

**Hypervigilance for bodily sensations in the back during a movement task in people
with chronic and recurrent low back pain**

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Abstract

Objectives. The current study assessed the role of hypervigilance for bodily sensations in the back in long term low back pain (LBP) problems. **Methods.** People with chronic low back pain (CLBP), recurrent low back pain (RLBP), and no LBP were compared on the extent to which they attended to somatosensory stimuli on the back during a movement task. To measure hypervigilance, somatosensory event-related potentials (SEP) to task-irrelevant tactile stimuli on the back were measured when preparing movements in either a threatening or a neutral condition, indicated by a cue signaling possible pain on the back during movement or not. **Results.** Results showed stronger attending to stimuli on the back in the threat condition than in the neutral condition, as reflected by increased amplitude of the N96 SEP. However, this effect did not differ between groups. Similarly, for all three groups the amplitude of the P172 was larger for the threatening condition, suggesting a more general state of arousal resulting in increased somatosensory responsiveness. No significant associations were found between somatosensory attending to the back and theorized antecedents such as pain catastrophizing, pain-related fear and pain vigilance. **Discussion.** The current study confirmed that individuals preparing a movement attended more towards somatosensory stimuli at the lower back when anticipating back pain during the movement, as measured by the N96 SEP. However, no differences were found between participants suffering from CLBP or RLBP, or the healthy controls.

Keywords

SEP, EEG, chronic low back pain, recurrent low back pain, somatosensory attention

1. Introduction

The fear-avoidance model states that long term back pain problems might develop as a result of persistent fear-induced 'safety-seeking behaviors', including activity avoidance and 'hypervigilance' (i.e., heightened attending) to pain-related information and bodily sensations. While the evidence for the role of avoidance behavior is abundant², the status of hypervigilance is less clear. Research has focused almost exclusively on attending to semantic representations of pain. Although meta-analyses suggest increased attending to (sensory) pain words in chronic pain patients compared to healthy controls, these effects were small³ and no consistent associations with theorized antecedents (e.g., fear) were found⁴.

It has been argued that semantic pain stimuli, such as pictures showing painful situations or words related to the experience of pain, might be insufficient to evoke bodily threat, and it has been recommended to focus on attentional processing of somatosensory stimuli^{4,5}. There has been increasing effort to develop somatosensory attention paradigms^{6,7}, but research was mainly limited to pain-free populations. One notable exception is a study by Peters and colleagues⁸, who instructed chronic low back pain (CLBP) patients to detect electrical stimuli of slowly increasing intensity on the back while performing an auditory task. No evidence for hypervigilance was found, as CLBP patients were not faster than pain-free controls in detecting stimuli on the back. However, a potential problem is that both groups may have strongly focused on the somatosensory stimuli because of the task instructions, whereas attending to pain is rather a spontaneous reaction. It is therefore unclear how attention to pain is guided without any instruction to do so.

In an attempt to solve this problem Clauwaert and colleagues^{9,10} used electroencephalography (EEG) to assess somatosensory event-related potentials (SEPs) to task-irrelevant tactile stimuli on the hands⁹ or the lower back¹⁰ while preparing pain-conditioned and neutral movements that recruited the simulated body part. In both studies, they identified similar increased SEPs indicative for somatosensory attending (i.e. the N120 when stimulating at the hand and N95 when stimulating at the lower back) when stimuli were presented on the body part that was recruited to perform the pain-conditioned movement.

The current study used a similar paradigm as Clauwaert and colleagues¹⁰ for application to low back pain (LBP). SEPs to tactile stimuli on the back were recorded while participants prepared rapid arm movements in either threatening or neutral conditions, indicated by a cue signaling whether the movement could be accompanied by a painful stimulus on the back or not. We compared a control group without LBP to persons with CLBP and recurrent low back pain (RLBP), thereby taking the degree of chronicity of LBP into account.

We hypothesized that somatosensory attending to the back, as indicated the N120/N95 SEPs amplitudes in particular, would be increased in the threat than in the neutral condition, and that effects would be larger in the LBP groups than in the control group. Given the difference in chronicity, we expected larger effects in the CLBP than in the RLBP group. Finally, we explored associations between somatosensory attending to the back and theorized antecedents (pain catastrophizing, pain-related fear, pain vigilance).

2. Method

2.1 Participants

An a priori sample size calculation showed that a minimum of 66 participants was necessary to detect a medium effect size (Cohen's $f = 0.25$) at $\alpha = 0.05$ and 95% power. We exceeded this amount in order to increase the chance to detect small-to-medium effects. One hundred and nine individuals with CLBP (N=32), RLBP (N=33), and without LBP (N=44) were recruited via social media, flyers distributed in public areas, several Belgian hospitals, and private practices of physicians or physiotherapists. All included LBP patients had to suffer from non-specific LBP, which was defined as pain in the lumbar region that is not attributable to a recognizable, known specific pathology (e.g. histories of spinal traumata or deformities, severe degenerative changes or scoliosis, osteoporosis, obesity, radicular signs, malignancies, metabolic or rheumatologic diseases, spinal surgery, neuropathic pain, etc.¹¹). The LBP had to be initiated ≥ 6 months ago and was of such a severity that it lasted for at least 24 hours, interfered with daily activities and a clinician (medical doctor or physiotherapist) had been consulted^{12,13}.

