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Pain perception in the self and observation of others: An ERP investigation

Jing Meng, Todd Jackson *, Hong Chen *, Li Hu, Zhou Yang, Yanhua Su, Xiting Huang

Key Laboratory of Cognition & Personality, Faculty of Psychological Science, Southwest University, Chongqing 400715, China

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ABSTRACT

The nature of interactions between observing pain in others (other-pain) and subjective pain perception (self-pain) has been debated. To test whether other-pain and self-pain primes increase or decrease responsiveness to complementary self-pain or other-pain targets, two ERP studies were conducted. In Study 1, twenty participants (10 women, 10 men) were exposed to pictures depicting other-pain or other non-painful situations, followed by self-pain or non-nociceptive heat stimulation delivered to the forearm. Significant visual prime × sensory target interactions indicated that compared to other non-painful primes, other-pain visual primes predicted faster reaction times (RTs) and smaller P2 amplitudes in response to self-pain stimuli while responses to self-heat stimuli were not affected by priming images. However, effects of other-pain primes on elevations in intensity ratings were not specific to self-pain and extended to self-heat targets. In Study 2, self-pain and self-heat stimuli were applied to the same participants followed by other-pain and other non-painful visual targets. Similar to the pattern for Study 1, sensory prime × visual target interactions indicated that compared to self-heat primes, self-pain sensory primes predicted marginally faster RTs and smaller P3 amplitudes in response to other-pain targets while responses to other non-painful targets were unaffected by sensory priming stimuli. Again, self-pain primes predicted higher intensity ratings for both target types compared to self-heat primes. Together, findings supported the shared-representation model of pain empathy more strongly than the threat value of pain hypothesis.

Introduction

Brain imaging studies indicate that subjectively-experienced pain (self-pain) and observations of pain in others (other-pain) both elicit activation of the "pain matrix" (e.g., Avenanti et al., 2010; Botvinick et al., 2005; Cheng et al., 2007; Gu and Han, 2007; Jackson et al., 2005; Lamm et al., 2010), overlapping cortical structures that include affective areas (e.g., Lamm et al., 2007; Singer et al., 2004, 2006) and sensory areas (e.g., Avenanti et al., 2006). However, it is less clear whether other-pain and self-pain experiences, respectively, influence complementary behavior and brain responses to subsequent self-pain and other-pain in precisely the same manner. To elucidate this issue, we examined effects of (1) other-pain primes on self-pain targets and (2) self-pain primes on other-pain targets in two ERP studies.

Effects of other-pain on self-pain

According to the shared-representation model of pain empathy, witnessing pain in another person activates affective and somatosensory pain representations in the observer that reflect a relatively automatic capacity to understand experiences of others in pain (Decety and Jackson, 2004; Jackson et al., 2005). Hence, from this perspective, perception of other-pain should facilitate processing of subsequent subjective pain experiences, due to the activation of similar brain areas and similar affective and autonomic responses (Coll et al., 2012). Supporting this account, research in which participants experienced self-pain stimuli after observing images of noxious or non-noxious stimulation applied to hands and feet indicates that exposure to other-pain corresponds to faster reaction times (RTs) in evaluating self-pain experiences (Meng et al., 2012b) and higher intensity ratings (Godinho et al., 2006, 2012; Meng et al., 2012b). Vachon-Presseau et al. (2011) indicated, further, that priming effects of other-pain on unpleasantness ratings are stronger in response to images depicting pain sensory information (i.e., noxious stimuli applied to extremities) than emotional-communicative depictions (i.e., painful facial expressions).

In early ERP work, Valeriani et al. (2008) exposed participants to noxious laser stimulation while they observed video clips of a model receiving noxious or non-noxious stimuli. Self-pain stimuli accompanied by other-pain video clips resulted in lower N1/P1 amplitudes than did self-pain stimuli accompanied by other non-painful depictions, a pattern that reflected early cortical processing of the ascending nociceptive input. More recently, Valentini et al. (2012) recorded cortical responses elicited by self-pain stimulation among respondents who viewed video clips of noxious or non-noxious stimuli applied to a model's hand. They found an event-related desynchronization in the beta band (β-ERD) to self-pain stimulation accompanied by other-pain clips but

* Corresponding authors at: Key Laboratory of Cognition and Personality, Southwest University, Beibei, Chongqing 400715, China.
E-mail addresses: toddjackson@hotmail.com (T. Jackson), chenhg@swu.edu.cn (H. Chen).

