# SOMATOSENSORY ATTENDING TO THE LOWER BACK IS ASSOCIATED WITH RESPONSE SPEED OF MOVEMENTS SIGNALING BACK PAIN

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**Abstract**

The present study investigated if preparing a movement that is expected to evoke pain results in hesitation to initiate the movement (i.e., avoidance) and, especially, if the allocation of attention to the threatened body part mediates such effect. To this end, healthy volunteers (N = 33) performed a postural perturbation task recruiting lower back muscles. In 'threat trials', the movement was sometimes followed by an experimental pain stimulus on the back, whereas in 'no-threat trials', a non-painful control stimulus was applied. Electroencephalography (EEG) was used to assess attending to the lower back. Specifically, somatosensory evoked potentials (SEPs) to task-irrelevant tactile stimuli administered to the lower back were recorded during movement preparation. Reaction times (RTs) were recorded to assess movement initiation. The results revealed faster responses and enhanced somatosensory attending to the lower back on threat trials than on no-threat trials. Importantly, the amplitude of the N95 SEP component predicted RTs and was found to partially mediate the effect of pain anticipation on movement initiation. These findings suggest that somatosensory attending might be a potential mechanism by which pain anticipation can modulate motor execution.

**Keywords**

Pain, somatosensory attention, sensorimotor, EEG

## 1. Introduction

Though pain is a highly unpleasant sensation, it serves a crucial and sophisticated protective role: it signals potential danger to the body, allowing us to interrupt ongoing behavior and to respond appropriately to the threat (Eccleston & Crombez, 1999; Wiech & Tracey, 2013). In line with this view, research has shown that pain has widespread effects on motor output. On the one hand, the presence of pain can lead to movement adaptation, with effects ranging from subtle changes in muscle coordination to outright avoidance behavior (Hodges & Smeets, 2015). On the other hand, pain can also prime the motor system to facilitate escape responses (Morrison, Perini, & Dunham, 2013; Perini, Bergstrand, & Morrison, 2013).

However, this work focused mainly on the experience of pain. In contrast, in daily life, it is important to change behavior not only when experiencing but also when expecting pain to avoid potential tissue damage. Because pain is often experienced during or following the execution of certain movements, movement is an important cue to anticipate pain. As a result, it has been proposed that movement, like other pain associated cues, evoke conditioned protective responses including escape/avoidance, arousal, and hypervigilance (Vlaeyen & Linton, 2012). There is some evidence for this view. For example, learning that a movement can be followed by pain has been shown to evoke fear and cause hesitance to initiate the movement (Meulders & Vlaeyen, 2013; Meulders, Vansteenwegen, & Vlaeyen, 2011), but also increase movement velocity and acceleration (Karos, Meulders, Gatzounis, Seelen, Geers, & Vlaeyen, 2017). Both effects have even been found in the same study (Neige, Mavromatis, Gagné, Bouyer, Mercier, 2018). Thus, depending on the situation, expecting pain can trigger both avoidance- and escape-like responses. Interestingly, in a recent study by Postorino and colleagues (2017), the authors found decreased preparatory readiness potential amplitudes for movements to stop either painful or non-painful stimuli compared to movements without any stimulation. This

might suggest that there is no specific influence of pain on motor preparation, however, this study did not directly compare painful and non-painful tactile stimuli. Therefore, the question remains whether painful stimuli influence motor preparation differently compared to non-painful stimuli.

Consistent with the principle that attention is about selecting stimuli relevant for action (Allport, 1989), it has been argued that the initiation of such protective behavioural responses is facilitated by increased attending to somatosensory input at the threatened body location (Durnez & Van Damme, 2015; Van Damme, Legrain, Vogt, & Crombez, 2010). In support, attending to somatosensory input has previously been linked to motor processes, especially action preparation (Eimer, Forster, Van Velzen, & Prabhu, 2005; Galazky, Schütze, Noesselt, Hopf, Heinzez, & Schoenfeld, 2009; Van Ede, van Doren, Damhuis, de Lange, & Maris, 2015). Yet, how this process is affected by the anticipation of pain remains to be established. An interesting paradigm to investigate this question is the paradigm of Clauwaert, Torta, Danneels, and Van Damme (2018). These authors recorded somatosensory evoked potentials (SEPs) in response to tactile stimuli at the hand while participants prepared hand movements that were either associated or not associated with pain. The results showed that preparing hand movements that could be followed by pain resulted in heightened attending to somatosensory input at the threatened hand relative to the other hand, as indicated by enhanced N120 and P200 SEPs. How this effect was related to motor output was, however, was not examined.