Individuals were classified as RLBP when they experienced ≥ 2 reoccurring LBP flares per year during which the mean LBP intensity was ≥ 2 on a visual analogue scale (VAS)^{13,14}. The painful episodes were alternated with pain-free episodes of LBP remission lasting ≥ 1 month^{13,15}. The RLBP patients included in this study were examined while in a state of remission. This way, potential differences between the RLBP and control group could not be confounded by current presence of pain, and thus should reflect cognitive processes. Individuals were classified as CLBP when the pain complaints were present weekly, occurring at least on 3 out of 7 days^{16,17}.

For the control group participants were allowed if they did not suffer from any pain disorders (in the past) nor pain complaints at the moment the experiments took place, and who never experienced LBP complaints >24 hours of that severity that they consulted a (para)medic.

Individuals with (a history) of severe respiratory, orthopedic, neurological, systematic, metabolic or circulatory conditions, or with a history of spinal surgery, spinal trauma or severe spinal deformities were not eligible for study inclusion. Additionally, pregnant women were not allowed to participate. Furthermore, only Dutch speaking participants between the ages of 18 and 45, with a healthy body mass index ($BMI \leq 25$) were eligible. Importantly, the 3 groups were matched on gender and age. Both right- and left-hand dominant participants were included.

Participants were asked to refrain from consuming caffeine, alcohol, nicotine and physical exertion 48 hours before or on the day of the experiments. Moreover, participants were asked not to take in painkillers, muscle-specific or general relaxant medication to eliminate strong acute effects, and to maintain a normal sleep pattern the night before testing.

Before the start of the testing, participants received an information brochure about the study. Participants were also told that they were free to not participate or to terminate the experiment at any time should they so desire. All participants agreed to continue with the experiment and signed an informed consent. The participants took part in the experiment in exchange for a monetary reward and were not informed about the specific goals of this study before the start of the experiment. However, at the end of the experiment all participants received an elaborate debriefing. This study was approved by the committee of medical ethics of Ghent University (study 2016/0168), where the

experiment took place, and was performed according to the ethical standards laid down in the declaration of Helsinki.

2.2 Materials

2.2.1 Tactile stimulus. A resonant-type tactor (C-2 TACTOR, Engineering Acoustics, Inc., Florida) was used to administer vibrotactile (VT) stimuli (200 ms) to the low back, centrally at the L3 spinous process level. The amplitude and frequency were controlled by a self-developed software program. The tactor was attached directly to the skin surface by means of a double-sided tape ring and was driven by a custom-built device at 200 Hz. To prevent any interference from environmental noise, participants were asked to wear earplugs. The intensity of the VT stimulus was the same for all participants and did not vary across the experiment.

2.2.2 Electrocutaneous stimulus. The painful electrocutaneous stimulus (ES, bipolar; 50Hz; 200 ms; instantaneous rise and fall time) was delivered by means of a Constant Current Stimulator (DS5, Digitimer Ltd, Hertfordshire, UK) with two lubricated Medcat surface electrodes (1cm diameter). These electrodes were placed directly underneath the tactor at the L4 spinous process. Participants were first presented with an ES of low amplitude (0.5 mA) to prevent the initial surprise from influencing the evaluation of the stimulus. After this, the participants were presented with the same stimulus and were motivated to choose an intensity that they evaluated as unpleasant as possible but that they were still willing to receive during the experiment. After every stimulation, the participant was asked to indicate whether the researcher was allowed to increase the intensity or not. If the participant agreed, the amplitude was elevated in steps

of 0.5 mA until the participant indicated to have reached the maximum intensity (procedure in line with ^{9,18}). Once a higher amplitude was chosen, the participants could not go back to a lower amplitude. Since movement can suppress the perception of sensory information (i.e. sensory suppression)^{19,20}, the participants also received their individually chosen maximum intensity while performing a rapid arm movement and were asked again whether they agreed to increase the intensity or not. If they agreed, the intensity was increased in steps of 0.5 mA until they reached their maximum intensity during movement execution.

2.2.3 Sensor-box. To register the start of the movement execution, a custom-built optical sensor-box was used. This sensor-box was attached to the participant's hip at the side of the dominant arm, at a height which the participants could easily reach with fingertip.

2.2.4 EEG. Brain activity was recorded continuously using the eego sports (ANT neuro system) recording system at a sampling rate of 2,000 Hz from 32 active electrodes, placed according to the international 10/20 setting. The ground electrode was located in the active-shield cap fronto-centrally between the FPz and the Fz electrode and all channels were referenced online to the average of all signals. Impedances were kept below 10 k Ω . Data were further preprocessed off-line by using Brainvision Analyzer 2.1 (Brain Products GmbH, Munich, Germany).

2.2.5 Experiment software. The experiment was programmed in C-language using the Tscope 5 library package²¹. Triggers were controlled by the experimental software and sent through a custom-made device which allowed to send triggers to the EEG system.

