected. That tactile cues contribute to proprioception of the hand [29] raises the possibility that comparatively simple functional and tactile requirements of the knee may explain the result (i.e. a cutaneous contribution to proprioception is less likely in the knee than it is in the hand). However, this would suggest against a relationship in the back. We have previously observed a clear relationship between increased tactile acuity at the back and reduced lumbopelvic sensorimotor control [36], which is consistent with the current results, but it remains difficult to explain why the relationship does not exist in painful knee OA. One possibility might be that proprioceptive function specific to the back is lost in people with back pain [49]. This change in proprioceptive strategy might be accompanied by an impairment to MIP that parallels TPD threshold impairments. Perhaps TPD disruption in OA is not accompanied by disregarding proprioception. While proprioceptive deficits certainly exist in people with painful knee OA, it is possible that an alternative proprioceptive strategy (e.g. ankle proprioception) is not used at the expense of knee proprioception, which may uniquely maintain left/right performance.

The lack of relationship between TPD threshold and MIP in people with knee OA, but the presence of a relationship in people with back pain, may also relate to different peripheral aetiologies between the two conditions. A recent study using a rat model of OA demonstrated impairment in the function of the A-fibre mechanoreceptors throughout the entire limb, not just the affected joint [50]. These fibres are considered to play an important role in proprioception, and their impairment would clearly disrupt tactile acuity, but our results seem to suggest that their impairment does not have an isomorphic effect on working body schema. Perhaps if we evaluated proprioception in a manner that relied on real-time proprioceptive feedback from the periphery (i.e. joint reposition sense), we would see a relationship with TPD emerge. There are no data concerning an analogous widespread impairment in A-fibre mechanoreceptors in back pain, which may explain why the relationship between TPD and MIP is maintained in this group, but not in those with knee OA.

Perhaps the problem lies with our experimental approach. That is, we used images of feet, rather than knees, to interrogate motor imagery of the lower limb in painful knee OA. We did so because we know MIP of foot

images is reduced in painful knee OA [4] and because one must mentally manoeuvre the whole leg in order to mentally reposition the foot. That said, one might suggest that tactile acuity of the knee is relatively unimportant for working body schema of the whole leg. If so, that there was a relationship between accuracy of foot left/right judgements and knee TPD threshold in the healthy controls might simply reflect that, in healthy people, knee TPD reflects TPD throughout the leg. This line of reasoning is not confounded by the presence of a relationship in people with back pain, because the location of tactile acuity loss corresponds closely and uniquely with the target of motor imagery (i.e. images of the back).

Our final hypothesis that pain (intensity/duration) would relate to both TPD thresholds and left/right judgement accuracy in people with chronic pain was not supported. Although this finding does not refute the idea that S1 reorganization may cause pain via a sensory-motor mismatch [19, 51–53], it does add to several empirical findings that suggest against it (see [31]). Of course, it remains possible that sensory-motor mismatch does play a contributing role in specific conditions, for example CPRS [32]. It is interesting that the presence of pain is important (i.e. impaired tactile acuity/MIP in knee OA compared with healthy controls) but that the intensity and duration of this pain is not.

The current results are of potential clinical importance because they suggest that rehabilitation might include training the cortical representation of the painful body part as well as the body part itself. This approach has been used to good effect in other painful conditions [10, 13, 14, 54]. A recent case series in people with chronic LBP evaluating a 12-week programme of sensory and motor retraining demonstrated promising results—there was an average of 85% reduction in pain [14]. However, that there was a clear relationship between the variables in back pain (and controls), but not in knee OA, suggests against a one size fits all approach. It may be that tactile training has limited effect in painful knee OA by virtue of widespread mechanoreceptor impairment [50]. Clearly, further work is required before specific clinical interventions can be suggested.

Limitations

In Experiment 1, age differences were present between the healthy controls and the patient samples. Age affects TPD thresholds (older age, larger TPD thresholds) [55]. However, that our result stood when compared with age-matched arm pain patients shows that age does not explain the result, but it may modulate it. We previously established [4] that the age difference between groups was not critical to accuracy findings [4].

Conclusion

Painful knee OA is associated with decreased tactile acuity, a clinical signature of S1 organization, but the extent of tactile acuity deficit does not relate to pain intensity or duration. Tactile acuity and MIP are not related in people with painful knee OA, but they are related in people with back pain and in healthy pain-free controls.

Rheumatology key messages

- People with knee OA have disrupted tactile acuity at the knee.
- The relationship between tactile acuity and motor imagery performance is unique to knee OA/back pain.
- In knee OA/back pain, pain relates to neither tactile acuity nor motor imagery performance.

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Supplementary data

Supplementary data are available at *Rheumatology* Online.

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