Despite its importance in many theories to explain chronic pain (e.g. the fear-avoidance model, Vlaeyen & Linton, 2012), little is known about how and why attention is recruited, let alone how attentional processes may interfere with behavior. Therefore, understanding these processes may potentially be key to aid chronic pain. The aim of the current study was to investigate the contribution of somatosensory attention (Clauwaert et al., 2018) to the influence of pain anticipation on the execution of pain-related actions. Specifically, using an adapted

version of the Clauwaert et al. (2018) paradigm, we instructed healthy volunteers (N = 36) to perform rapid forward/backward arm movements, eliciting anticipatory postural activity of the lower back muscles (MacDonald, Mosely, & Hodges, 2009; Park, Tsao, Cresswell, & Hodges, 2014). During movement preparation, a visual cue indicated that these arm movements could be followed by either a painful electrocutaneous (ES) or a non-painful somatosensory stimulus to the lower back (threat versus no-threat trials; see figure 1 for an overview of the paradigm). Attending to the back was assessed in the preparation period by recording SEPs to task-irrelevant innocuous somatosensory stimuli presented at the lower back. Reaction times (RTs) were measured to assess arm movement initiation, as an index of motor output. While research has found both faster (Meulders et al., 2011; Meulders & Vlaeyen, 2013) and slower (Karos et al., 2017) responses when expecting pain, it can be predicted in the current study that participants will initiate the movements more slowly since escaping pain is not an option. Based on this, the following hypotheses were put forward: (1) pain anticipation modulates movement initiation, leading to slower RTs on threat trials than on no-threat trials; (2) pain anticipation results in heightened attending to somatosensory input at the lower back, indicated by enhanced SEPs in threat trials compared to no-threat trials; (3) this heightened somatosensory attending mediates the effect of pain anticipation on movement initiation.

[Figure 1 about here]

## 2. Results

### 2.1 Self-report data

The average ES intensity was 4.12 mA (*SD* = 2.25, range = 0.5 – 10.0 mA). The ES were rated as painful (*M* = 6.12, *SD* = 2.06) and unpleasant (*M* =6.63, *SD* = 2.00). Furthermore,

participants reported that they expected more pain when having to perform the movement in threat trials ($M$= 6.09, $SD$= 2.17) compared to no-threat trials ($M$=0.76, $SD$=1.62; $t$(32)= 13.164, $p$<.001, $d$=2.29), or rest trials ($M$= 2.12, $SD$= 2.26; $t$(32)= 3.004, $p$=.005, $d$=.52), indicating that the manipulation was successful.

**2.2 Does pain anticipation influence movement initiation?**

The effect of pain anticipation on RTs was significant, $t$(32.07)= -5.34**,** β = -0.13, *CI 95%* = [-0.17, -0.08], $p$ < 0.001. However, contrary to our hypotheses, this showed that RTs were not slower but faster on threat trials ($M$ = 583 ms, $SD$ = 78 ms) compared to no-threat trials (617 ms, $SD$ = 99 ms).

**2.3 Does pain anticipation influence somatosensory attending to the back?**

Three SEP components could be distinguished in the EEG signal: P22, N95, and P166. The effect of pain anticipation on SEP amplitude was significant for the N95 SEP, $t$(32.22) = -3.54, β = -0.05, *CI 95%* = [-0.08, -0.02], $p$ < 0.001, and P166 components, $t$(31.71) = 2.23, β = 0.04, *CI 95%* = [0.004, 0.067], $p$ = 0.033, but just failed to reach significance for the P22 SEP component, $t$(31.10) = -2.03, β = -0.03, *CI 95%* = [-0.05, 0.00], $p$ = 0.051. As predicted, N95 SEP and P166 SEP amplitudes were stronger in threat trials than in no-threat trials. The (non-significant) effect on the P22 SEP was in the opposite direction, with smaller amplitudes in threat trials than in no-threat trials.

| | Condition | |
|---|---|---|
| | No Threat | Threat |
| P22 | 1.93 (7.46) | 1.52 (7.87) |
| N95 | -6.84 (8.15) | -7.84 (8.69) |
| P200 | 6.02 (8.80) | 6.67 (9.13) |

*Table 1. Mean SEP amplitudes and standard deviations of the three components (P22, N95 and P166) for the threat and no-threat condition.*

**2.4 Does somatosensory attending to the back mediate the effect of pain anticipation on movement initiation?**

To establish that SEP amplitudes mediate the effect of pain anticipation on RTs, it has to be shown that pain anticipation influences both RTs and SEP amplitudes, and that SEP amplitudes predict RTs (Baron & Kenny, 1986). The first two criteria were confirmed in the previous two sections, namely the significant effects of pain anticipation on both RTs and the N95 and P166 SEPs. To evaluate the third criterion, we tested whether RTs could be predicted by trial-by-trial variations in SEP amplitude. This revealed a significant effect of N95 amplitude on RTs, $t(3624) = 3.09$, $\beta = 0.04$, *CI 95%* = [0.02, 0.07], $p = 0.002$ (Figure 3). However, RTs were not significantly associated with P22 amplitude, $t(3609) = -0.31$, $\beta = -0.004$, *CI 95%* = [-0.03, 0.02], $p = 0.757$, or with P166 amplitude, $t(3626) = -0.27$, $\beta = -0.004$, *CI 95%* = [-0.03, 0.03], $p = 0.790$. Note that none of these effects interacted with pain anticipation (i.e., threat vs. no-threat), all $p \geq 0.360$.