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not non-painful clips, arguing that this effect reflected reduced motor preparatory responding and increased attention towards self-pain stimulation.

Together, these findings suggest that other-pain facilitates behavioral and brain responses to self-pain. Furthermore, select research has found that the effects of other-pain (rather than non-specific negative stimuli) increase responsiveness to processing self-pain experiences (de Wied and Verbaten, 2001; Godinho et al., 2006). Nevertheless, some research has found that pain-related primes affect responses to both self-pain and non-nociceptive (i.e., heat) somatosensory stimuli (Kirwilliam and Derbyshire, 2008), arguably because the pattern of spatial activation to non-nociceptive somatosensory stimuli is similar to that activated for nociceptive stimulation.

Effects of self-pain on other-pain

In contrast to evidence for facilitative effects of other-pain on self-pain responses, conflicting predictions have been generated to explain how self-pain affects other-pain (Coll et al., 2012). On one hand, in line with facilitative effects of other-pain on self-pain perception, tenets of the shared-representation model (Decety and Jackson, 2004; Jackson et al., 2005) imply that self-pain should increase responsiveness to other-pain by activating similar representations and automatic experiences. Thus, relative to non-nociceptive sensory stimulation, self-pain experiences should enhance responsiveness to other-pain.

Conversely, the "threat value of pain hypothesis" suggests that vicariously-instigated activation of the pain matrix is not specific to sensory qualities of pain, and is associated, instead, with more general survival mechanisms such as withdrawal and avoidance upon exposure to danger and threat (Decety, 2010; Decety et al., 2012; Ibáñez et al., 2011; Yamada and Decety, 2009). From this perspective, other-pain can serve as a threatening signal to be escaped or avoided. Of note to the current focus, self-pain should exacerbate concerns with other-pain; ultimately, withdrawal from/avoidance of such threats should result in reduced responsiveness to other-pain (Coll et al., 2012).

Coll et al. (2012) have provided partial support for these contentions. Their participants were exposed to painful and non-painful heat stimuli and asked to judge pain intensities in video clips featuring facial expressions of male and female models. Compared to heat stimuli, self-pain predicted lower intensity ratings for highly painful expressions of female models consistent with the threat value hypothesis. However, coinciding with the shared-representation model, self-pain also predicted higher intensity ratings for pain expressions of males. The results highlighted how "other" characteristics such as gender moderate effects of self-pain. Meng et al. (2012b) also obtained results in line with the shared representation model, not the threat value of pain hypothesis. In that study, RTs were faster in rating sensory other-pain images following self-pain primes compared to non-painful self-heat primes, while RTs to other non-painful images were not modulated by sensory prime types.

To the best of our knowledge, effects of self-pain and non-painful sensory primes on ERP responses to other-pain and other non-painful depictions have not been assessed. Such research can help to clarify whether the shared representation model or the threat value of pain hypothesis best explains how self-pain affects responses to other-pain and may provide a more nuanced account of effects than can be garnered from behavior responses alone.

Based on the preceding review, two studies evaluated competing predictions about the impact of other-pain and self-pain on complementary self-pain and other-pain responses. In Study 1, we assessed effects of gender-neutral depictions of sensory other-pain primes on behavior (RTs, pain intensity rating) and ERP (amplitudes) responses corresponding to self-pain experiences. In Study 2, effects of self-pain primes on these responses to sensory other-pain were evaluated. Non-painful self-heat sensory stimuli and other non-painful visual images were incorporated within experimental designs to evaluate the specificity of pain-related primes on responses to pain-related targets.