2.3 Design

This study was part of a larger project in which participants were invited for two different testing sessions: a session in which the effect of threat of experimentally induced back pain was examined (*pain session*), and a session without any administration of experimental pain (*no-pain session*). The pain session included both painful and pain-free trials, whereas the no-pain session included only pain-free trials. There were at least 5 days between the two sessions, and the order of these sessions was randomized across participants. Note that only the procedure and the data of the pain session will be reported in the current paper, since the no-pain session had different research goals and used a somewhat different paradigm. However, because the same movements had to be performed in both sessions, any order effects will be controlled for.

2.4 Experimental procedure

Participants were asked to stand straight in front of a computer, with the feet at shoulder width and the arms hanging relaxed alongside their body. A screen was positioned at the eye level of the participant 2m in front of them. First, the intensity of the ES was calibrated for each participant. Next, the participants learned to execute the arm movement correctly as instructed by one of the experimenters. This movement consisted of moving the dominant arm^{22,23,24} away from a sensor box attached at the hip, by performing either a forward arm movement with a stretched arm towards 90° of shoulder flexion, or backwards in an angle of 30° of shoulder extension, and back to the sensor-box as quickly as possible²². These rapid arm movements (RAM) were chosen because

they disturb the trunk posture, eliciting an anticipatory postural response of the lower back muscles to restore balance. Moreover, this task has been previously used to this end in healthy people as well as those with LBP^{25,26}. This specific motor task was selected to allow the recording of electromyography (EMG) during movement execution, which was not the focus of this study but part of the larger project. EMG data were not reported in the present manuscript. In the movement practice phase, participants practiced the movement under supervision of the experimenters and received feedback about their performance for a total of 6 trials (3 in each direction). Meanwhile, one of the experimenters evaluated the accuracy and speed of the movement, as well as whether a stretched arm during movement execution was maintained. If needed, the practice phase was repeated until the movement was executed as requested.

Following the movement training, the experiment started with a practice block of 24 trials, which was not included in the analyses. In this block, the participants learned the association between the color of a cue presented on the screen (i.e. a blue or pink ball) and the possible administration of either an unpleasant ES stimulus or a non-painful VT stimulus during movement execution. The association between the color of the ball and the type of stimulus was randomized across participants. Moreover, participants were verbally informed of this association by the experimenter before the start of the experiment, to facilitate learning²⁷. Each trial started with the presentation of a fixation cross (500ms), followed by a blue or pink ball. The duration of this cue was 3000ms. During this interval, a VT with a duration of 200ms was presented at the lower back to induce the somatosensory evoked potentials. The moment of stimulation onset varied between 2000 and 2500ms after the presentation of the cue. After this cue had disappeared, a second cue was presented, indicating which movement had to be

performed. When a white upwards-pointing arrow was presented, the participants were instructed to execute a forward arm movement as quickly as possible. When the arrow pointed downwards, the participants were instructed to perform the backward movement. When the cue “STOP” was presented, the participants were instructed to refrain from moving. Reaction times were defined as the time between movement cue (the arrow) onset and the release of the sensor box. Participants were motivated to perform the complete movement as fast and as accurately as possible. By including the backwards movement, the direction of the movement was made unpredictable.

The participants received either a painful ES or a VT stimulus during movement execution in 1 out of 3 trials, depending on the color of the first cue. When the participants did not have to perform a movement, they never received any additional stimulation. The VT was triggered as soon as the participants lifted their finger from the light-sensitive sensorbox. To make sure the participants executed the movement correctly and in the correct direction, the participants were monitored by the experimenters. When participants had executed the movement, a timeline counting down 12 seconds was shown on the screen. In this timeframe, participants were instructed to keep breathing and to relax their muscles and had the time to position their fingertip back on the sensor-box and prepare for the next trial ^{22,23}. The next trial started after an inter-trial interval of 500ms (see figure 1 for an overview). After this practice block, the experimental blocks started. The experimental blocks were exactly the same as the practice blocks, except for the number of trials. The experiment was divided into 2 blocks of 120 trials, with a seated rest of 90 seconds in-between blocks.

[figure S1 about here]

2.5 Self-report instruments

After obtaining informed consent, all participants were asked to fill out a general questionnaire in which the participants' sociodemographic variables (name, date of birth, gender, civil status, parenthood, education, and profession), general health (pregnancy, medical and psychological health problems, medical procedures, treatments and therapy, and pain complaints). Participants with LBP were asked to fill out an additional questionnaire to register LBP related information (e.g. the type, intensity, duration, localization, of the LBP, current and past treatments). In case the latter questionnaire indicated potential red flags for LBP of neurogenic origin a clinical examination was performed by a PT to rule out this was the case. Additionally, all participants were asked to complete Dutch versions of several validated questionnaires to assess theoretical antecedents of attention, severity of the problems, and psychological distress.