Altogether, these results indicate that the N95, but not the P22 or P200, mediates the effect of pain anticipation on movement initiation. This was confirmed by a causal mediation analysis (Tingley, Yamamoto, Hirose, Keele, & Imai, 2014), which revealed not only a significant

direct effect of pain anticipation on RTs, β = 0.134, *CI 95%* = [0.084, 0.184], $p < 0.001$, but also a significant indirect effect via N95 amplitudes, β = 0.003, *CI 95%* = [0.001, 0.005], $p = 0.003$, indicating partial mediation (Figure 4).

[Figures 2, 3 and 4 about here]

## 3. Discussion

The aim of the current study was to investigate whether the expectancy of movement-related pain would enhance somatosensory attending to the back, resulting in slower initiation of movements involving the back muscles. Moreover, it was investigated whether attending to the back would mediate the effect of pain anticipation on movement initiation. In sum, our findings showed that (1) preparing a movement that was expected to be followed by pain on the back indeed enhanced somatosensory attending to the back, as reflected by different SEP components, (2) the expectation of pain during movement did not result in slower but rather in faster movement initiation, and (3) this effect of pain anticipation on movement initiation was partially mediated by enhanced somatosensory attending to the back. In the following, these three results will be discussed in more detail.

Support for the first hypothesis, that the anticipation of movement-related pain on the back would enhance somatosensory attending to the back, was found in the SEP results. Specifically, we found a negative component peaking at 95 ms (N95) of which the amplitude was significantly larger in threat trials than in no-threat trials. This effect resembles the results of Clauwaert et al. (2018), who used a similar design. More specifically, in that study, participants performed movements with the left and right hand, and one of these movements was conditioned with pain on the moving hand. Their results revealed that that the N120 SEP was

significantly larger when preparing the movement to be followed by pain. Nevertheless, compared with the study of Clauwaert et al. (2018), this negative component showed notable differences in timing (i.e. 95 instead of 120 ms after stimulus onset) and topography (vertex instead of lateralized, central topography). Procedural differences between both studies are likely to account for this. For example, in the current study, the same right arm movement was followed by either a painful or non-painful stimulus, whereas in the study of Clauwaert et al. (2018), movements with either the left or right hand were followed by a painful stimulus and movements with the other hand were followed by a non-painful control stimulus instead. Moreover, in the current study, SEPs were recorded to tactile stimuli presented to the lower back, whereas in the study by Clauwaert and colleagues (2018), SEPs were recorded to tactile stimuli that were presented to the left versus right hand.

Similar to the results of Clauwaert and colleagues (2018) the current results suggested an enhancement of somatosensory attending when threatened with pain. It must be noted though that the physiological meaning of the N95 and P166 amplitude are not completely known. Therefore, the possibility of them representing an inhibitory response rather than an excitatory one cannot be completely excluded. However, it has been shown that the magnitude of SEPs are typically enlarged when attention is focused at the somatosensory stimulus (Eimer & Forster, 2003; Franz, Nickel, Ritter, Miltner, & Weiss, 2015; Garcia-Larrea, Bastuji, & Mauguière, 1991; Zopf, Giabbiconi, Gruber, & Müller, 2004). Both the current study as the study of Clauwaert and colleagues (2018) reversed this logic and interpreted an increased amplitude as an indication of increased attending. This corresponds to several behavioral findings that show that tactile stimuli are processed faster or better at body parts under threat of pain (e.g. Vanden Bulcke, Van Damme, Durnez, & Crombez, 2013), thereby suggesting enhanced somatosensory attending rather than an inhibitory response. Alternatively, it can be argued that the faster movement during the threat trials are a sign of higher attention to the task, rather

than increased attending to the tactile stimulus. However, this is unlikely since the tactile stimuli are task-irrelevant and attending to them would not be beneficial for executing the task (i.e. performing the arm movement).

The second hypothesis stated that movements should be initiated more slowly when anticipating pain. In contrast, the results showed RTs were not slower but faster in threat trials compared with no-threat trials. This goes against the hypothesis that participants would hesitate more when initiating a movement that could be followed by pain, as previously demonstrated by Meulders and colleagues (Meulders & Vlaeyen, 2013; Meulders et al., 2011) and Neige and colleagues (Neige, Mavromatis, Gagné, Bouyer, & Mercier, 2018). It should be noted, though, that in these studies participants manipulated a joystick towards a certain direction, rather than performing an actual arm movement, and this procedural difference might have resulted in different effects. One potential explanation for the faster response times in threat trials compared to no-threat trials in the current study might be that the fearful anticipation of a painful stimulus activated a defensive response priming the motor system for escape from the threatening situation (Hagenaars, Oitzl, Roelofs, 2015; Krypotos, Effting, Kindt, & Beckers, 2015; Löw, Weyemar, & Hamm, 2015; Morrison et al., 2013; Perini et al., 2013). Even if it was technically not possible for participants in this study to escape from the threat, this speculation might provide an interesting avenue for future research. It could be particularly interesting to manipulate the possible functions of motor actions, such as escaping or controlling a painful stimulus. An alternative explanation for the faster initiation of pain-related relative pain-unrelated movements could be that participants tried to get the pain over with as soon as possible, thus reflecting a coping strategy ("let's get it over with"). This is in line with the study by Karos et al. (2017), who found that pain-conditioned movements using a robotic arm were performed faster than neutral movements. However, it should be noted that they examined movement velocity (the time it takes to perform the whole movement) rather than movement

initiation (the time it takes to initiate a movement), which are fundamentally different movement parameters.