The "threat value of pain" hypothesis represents a plausible alternative supported under certain conditions (Coll et al., 2012), yet a preponderance of behavioral evidence (e.g., de Wied and Verbaten, 2001; Godinho et al., 2006, 2012; Meng et al., 2012b), is aligned with the shared-representation view suggesting that other-pain and, more implicitly, self-pain elicits pain representations that increase responsiveness to perceptually-congruent (i.e., pain-related) experiences compared to non-painful experiences. Therefore, we expected that pain-related primes would elicit faster RTs in judging pain intensities and higher pain intensity ratings for pain-congruent targets than non-pain-related primes while responses to non-pain-related targets would not differ as a result of prime types. Despite the lack of directly-related ERP evidence, based on research suggesting that decreased amplitudes for late latency components (e.g., P300 for picture targets, N400 for linguistic targets) are indices of congruency effects (e.g., Bartholow et al., 2009; Chwilla et al., 1995; Friedman et al., 2001; Goerlich et al., 2012; Ito et al., 1998), we hypothesized that pain primes would be related to decreased late ERP amplitudes for complementary pain-congruent targets relative to pain-incongruent targets.

Methods

Participants

Twenty pain-free emerging adults (10 men, 10 women) from Southwest University (SWU), Chongqing, China, participated as paid volunteers. All participants were right-handed, aged 18–23 years ($M = 22.6$ years, $SD = 1.56$ years), and had normal/corrected-to-normal vision. None had been diagnosed previously with a medical, neurological or psychiatric disorder.

Apparatus

Self-pain and self-heat sensory stimulation

A contact heat-evoked potential stimulator (CHEPS; PATHWAY sensory evaluation system; Medoc Ltd., Ramat Yishai, Israel) with a round thermode contacting a cutaneous area of 572.5 mm^2 (27 mm diameter) delivered pain and non-nociceptive heat stimulation. The thermode was composed of a heating thermofoil (Minco Products, Inc., Minneapolis, MN), covered with a 25-μm layer of thermo-conductive plastic (Kapton, thermal conductivity at 23 °C of 0.1 to 0.35 W/mK). The thermofoil permits a heating rate of up to 70 °C/s, and the Peltier device allows a cooling rate of up to 40 °C/s.

The baseline temperature was 40 °C. Peak intensities of stimulation applied to the proximal volar left forearm were 43 °C and 45 °C for heat stimuli, and 50 °C and 51 °C for pain stimuli based on published accounts (Greffrath et al., 2007; Meng et al., 2012b) and pilot testing. Each heat/pain pulse was 432 ± 2 milliseconds (ms) in duration (rise time, about 157 ms; return to baseline time, about 275 ms). Heating and cooling rates of each heat/pain pulse were as follows: 51 °C: heating rate = 70 °C/s, cooling rate = 40 °C/s; 50 °C: heating rate = 63.7 °C/s, cooling rate = 38.9 °C/s; 45 °C: heating rate = 31.8 °C/s, cooling rate = 18.2 °C/s; 43 °C: heating rate = 19.1 °C/s, cooling rate = 10.9 °C/s.

Other-pain and other non-painful visual images

Sixty digital color pictures depicting a model's hand, forearm, or foot in painful or non-painful situations (30 pictures each) were taken from other published research (Meng et al., 2012a,b). All depictions were of events that can occur in everyday life (e.g., hand cut by a knife). Other non-painful pictures corresponded to those shown in "other-pain" depictions without a nociceptive component (e.g., hand using a knife to cut vegetables). Luminance, contrast, and color were matched between other-pain and other non-painful pictures, as illustrated in

Fig. 1. Each picture had dimensions of 9 × 6.76 cm (width × height) and 100 pixels per inch. Participants were seated at a viewing distance of 100 cm from the computer screen, sub-tending a visual angle of 5.15° × 3.87°.

EEG apparatus and recording

Electroencephalography (EEG) data were recorded from 64 scalp sites using tin electrodes mounted in an elastic cap (Brain Products, Munich, Germany). The electrode at the right mastoid was used as a reference while the electrode on the medial-frontal aspect was used as a ground electrode. Vertical electrooculograms (EOGs) were recorded supra- and infra-orbitally at the left eye. Horizontal EOGs were recorded at the left versus right orbital rim. EEG and EOG activity was amplified with a DC~100 Hz band-pass and continuously sampled at 500 Hz. All electrode impedances were maintained below 5 kΩ.