2.5.1 Hospital Anxiety and Depression Scale (HADS) is developed to identify anxiety disorders and depression among patients with a general medical condition²⁸. The HADS is a 14-item scale in which the participants have to report on a 4-point Likert scale the degree to which they have experienced anxious and or depressive feelings over the last week. The scale is divided into two subscales: 7 items for anxiety and also 7 for depression. Higher scores indicate greater levels of depression and anxiety, with scores between 8 and 10 considered as mild, 11 and 14 as moderate and between 15 and 21 as severe, for each subscale. The HADS has been shown to be a reliable and valid questionnaire in both general^{29,30} and chronic pain populations^{31,32}.

2.5.2 Pain Catastrophizing Scale (PCS) is a 13-item scale in which the participants are asked to reflect on previous painful experiences and to indicate their thoughts and feelings when experiencing pain³³. Responses are given on a 5-point Likert scale ranging from 0 (not at all) to 4 (all the time). Larger scores represent larger catastrophizing behavior levels. Scores are clinically relevant when ≥ 30 . Larger scores represent larger catastrophizing behavior levels. The PCS consists of three subscales: magnification, rumination, and helplessness. The Dutch version of the PCS has been shown to be valid and reliable both in healthy and chronic pain patients³⁴.

2.5.3 Pain Vigilance and Awareness Questionnaire (PVAQ) is a scale that consists of 16 items in which participants are asked to report on their vigilance for pain sensations on a Likert scale from 1 (“never”) to 5 (“always”)³⁵. The PVAQ consists of two subscales, namely attention to pain and attention to changes in pain. High scores reflect increased levels of hypervigilance to pain sensations. The Dutch version of the PVAQ has been shown to be valid and reliable in both healthy populations and chronic pain patients^{36,37}.

2.5.4 The Roland Morris Disability Questionnaire (RMDQ) was used to assess how daily physical activities and functioning are affected by LBP³⁸. Participants are asked to answer 24 ‘yes-no’ questions on whether they experienced a specific situation regarding their low back pain that day. The total score ranges from 0 (no disability) to 24 (severe disability) with higher scores indicating higher degrees of LBP related disability. The Dutch version of the RMDQ is shown to be valid and reliable to assess dysfunction in chronic low back pain³⁹.

2.5.5 The Tampa Scale of Kinesiophobia (TSK) a 17-item questionnaire that measures the fear of movement and (re)injury⁴⁰. Items are answered on a 4-point Likert

scale ranging from 1 (“strongly disagree”) to 4 (“strongly agree”). A high value on this scale indicates a high degree of kinesiophobia, with a cutoff score of 37. The TSK has shown to be valid and sufficiently reliable^{41,42}.

2.5.6 Other self-reports. Immediately after the experiment, participants were asked to rate several items on a Likert scale from 0 to 10: expectations of pain (‘to what degree did you expect a white/pink/blue ball/rest trial to be followed by an unpleasant stimulus?’), fear of pain (‘to what degree did you fear that a white/pink/blue ball/ rest trial would be followed by an unpleasant stimulus?’) during the experiment, and unpleasantness (‘how unpleasant do you rate the electrocutaneous stimulus?’) and painfulness (‘how painful do you rate the electrocutaneous stimulus?’) of the ES (only in the pain session). Additionally, all participants were asked to indicate the amount of back pain they experienced at the day of testing on a VAS scale from 0 to 10.

2.6 Data processing and analyses

2.6.1 Self-reports. Only participants’ the scores on theoretically relevant self-reports and questionnaires (i.e. the PVAQ, PCS, and TSK) were compared between groups by conducting repeated-measures ANOVA’s and/or t-tests where applicable. Additionally, the total scores on the PCS, PVAQ, TSK, fear and expectancy ratings were correlated with the participants’ SEP amplitudes and RTs.

2.6.2 EEG. Channels were re-referenced off-line to the average of all electrodes. EEG-signals were filtered with a low cutoff of 1 Hz and a high cutoff of 30 Hz. Next, an automatic artefact rejection was applied to the segments that ranged from -200 to 500 ms around the onset of the tactile stimulus. Through artefact rejection, all eye movements

that occurred right before and during tactile stimulus presentation were also removed (7.0 % no threat trials and 7.0 % of the neutral trials for the controls, 8.4% and 8.8% for the RLBP, and 5.9% and 6.9% for the CLBP group correspondingly). Finally, baseline corrections were applied, and the average was calculated for each condition (trials with the threat of receiving the painful ES and trials without the threat of pain). A collapsed localizer was created by averaging the waveforms of all participants and all conditions⁴³.

Based on previous studies^{9,10,44} and visual inspection, clear peaks were detected at 23 ms (positive), 30 ms (negative), 40 ms (positive), 96 ms (negative), and 172 ms (positive) (see figure 2). The presence of these peaks was confirmed by calculating the global field power across all participants and conditions. All peaks had a central topography centered around the Cz electrode. Mean area amplitudes were therefore exported from electrodes FC1, FC2 and CZ for the P23, N30, P40 and N96, and from electrodes FC1, FC2, Cz, CP1 and CP2 for the N172 component. This area information was extracted from an interval between 22 and 28 ms (P23), 26 and 34 ms (N30), 35 and 45 ms (P40), 71 and 121 ms (N96), and an interval between 132 and 212 ms (P172). Mean area amplitudes were used because these are known to provide an unbiased measure of amplitude⁴⁵. Comparisons between the participant groups (3 levels) and the two conditions (2 levels: trials with the threat of receiving the painful ES and trials without the threat of pain) were made by means of a 3 x 2 repeated-measures ANOVA, with condition as within-subjects factor and group as between-subjects factor, and additional t-tests where applicable.