Finally, the third hypothesis stated that somatosensory attending to the back would mediate the effect of pain anticipation on movement initiation. In line with this hypothesis, N95 amplitude predicted response speed, and was found to partially mediate the effect of pain anticipation on movement initiation. This suggests that expecting movement- related pain on the back increased somatosensory attending to the relevant effector (i.e. in this case the lower back; as measured by the N95 SEP), which then primes the motor system, and leads to faster responses. Interestingly, our results are in line with a recent study by Misra, Ofori, Chung, and Coombes (2017) who found that pain-related suppression of premotor beta oscillations was associated with faster reaction times, and speculated that the presentation of a pain-eliciting stimulus at the arm may have led to an increase in attention towards that arm prior to movement execution, facilitating motor responses. Alternatively, it could also be that SEPs were affected by motor preparation. Indeed, research has shown that motor preparation enhances processing of somatosensory stimuli (Eimer, Forster, Van Velzen, & Prabhu, 2005; Juravle & Deubel, 2009). Van Ede and colleagues (2015) explained these effects by stating that motor preparation enhances attending towards the body part about to perform the movement. The current study adds to this research by showing that the extent to which one attends to an action-relevant body part may predict how fast the movement will be executed. Interestingly, the fact that faster RTs were found shows that this mechanism plays a role whether or not escape is an option.

A number of other findings regarding the SEPs should be mentioned. We found a positive component peaking at 166 ms. This P166 was more pronounced in threat trials than in no-threat trials, which resembles the P200 effect observed by Clauwaert et al. (2018), and which may suggest a higher aroused state when anticipating the pain-related movement. We also identified

a very early positive component at 22 ms. It has been suggested that such early SEPs may originate from activity in the primary somatosensory cortex (SI), while later negative components around 100 ms are believed to represent activity in SII (Bradley, Joyce, & García-Larrea, 2016). Furthermore, a study by Asanuma and colleagues (2003) described gating of the early P40 SEP when preparing a limb movement. This finding is similar to studies showing that movements may suppress the processing of sensory information (i.e. sensory suppression; Juravle, Binsted, & Spence, 2017). Likewise, the early P22 in the current study may thus reflect suppression of the somatosensory stimulus while preparing an arm movement. Interestingly, in contrast to the later components, P22 amplitudes tended to be larger when participants prepared a movement associated with pain compared to when they prepared a movement not associated with pain, which might suggest reduced sensory suppression by movements that are expected to be followed by pain. However, this interpretation should be interpreted with caution, because the effect was only marginally significant.

The current study showed that expecting pain on the back following a rapid arm movement has an impact on both somatosensory attending to the back and motor execution. Moreover, somatosensory attending, as reflected by the N95, was found to be associated with movement latency, and partially mediated the effect of pain anticipation on movement initiation, making it a potential mechanism by which pain expectation modulates motor execution. An important avenue for future research is to investigate the interactions between pain, action, and attention in clinical populations such as those suffering from lower back pain, for which the presented paradigm might be particularly suitable, because muscle recruitment in the back during rapid arm movements is typically altered in lower back pain patients (MacDonald, Mosely, & Hodges, 2009; Park, Tsao, Cresswell, & Hodges, 2014).

## 4. Experimental Procedure

### 4.1 Participants

Participants (N=36) were recruited through an online recruiting system and took part in exchange for a monetary reward. Based on self-report, all participants confirmed on arrival to be free from back pain, systemic diseases or neurological disorders. However, 3 participants did not complete the full experiment because they fainted[1] and were removed from the analyses. Of the remaining 33 participants, 14 were male and the mean age was 21.98 (SD= 2.38, range= 18-28). All participants were right-handed, except for a single participant who reported to be ambidextrous. This was confirmed by his/her score on the Edinburgh Handedness Inventory scale (Oldfield, 1971). However, this participant was retained because (s)he reported to be right-dominant for the gross motor functions measured in the current study.

No information was provided about the goal of the study prior to the start of the experiment. Instead, participants were informed that the experiment consisted of a simple behavioral task in which harmless sensory stimuli would be administered. To prevent that only participants without fear of pain would sign up, the use of painful stimuli in the study was not explicitly mentioned during recruitment. However, the painful nature of the stimuli was disclosed to participants as soon as they arrived in the lab. Participants were told that they were free to terminate the experiment at any time without any consequence. All participants agreed to continue with the experiment and signed an informed consent. The study protocol was approved by the local ethical committee (file number 2016/60) and was performed according to the ethical standards laid down in the declaration of Helsinki.

[1] We have encountered the same phenomenon in other studies using similar movements in different locations and set-ups. Therefore, this reaction is presumably related to the nature of the movements participants had to execute. All participants recovered well shortly after fainting, and did not continue with the experiment.