Experimental procedure

The research was approved by the SWU human research ethics committee. Sixty undergraduate students responded to a request for volunteers for a pain perception study advertised on the campus electronic bulletin board system. Twenty who reported no pain, medical, and psychiatric problems were randomly selected and randomly assigned into two subgroups. Specifically, the presentation order of the studies was randomized so that one subgroup (n = 10) completed the picture priming study (Study 1) first while the other subgroup (n = 10) completed the sensory priming study (Study 2) first. For each subgroup, there was a two week lag between studies. After signing an informed consent form and prior to ERP recordings, participants engaged in a training session to become familiar with experimental procedures in a quiet room with an ambient temperature of 22 °C. The procedures for Studies 1 and 2 are illustrated in the left and right columns, respectively, of Fig. 2.

Study 1

Prior to the study, participants were told that trials would involve presentations of painful or non-painful pictures on the computer screen followed by a brief pain or heat stimulus delivered at random to the forearm. Thus, responses were assessed in a 2 (visual prime) × 2 (sensory target) design that comprised (1) other-pain prime/self-pain target, (2) other non-painful prime/self-pain target, (3) other-pain prime/self-heat target and (4) other non-painful prime/self-heat target conditions.

In each trial, a fixation cross was presented on a black screen for 200 ms. Next, a visual image prime was presented onscreen for 1000 ms during which participants were instructed to observe the picture carefully but not respond. Subsequently, a blank screen was presented for 500–1000 ms before delivery of a sensory target stimulus. Because preparation and movements associated with behavior can confound EEG data, participants were not permitted to respond immediately after target stimulus onsets. Instead, sensory targets were followed by a 1000 ms blank screen, after which a 9-point pain intensity scale ("1 = no sensation", "4 = pain threshold", "9 = unbearable pain") appeared. Participants then provided a rating, as quickly and accurately as possible, via right-handed key-presses on a square keyboard pad with a 3 × 3 numeric display. The scale remained onscreen until a response had been made, or for a 4 s maximum. Hence, the interval between trials was 6–7 s and the interval between presentations of sensory stimuli was over 10 s.

Study 2

Procedures were identical except that sensory stimuli (self-pain and self-heat) were used as primes followed by other-pain and other non-painful visual image targets. The study featured a 2 (sensory prime) × 2 (visual target) design comprising (1) self-pain prime/other-pain target, (2) self-heat prime/other-pain target, (3) self-pain prime/other non-painful target and (4) self-heat prime/other non-painful target conditions.

Both studies included four blocks of 60 trials each. Trial sequences were presented pseudo-randomly so that self-pain stimuli never occurred on three consecutive trials. There was a 15 minute break between blocks to control for possible effects of desensitization. The thermode was moved at random to an adjacent area within an approximate area of 6 × 8 cm between blocks.

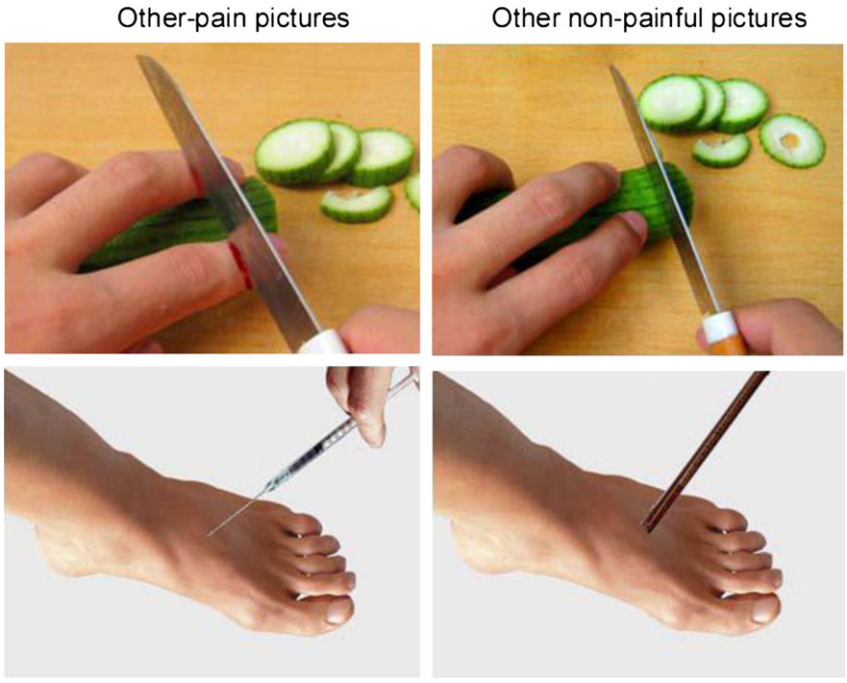

Fig. 1. Examples of other-pain (left panel) and other non-painful pictures (right panel).