[figure S2 about here]

2.6.3 Reaction times (RT). Since we did not have any predictions about movement direction, the data from both directions were combined. All outliers were removed from the dataset (1.90%). This was done by eliminating all RTs lower than 100 ms and larger than 2000 ms. Next, all RTs that were faster or slower than 3 times the standard deviation were also removed. RT data will be analyzed by conducting a 3x2 (group x condition) repeated-measures ANOVA.

3. Results

3.1 Participants

One participant fainted during the pain session and was excluded from the analyses. Additionally, two participants dropped out after the no-pain session. Moreover, due to technical problems the EEG data of 16 participants was not recorded properly. Finally, 3 RLBP participants reported to experience a pain episode during the sessions and were excluded. In total, the data of 88 (34 control, 28 CLBP and 26 RLBP) individuals were included in the analysis (see table 1 for demographics).

The RLBP individuals rated on a VAS scale from 0 to 10 their average pain intensity a 5.15 ($SD = 1.75$) out of 10, and their maximum pain intensity a 6.70 ($SD = 1.75$) out of 10. The duration of the pain free periods ranged across participants between 1 day and several weeks (up to 6 weeks). The participants' last pain flare ranged between 1 day to 9 months before the testing day. On average, the participants rated the intensity of their last pain flare a 4.97 ($SD = 2.03$) on a VAS scale from 0 to 10. The maximum pain intensity they felt during their last pain flare was rated 5.71 ($SD = 2.32$). The duration

of the last pain flare ranged between 3 hours and 14 days. Twelve participants have sought non-pharmacological treatment and 13 participants have used pharmacological treatment. Seven participants have used other types of treatment. Note that participants have combined several types of treatment.

On a VAS scale from 0 to 10, the CLBP individuals on rated their pain intensity on average (not specifically on the testing day) 4.23 ($SD = 1.47$) and the maximum intensity 7.04 ($SD = 1.78$). Seventeen participants have sought non-pharmacological treatment and 15 participants have used pharmacological treatment. Eight participants have used alternative treatments.

[table 1 about here]

3.2 Self-reports

Table 1 shows the average scores on the questionnaires and other ratings. For the PCS, PVAQ, and TSK, there were no significant differences between the three groups (p 's $>.05$). Moreover, the groups did not differ significantly in the ratings on the painfulness and unpleasantness of the ES, pain expectancies, pain-related fear and low back pain ratings (all p 's $>.05$).

There were no differences between the groups on their total HADS score (all p 's $>.05$) or their scores on the depression subscale (all p 's $>.05$). However, on the anxiety subscale, there was a significant difference between the healthy and the RLBP group ($t(43.479)=-5.24$, $p<.001$, $d = 0.68$), and a significant difference between the healthy

group and the CLBP group ($t(53.27)=-3.20, p=.002, d=.41$). There was no difference between the CLBP and RLBP groups ($t(51.73)=-.75, p=.455, d=.11$).

3.3 SEPs

[table 2 about here]

3.3.1 P23. There were no significant main effects (condition: $F(1,85)=.05, p=.820, d=.02$, group: $F(2,85)=.30, p=.740, d=.06$), and no significant interaction between group and condition ($F(2,85)=2.53, p=.085, d=.17$).

3.3.2 N30. There was no significant main effect of condition, $F(1,85)=1.65, p=.202, d=.14$, nor a significant main effect of group, $F(2,85)=1.91, p=.154, d=.15$. Also, no significant interaction effect was found ($F(2,85)=2.62, p=.079, d=.17$).

3.3.3 P40. There were no significant main effects (condition: $F(1,85)=.36, p=.550, d=0.06$, group: $F(2,85)=2.79, p=.067, d=.18$), and no significant interaction between group and condition ($F(2,85)=.52, p=.597, d=.08$).

3.3.4 N96. There was a significant main effect of condition, $F(1,85)=11.59, p=.001, d=0.36$, with a stronger negative waveform in threat trials than in neutral trials. No significant main effect of group $F(2,85)=.08, p=.925, d=.03$, nor a significant interaction between group and condition $F(2,85)=.06, p=.943, d=.03$, was found.

3.3.5 P172. A significant main effect of condition was found, $F(1,85)=9.75, p<.005, d=.33$, with a stronger positive waveform in threat trials compared to neutral

trials. There was no significant main effect of group ($F(2,85)=3.05, p=.053, d=.19$) and no significant interaction between group and condition ($F(2,85)=.29, p=.750, d=.06$)¹.