## 4.2 Materials

**4.2.1 Vibrotactile stimuli.** A resonant-type tactor (C-2 TACTOR, Engineering Acoustics, Inc., Florida) was used to administer vibrotactile stimuli (200 ms) to the low back at the L3 spinous process level. The tactor was attached directly to the skin surface by means of a double-sided tape ring. The amplitude and frequency (300 Hz) were controlled by a custom-made software program. To prevent interference from environmental noise and the tactor, participants were asked to wear earplugs during the experiment.

**4.2.2 Electrocutaneous stimuli.** 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. These electrodes had a diameter of 1 cm and were placed directly underneath the tactor.

**4.2.3 EEG.** EEG was recorded using a Biosemi ActiveTwo recording system at a sampling rate of 2,048 Hz from 64 active electrodes, placed according to the international 10/20 setting. EEG signals were referenced online to the active Common Mode Sense and passive Driven Right Leg (CMS-DRL) electrodes. Bipolar electrodes were placed respectively above and below the left eye and next to the outer left and right canthi to measure eye movements. Electrode offsets were kept between -50 and 50 µV at all electrodes.

**4.2.4 Experiment.** The experiment was programmed in the C-language using the Tscope 5 library (Stevens, Lammertyn, Verbruggen, & Vandierendonck, 2006).

## 4.3 Design and procedure

**4.3.1 Staircase procedure.** The experiment started by individually determining the intensity of the electrocutaneous stimulus (ES) by means of a staircase procedure previously used in our lab (Clauwaert et al., 2018; Notebaert, Crombez, Van Damme, De Houwer, Theeuwes, 2010). Participants were first presented with an ES of low amplitude (0.5 mA). Next, the same stimulus was presented again to rule-out that stimulus evaluation was influenced by the initial surprise of the stimulation. Next, participants were asked to press the space bar to increase the intensity of the stimulus. Each time the bar was pressed, the ES amplitude was elevated by 0.5 mA. Participants were motivated to choose a stimulus intensity that was as unpleasant as possible but that they were still willing to tolerate during the experiment. Once a higher stimulus amplitude was chosen, participants could not go back to a lower amplitude.

**4.3.2 Movement training phase.** Following the staircase procedure, participants completed a practice phase in which they practiced the arm movement task. In this practice phase, participants were seated in a chair with a sensor box taped to its side and had to respond as quickly as possible to the visual response cue (i.e. the words “forward” or “backward” in Dutch) by performing either a forwards or backwards arm movement. The box was located at the height of the distal phalanx of the right index finger. To respond, participants had to make a forward arm movement with a stretched arm in an angle of approximately 90°, or a backward arm movement in an angle of approximately 30°, and back to the sensor box. These rapid arm movements were chosen because they disturb the deep trunk posture, eliciting an anticipatory response of the lower back muscles to restore balance (MacDonald et al., 2009; Park et al., 2014).

Participants were motivated to perform the complete movement as fast and accurate as possible. The backwards movement was included to make the direction of the movement unpredictable. In the 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 of the movement, as well as whether participants maintained a stretched arm during movement execution. If needed, the practice phase was repeated until the movement was executed as requested. Note that the current study also served to pilot a subsequent patient study focusing on electromyography (EMG) of the lower back. However, the EMG data will not be reported here.

**4.3.3 Experimental phase.** See Figure 1. In the experimental phase, the movement cue was preceded by a threat cue, indicating whether the response would be followed by a painful or non-painful stimulus to the back (threat versus no-threat trials). The experiment consisted of 7 blocks, each comprising 40 trials. The first block served as a practice block, in which participants learned the association between the threat cues (a filled or empty lightning bolt) and the somatosensory stimuli. The association between this cue and the corresponding type of stimulus was explained by the experimenter before the start of the experiment and was conditioned during the practice phase. This way, participants learned about the association both through experience and through instruction, which optimizes fear conditioning (Field & Storksen-Coulson, 2007). In order to limit the amount of arm movement, and to prevent arm fatigue, half of the trials in each block were rest trials in which no movement was required, and the other half were movement trials in which the forward and backward movement had to be executed 60 times each. Importantly, half of the movement trials were threat trials and the other half were no-threat trials. Participants were able to take a short break at the end of each block.

As shown in Figure 1, trials started with the presentation of a white fixation cross (500 ms) on a black display 1 m in front of the subject at eye-height. Next, a filled or empty lightning bolt (i.e., threat cue) indicated whether the subsequent forward/backward movement would be followed (in 33% of the trials) by a painful electrocutaneous stimulus (filled bolt) or by a non-painful vibrotactile stimulus (empty bolt) on the lower back (spinous process L3) after releasing

the optical sensor. The presentation time of the threat cue varied between 2000 and 3000 ms. At a random moment between the appearance and disappearance of this cue, a vibrotactile stimulus was presented in 100% of the trials to evoke an SEP. The SEP-evoking stimulus was administered between 1000 and 1500 ms after the onset of the lightning cue. The lightning cue was followed by the response cue, indicating whether a forward movement (25%), a backward movement (25%) or no movement (50%) had to be executed. The “FORWARD” cue (“VOOR” in Dutch) signaled that the forward arm movement had to be executed as quickly as possible. The “BACKWARD” cue (“ACHTER” in Dutch) signaled that the backward arm movement had to be executed as quickly as possible. Finally, the “STOP” cue signaled that participants had to refrain from moving. Reaction times were defined as the interval between the onset of the response cue and the moment the sensor box was released.