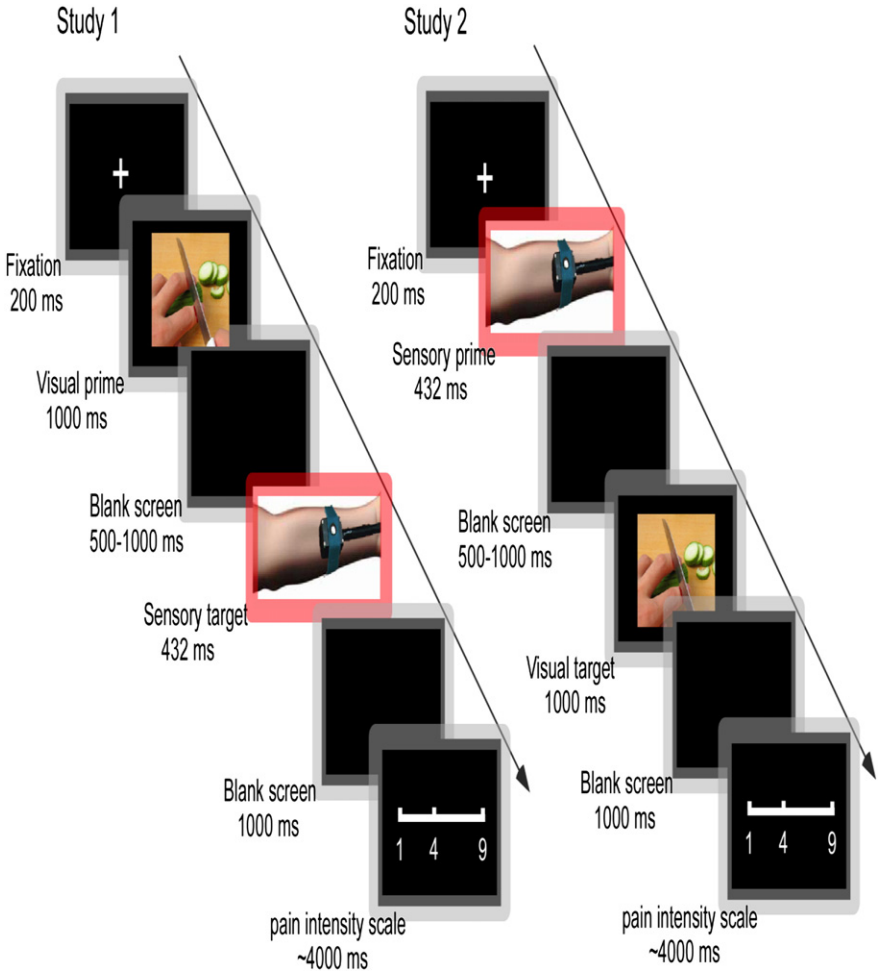

Fig. 2. Flowchart describing experimental designs. Left column: Procedure of Study 1. Right column: Procedure of Study 2. Panels with red frame represent applications with self-pain or self-heat sensory stimulation from the thermode.

Data analysis

Behavioral data

RTs and pain intensity ratings were calculated in each condition for each study. Two within-participants factors – visual image type (other-pain versus other non-painful) and sensory stimulus type (self-pain versus self-heat) – were included in two-way repeated-measures analyses of variance (ANOVA).

EEG data

EEG data were pre-processed and analyzed using Matlab 7.0 (MathWorks, US) and EEGLAB toolbox (Delorme and Makeig, 2004). EEG signals passed through an off-line 0.01–30 Hz band-pass filter. Time windows of 200 ms before and 1100 ms after onset of target stimulus segments were extracted from EEG, and the whole epoch was baseline-corrected by the 200 ms time interval prior to target onset. Epoched EEGs were inspected, and trials contaminated by gross movements were removed. EOG artifacts were corrected using an independent component analysis (ICA) algorithm (Jung et al., 2001). Epochs with amplitude values exceeding ± 60 μV at any electrode were excluded from the average. These epochs constituted 5 ± 1.3% of the total number of epochs.