3.4 Movement latency

The reaction time data of three participants were not registered correctly. Therefore, the data of these participants were not included in the analyses. The results showed that a significant main effect of condition ($F(1,81)=42.74, p<.001, d=.70$), with faster reaction times in threat trials ($M=454.53, SD=65.14$) compared to neutral trials ($M=467.93, SD=65.36$). There was no significant main effect of group ($F(1,81)=0.78, p=.461, d=.09$). There was also no significant interaction effect ($F(2,81)=.28, p=.755, d=.06$).

3.5 Correlations

The participants' scores on the PVAQ, PCS, TSK, and their fear and expectancy ratings on the pain and no pain trials were correlated to the amplitudes of the different components. After Bonferroni correction was applied, none of the correlations reached significance.

4. Discussion

¹ To check whether order of the session had an influence on the results, separate analyses were conducted which included session order as a factor. The effect of order never reached significance (always $p>0.1$) and was therefore excluded from the analyses described in this paper.

The current study evaluated the role of hypervigilance in long term low back pain problems. SEPs were assessed to examine attentional processing of somatosensory stimuli at the lower back when preparing arm movements with or without threat of pain on the back. Individuals with CLBP, RLBP, and no LBP were compared. Results showed a significant effect of condition on the amplitude of the N96 SEP, indicating that, as expected, the participants attended more towards the stimuli on the back in threat trials than in neutral trials. However, the P172 SEP was larger when anticipating a threatening movement compared to a safe movement, but there was no difference between groups. Additionally, no significant associations between somatosensory attending to the back and theorized antecedents of hypervigilance were found.

The results showed larger N96 amplitudes in the threat trials compared to the neutral trials. This effect seems reminiscent to what was previously found in a study by Clauwaert and colleagues¹⁰. Similar to the current study, we measured SEPs to task-irrelevant tactile stimuli on the lower back while healthy individuals prepared pain-conditioned and neutral arm movements and found that the N95 component was larger when preparing the pain-conditioned movement, indicating increased somatosensory attending. The current study replicates these findings by showing an increased N96 for somatosensory input at the lower back when preparing a back-threatening movement. Contrarily to our hypothesis, the N96 effect did not differ between the CLBP, RLBP, and control groups. This seems to suggest that, in line with the study by Peters and colleagues⁸, persons with LBP problems were not hypervigilant for bodily sensations in the back. Note that also in persons with fibromyalgia, no behavioral evidence has been found for somatosensory hypervigilance^{46,47}. However, such conclusion might still be

premature, and a number of issues should be considered. First, we examined how the threat of brief phasic electrocutaneous stimuli affected somatosensory attending to the back. It is possible that such effect is not representative for the attentional processes involved in naturally occurring back pain. However, it should be noted that also in the neutral condition (perhaps better reflecting the natural situation), no differences were found between groups. Nevertheless, it might be worthwhile for future studies to consider using tonic pain inductions. Second, it could well be that the threat of experimentally induced pain was not different between the groups, and therefore affected attending to the back to the same extent. The fact that the ratings of the ES intensity, expectations and fear did not differ between the different groups, seems to support this explanation. Third, the CLBP and RLBP groups were recruited from the general population rather than specialized clinical settings alone, which might have resulted in a more heterogeneous sample. While only participants who met the inclusion criteria for CLBP and RLBP were selected, the questionnaire scores suggest that these were relatively well functioning samples. For example, the average scores on the RMDQ were only 4.54 and 5.89 out of a maximum score of 24 for the CLBP and the RLBP groups correspondingly. Furthermore, the scores on the PCS and TSK scales are lower than those achieved in other studies on CLBP individuals^{34,41,48}, and did not significantly differ from the scores of the control group. The samples achieved in the current study may therefore not be representative of LBP patients who are more severely disabled.

Interestingly, there was a significant effect of condition on the P172 component, which was larger in the threat condition compared to the safe condition. This component might correspond with the P166 component found in the study by Clauwaert et al.¹⁰, and which has been suggested to reflect a state of arousal during threat trials, resulting in

increased somatosensory responsiveness. Note that we also identified a number of earlier components (0-50 ms after tactile stimulus onset), which were not affected by condition or group. We had no hypotheses about these early SEPs, which have been suggested to originate from activity in the primary somatosensory cortex (SI)⁴⁹. Interestingly, it has been suggested that early onset SEPs may reflect the suppression of sensory information that usually occurs when executing and even preparing a movement (i.e. sensory suppression⁵⁰). It has been hypothesized that CLBP might negatively affect sensory suppression⁵¹, but the current study did not find indications for this.

An interesting observation in the present study was that participants initiated the movement faster when they expected it to be combined with a painful stimulus on the back. Based on literature one would rather expect participants to be more hesitant when initiating a movement associated with pain^{52,53}. Possibly, this counter-intuitive finding could be explained by the fact the participants wanted to end the pain as soon as possible (“let’s get it over with”)⁵⁴. Alternatively, the fearful anticipation of a painful stimulus may have activated a defensive response priming the motor system for escape from the threatening situation^{55,56,57} even though escaping from the threat was actually not possible for participants. Interestingly, the response latencies did not differ between the groups.