To ensure that participants executed the movements correctly, they were monitored by the experimenters through a webcam. At the end of each trial, a timeline counting down from 12 seconds was shown on the screen. In this time frame, participants could rest, position their fingertip back on the sensor box, and prepare for the next trial. Furthermore, the computer screen presented instructions asking participants to relax the abdominal muscles and to maintain a normal and relaxed breathing pattern in order to restore core muscle activation to baseline. After the 12 second interval, a black screen was shown for 500 ms, after which the next trial started.

**4.4 Self-report instruments**

After the experiment, participants were asked to rate, on 11-point Likert scales (anchored 0 = not at all and 10 = very strongly), their experimental pain experience ('How painful did you find the electrocutaneous stimulus?' and 'How unpleasant did you find the

electrocutaneous stimulus?'), as well as their expectation and fear of a painful stimulus when seeing the different cues ('To what extent did you expect that the full/empty lightning bolt or stop cue would be followed by a painful stimulus?' and 'To what extent were you afraid that the full/empty lightning bolt or stop cue would be followed by a painful stimulus?').

### 4.5 Data preprocessing and analyses

**4.5.1 EEG.** EEG data were preprocessed using Brainvision Analyzer 2.1 (Brain Products GmbH, Munich, Germany). EEG signals were referenced offline to the linked left and right mastoids. A high pass filter of 1 Hz, a low pass filter of 30 Hz, and a notch filter of 50 Hz were applied. SEPs were time-locked to the onset of the vibrotactile stimulation and segments were generated from -200 ms to 500 ms. Prior to averaging, artifacts due to eye blinks were automatically corrected by means of the standard Gratton et al. (1983) algorithm. Next, automatic artifact rejection was applied. SEP components of interest were localized using the collapsed localizer method described by Luck and Gaspelin (2017). That is, we first averaged the EEG waveforms across the two conditions (i.e., pain trials and no pain trials) and then visually inspected this waveform on the basis of similar previous work (Clauwaert et al., 2018; Lenoir, Huang, Vandermeeren, Hatem, & Mouraux, 2017). This revealed three clear peaks: a positive peak at 22 ms (P22), a negative peak at 95 ms (N95), and a positive peak at 166 ms (P166). This was confirmed by a global field power calculation across both conditions (Murray, Brunet, & Michel, 2008).

All three components had a central topography that was maximal around the Cz electrode (Figure 2). To quantify the SEP components, we calculated the mean area amplitude on the FCz and Cz electrodes for the positive components and on the Fz, FCz, Cz, F1, F2, F3, F4,

FC1, FC2, FC3, FC4, C1, C2, C3, C4 electrodes for the negative component[2]. Based on previous research (Clauwaert et al., 2018), we calculated the P22 between 15 and 29 ms, the N95 between 70 and 120 ms, and the P166 between 126 and 206 ms, all centered on the peak of the collapsed localizer (Luck & Gaspelin, 2017). Note that mean area amplitudes were used because these are known to provide an unbiased amplitude measure (Luck, 2014). To investigate whether somatosensory attention influenced response speed, we extracted the SEP data on a trial-by-trial basis using the Solutions package in Brainvision.

**4.5.2 Statistical Analyses.** To test our hypotheses, we fitted linear mixed effects models (LMMs), using the lme4 package in R (Bates, Mächler, Bolker, & Walker, 2015). First, to investigate whether pain anticipation influenced movement initiation, we compared RTs on threat trials with RTs on no-threat trials. Second, to investigate whether pain anticipation affected somatosensory attending to the back, we compared SEP amplitudes on threat trials with SEP amplitudes on no-threat trials. Third, to investigate whether somatosensory attending to the back mediated the effect of pain anticipation on movement initiation, we followed the criteria by Baron and Kenny (1986). Specifically, the mediation effect was only tested if pain anticipation affected RTs as well as SEP amplitudes, and if trial-by-trial variations in SEP amplitude predicted RTs. The mediation effect was tested with a causal mediation analysis, using the mediation package in R (Tingley, Yamamoto, Hirose, Keele, & Imai, 2014). Backward selection was used to determine the random effects structure of the LMMs (Matuschek, Kliegl, Vasishth, Baayen, & Bates, 2017). To maximize model similarity, this structure was kept the same for all three SEP components. That is, we used the random effects structure of the model with the least complex structure, as determined through backwards selection. This resulted in a

[2] Note that it could be argued that more electrodes were included in the N95 analysis compared to the P22 and P166 components, and that this caused the larger effects for the N95 component compared with the other two components. However, similar results were obtained if only the FCz and Cz electrodes were used in the N95 analyses, ruling out this explanation.

random intercept together with random slope for condition. However, note that none of the results changed if the random effects structure was instead determined separately for each component. The beta coefficients reported in the paper are standardized beta coefficients. P-values were calculated using Satterthwaite approximations (Luke, 2017).