The SEP amplitudes did not correlate with the self-reported fear and expectation of pain during the experiment nor with the fear and vigilance for pain, or the pain related catastrophizing behaviors. Since the expectancy or fear of pain is known to motivate people to scan their body for threats, these results are quite unexpected. It is possible, however, that the measures used in this study (both self-reports and/or ERP data) are not sensitive enough to detect the individual differences in the current experimental paradigm.

To conclude, the current study confirmed that individuals preparing a movement attended more towards somatosensory stimuli at the lower back when anticipating back pain during the movement, as measured by the N96 SEP. However, no differences were found for this component between participants suffering from CLBP or RLBP, or the healthy controls. Additionally, the present study was not able to find associations between somatosensory attention and pain-related attending and theorized antecedents such as (self-reported) pain catastrophizing, pain-related fear, and pain vigilance.

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Author contributions

All authors conceived of the present idea and paradigm. A.C. programmed the experiment. A.C. and S.S. conducted the study. A.C. processed all the data. A.C. wrote the manuscript in consultation with all other authors.

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Figure legends

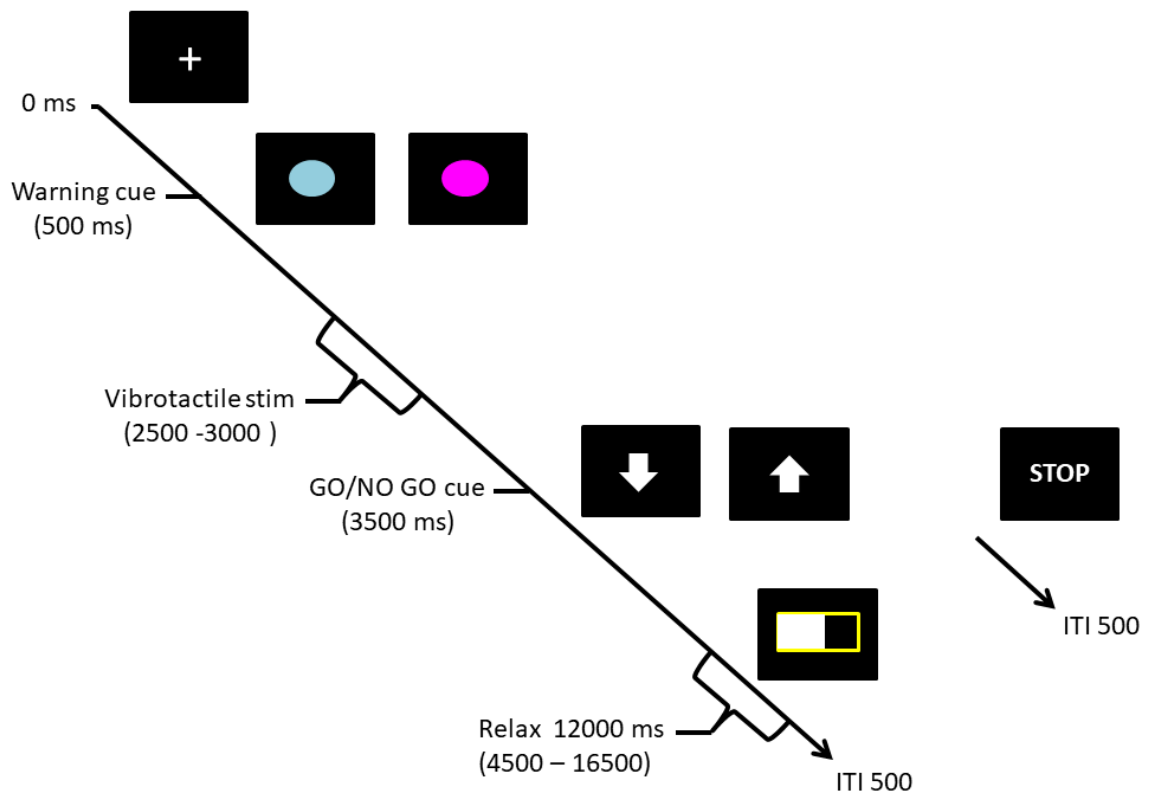
Figure 1. Overview of the trials. A fixation cross was presented at the start of the trial (0 ms). After 500 ms, the fixation cross was replaced by cue 1, indicating whether the participant could expect a painful ES or neutral VT during movement execution. Between 2500-3000 ms after the onset of the trial, participants received a VT (200 ms) to evoke an SEP. Next, at 3500 ms after onset of the trial, cue 1 was replaced by the movement cue. A movement trial ended after 16500 ms. The next trial started after an ITI of 500 ms.

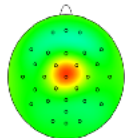
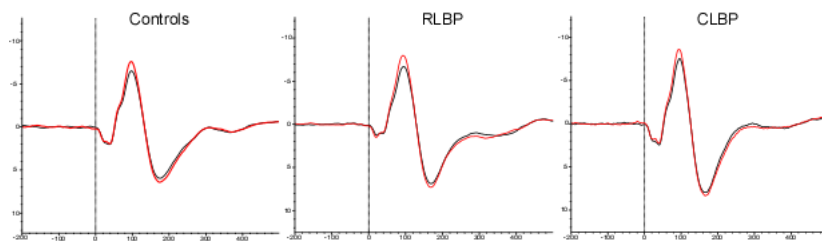
Figure 2. Above: waveforms as presented at the Cz electrode for both conditions (red lines: threat condition, black lines: no threat condition) for the three different groups (healthy controls, recurrent low back pain group and chronic low back pain group) Below: current source densities (CSD) at each peak (P23, N30, P40, N96, P172)

Table legends

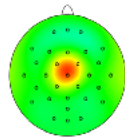
Table 1. Demographics and questionnaire scores for the different participant groups. All scores represent means (M) and standard deviations (SD) unless otherwise specified.

Table 2. Mean amplitudes and standard deviations for each condition and group.

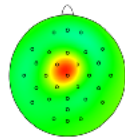




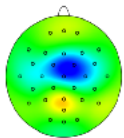
P23
20 ms - 26 ms



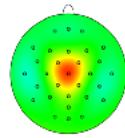
N30
20 ms - 35 ms



P40
35 ms - 45 ms



N96
71 ms - 121 ms



P172
132 ms - 212 ms

Table 1. Demographics and questionnaire scores for the different participant groups. All scores represent means (M) and standard deviations (SD) unless otherwise specified

| | Controls | RLBP | CLBP |
|--|---------------|---------------|--------------|
| <i>N</i> | 34 | 26 | 28 |
| gender (<i>N</i> female) | 17 | 15 | 15 |
| age in years | 32 (6.71) | 29 (6.64) | 31 (7.13) |
| righthandedness (<i>N</i> left dominant) | 2 | 3 | 4 |
| selected stimulus intensity in mA | 4.0 (2.12) | 4.6 (2.30) | 3.8 (2.77) |
| education years | 17.13 (2.93) | 17.42 (2.12) | 17 (2.63) |
| back pain at day of testing | .05 (.22) | .87 (1.13) | 2.46 (1.94) |
| painfulness electrocutaneous stimulus | 4.29 (2.22) | 4.49 (2.27) | 4.94 (2.32) |
| unpleasantness electrocutaneous stimulus | 5.11 (2.42) | 5.64 (1.93) | 5.75 (2.62) |
| rating pain expectancies after pain cue | 4.47 (1.90) | 5.43 (1.74) | 4.9 (2.70) |
| rating pain expectancies after no pain cue | .14 (.42) | .26 (1.12) | .34 (.79) |
| rating pain expectancies after rest cue | .77 (1.13) | .69 (1.52) | 1.18 (2.31) |
| fear for electrocutaneous stimulus after pain cue | 3.95 (2.90) | 5.06 (2.94) | 4.29 (2.84) |
| fear for electrocutaneous stimulus after no pain cue | .10 (.35) | .08 (.31) | .37 (.94) |
| fear for electrocutaneous stimulus after rest cue | .58 (1.18) | .44 (1.19) | .99 (2.11) |
| pain ratings during experiment | .68 (1.34) | 2.74 (2.68) | 4.80 (2.78) |
| HADS total score | 6.06 (5.12) | 8.08 (4.54) | 9.86 (6.21) |
| HADS depression subscale | 2.05 (2.53) | 3.00 (2.50) | 3.25 (3.34) |
| HADS anxiety subscale | 4.00 (2.90) | 5.08 (2.87) | 6.61 (3.41) |
| PVAQ total score | 29.38 (12.02) | 33.23 (11.05) | 34 (12.76) |
| PCS total score | 11.35 (9.05) | 12.69 (7.98) | 14.64 (7.52) |
| TSK total score | 31.82 (8.67) | 33.85 (7.69) | 33.14 (8.86) |
| RMDQ total score | - | 4.54 (4.25) | 5.89 (3.47) |

Table 2. Mean amplitudes and standard deviations for each condition and group.

| | | Controls | RLBP | CLBP |
|------|---------|--------------|--------------|--------------|
| P23 | pain | 1.32 (.97) | 1.27 (1.05) | 1.13 (1.13) |
| | no pain | 1.39 (.96) | 1.07 (.97) | 1.31 (1.01) |
| N30 | pain | 1.50 (1.13) | 1.06 (.91) | 1.19 (1.39) |
| | no pain | 1.60 (1.15) | .94 (.80) | 1.44 (1.17) |
| P40 | pain | 1.57 (1.38) | .83 (1.08) | 1.53 (1.72) |
| | no pain | 1.56 (1.46) | .83 (.91) | 1.67 (1.44) |
| N96 | pain | -5.71 (2.86) | -6.04 (3.49) | -5.98 (3.04) |
| | no pain | -5.03 (2.78) | -5.18 (3.21) | -5.28 (3.25) |
| P172 | pain | 3.34 (1.91) | 3.67 (2.26) | 4.50 (1.80) |
| | no pain | 2.95 (1.90) | 3.69 (2.12) | 4.30 (1.70) |