**4.5.3 Trial Exclusion**. To maximize statistical power, exclusion criteria were based only on the data being considered. However, the results did not change if we instead excluded all trials below from all analyses. To test the effect of pain anticipation on RTs, we excluded STOP trials (50%), trials without EEG data (0.10%), trials in which the movement was initiated before the response cue was presented (0.33%), trials with a RT faster than 100 ms (0.00%), trials with a RT slower than 2000 ms (1.32%), and trials with a RT exceeding the participant's mean by more than 3 standard deviations (0.90%). To test the effect of pain anticipation on SEP amplitude, we excluded trials without EEG data, trials with movement initiation before SEP stimulation (0.36%), and trials with EEG artefacts (5.20%). To test the effect of SEP amplitude on RTs, we excluded all above trials.

## Conflict of interest

The authors declare no conflict of interest.

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## Figure Legends

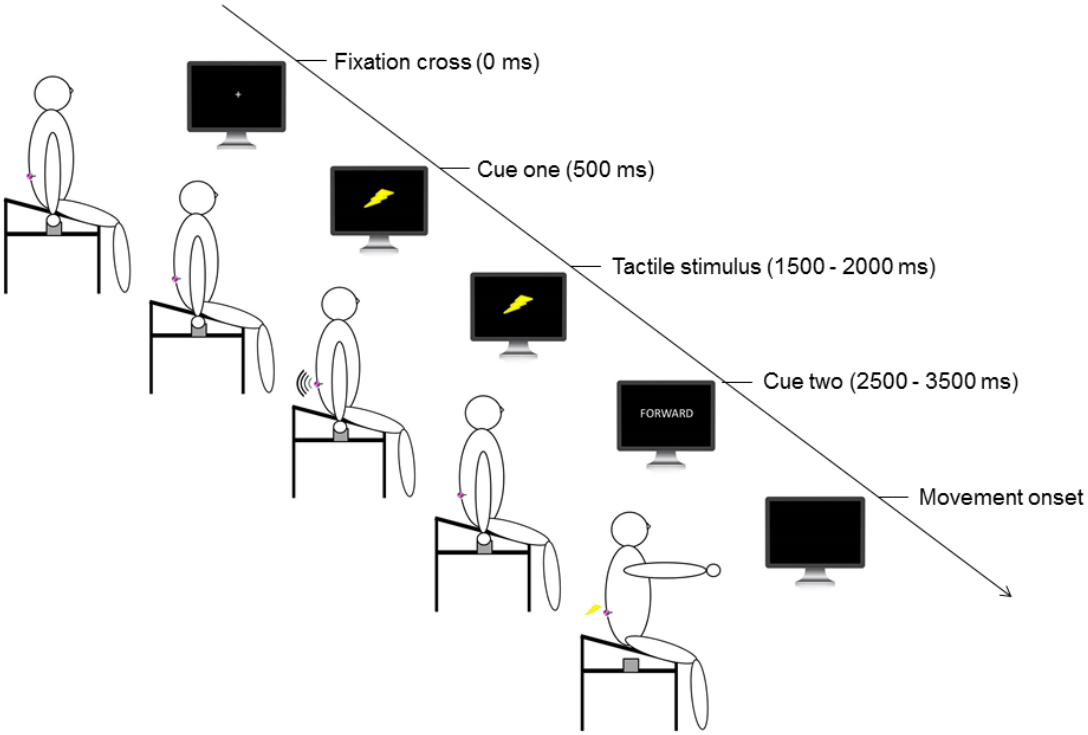
Figure 1. Overview of an experimental trial. At the start of the trial, participants were first presented a fixation cross, followed by a threat cue indicating whether the response would be combined with a painful (full bolt) or non-painful (empty bolt) stimulus to the back. At a random moment between the appearance and disappearance of this cue, a vibrotactile stimulus was presented to the lower back to evoke an SEP. The lightning cue was followed by the response cue, indicating whether a forward movement, a backward movement or no movement had to be executed.

Figure 2. Waveform as presented at the Cz electrode for the grand averages of both conditions (pain trials (red line) versus no pain trials (black line)), with the current source densities at each peak (P22, N95, P166). The Cz electrode was selected for the figure as this is the electrode that is common for the analyses of all three components

Figure 3. Effect of N95 amplitude on reaction times (RT) for both conditions (pain trials vs no pain trials). The interaction between amplitude and condition was not significant. High amplitudes of the negative component are related to faster RTs in both conditions. The dotted red line shows the 95% confidence interval.

Figure 4. Mediation model with standardized beta values. * $p<0.1$, ** $p<0.001$. This figure illustrates that pain anticipation (i.e., threat vs. no threat) influences N95 SEP amplitude (i.e., somatosensory attention) and that N95 SEP amplitude influences movement initiation. As such, it shows that N95 SEP amplitude mediates the influence of pain anticipation on movement initiation. Given that the effect of pain anticipation on movement initiation remained significant

after accounting for the effect of N95 SEP amplitudes, these results are indicative of a partial mediation.

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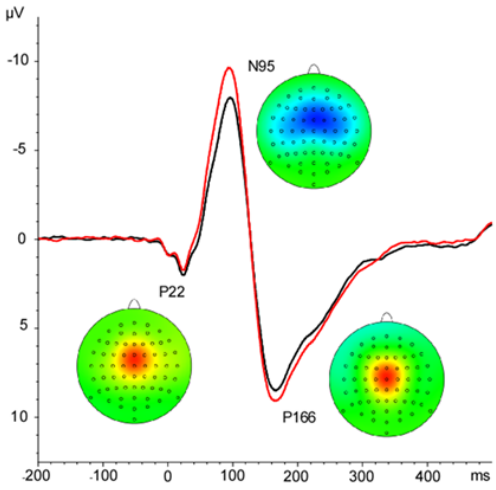


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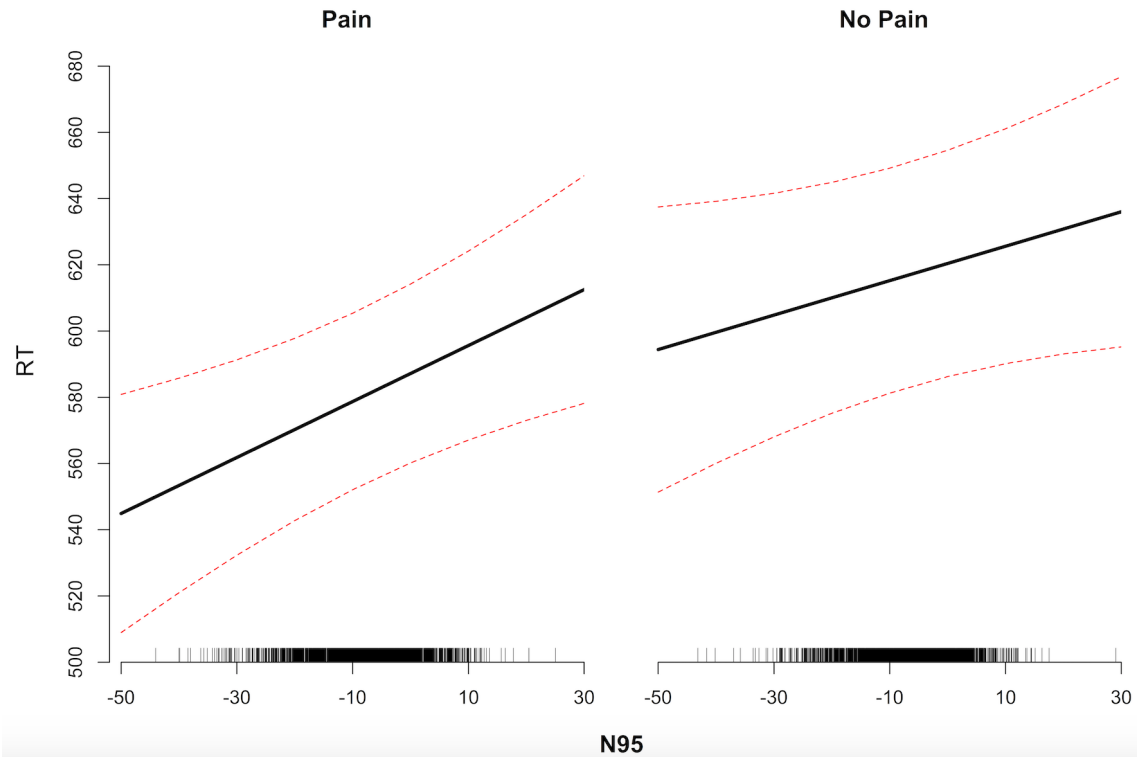
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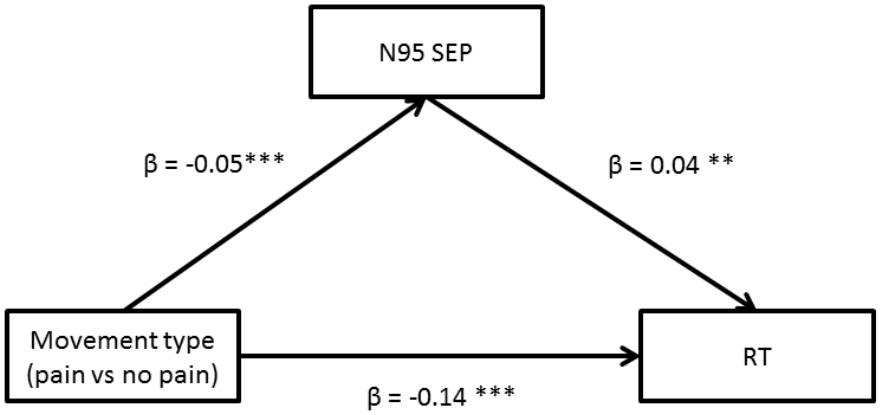


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