For ERP data, based on a preliminary point-by-point two-way repeated-measure ANOVA (Boly et al., 2011) and voltage scalp maps, the following electrodes were included for further analysis: F4, FC4, C4, CP4, and P4 (five right sites); Fz, FCz, Cz, CPz, and Pz (five midline sites); F3, FC3, C3, CP3, and P3 (five left sites). Mean amplitudes were obtained from each grand-averaged peak. A 2 (visual image type) × 2 (sensory stimulus type) × 3 (region — left, midline, right) × 5 (electrode) repeated measures ANOVA was performed for each component. Degrees of freedom for F-ratios were corrected according to the Greenhouse–Geisser method. Statistical differences were considered significant at $p<0.05$; post-hoc comparisons were Bonferroni-corrected at $p<0.05$.

Results

Study 1

Behavioral data

Mean RTs, pain intensity ratings, and ERP amplitudes for sensory targets in all conditions are summarized in Table 1.

In the ANOVA for RTs, the main effect for visual prime [$F(1,19)=6.28$, $p=0.021$, $\eta^2=0.248$] indicated that participants were faster in rating target stimuli that followed other-pain primes (1161.96 ± 71.97 ms) than other non-painful primes (1191.37 ± 70.60 ms). For sensory target [$F(1,19)=16.56$, $p=0.001$, $\eta^2=0.466$], RTs were faster in rating pain intensities of self-pain targets (1047.13 ± 72.45 ms) than self-heat targets (1306.20 ± 82.91 ms). Notably, main effects were qualified by a visual prime × sensory target interaction [$F(1,19)=5.00$, $p=0.037$, $\eta^2=0.209$]. Simple effects analyses indicated that RTs in rating self-pain targets were significantly shorter if they followed other-pain primes rather than other non-painful primes [$F(1,19)=17.88$, $p<0.001$, $\eta^2=0.485$]. In contrast, RTs for self-heat targets did not differ as a function of visual primes [$F(1,19)=0.233$, $p=0.635$, $\eta^2=0.012$].

Table 1
Descriptive statistics for behavioral and ERP data in Study 1.

Condition	RT (ms)	Pain intensity	N2 window (μV)			P2 window (μV)		
			Right	Middle	Left	Right	Middle	Left
OP/SP	1011.35 (70.43)	6.10 (0.23)	−2.98 (0.71)	−2.41 (0.80)	−2.09 (0.72)	9.54 (1.37)	11.11 (1.34)	9.52 (1.16)
ON/SP	1082.91 (75.37)	5.77 (0.23)	−2.89 (0.89)	−1.96 (0.96)	−1.77 (0.91)	11.49 (1.29)	13.85 (1.42)	11.16 (1.36)
OP/SH	1312.58 (88.58)	3.10 (0.21)	0.91 (0.63)	1.56 (0.67)	0.93 (0.49)	3.61 (0.81)	4.60 (1.00)	3.49 (0.88)
ON/SH	1299.83 (79.06)	2.85 (0.19)	−0.20 (0.65)	0.90 (0.95)	0.70 (0.74)	1.80 (0.79)	3.91 (0.92)	2.63 (0.79)

OP/SP, other-pain prime/self-pain target; ON/SP, other non-painful prime/self-pain target; OP/SH, other-pain prime/self-heat target; ON/SH, other non-painful prime/self-heat target. Means and (SD) are shown.

For pain intensity, the main effect for visual prime [$F(1,19)=28.65$, $p<0.001$, $\eta^2=0.601$], indicated that target stimuli that followed other-pain primes were judged to be more painful (4.60 ± 0.19) than those that followed other non-painful primes (4.31 ± 0.19). For the main effect of sensory target [$F(1,19)=253.64$, $p<0.001$, $\eta^2=0.930$], average pain intensities were higher for self-pain targets (5.93 ± 0.23)

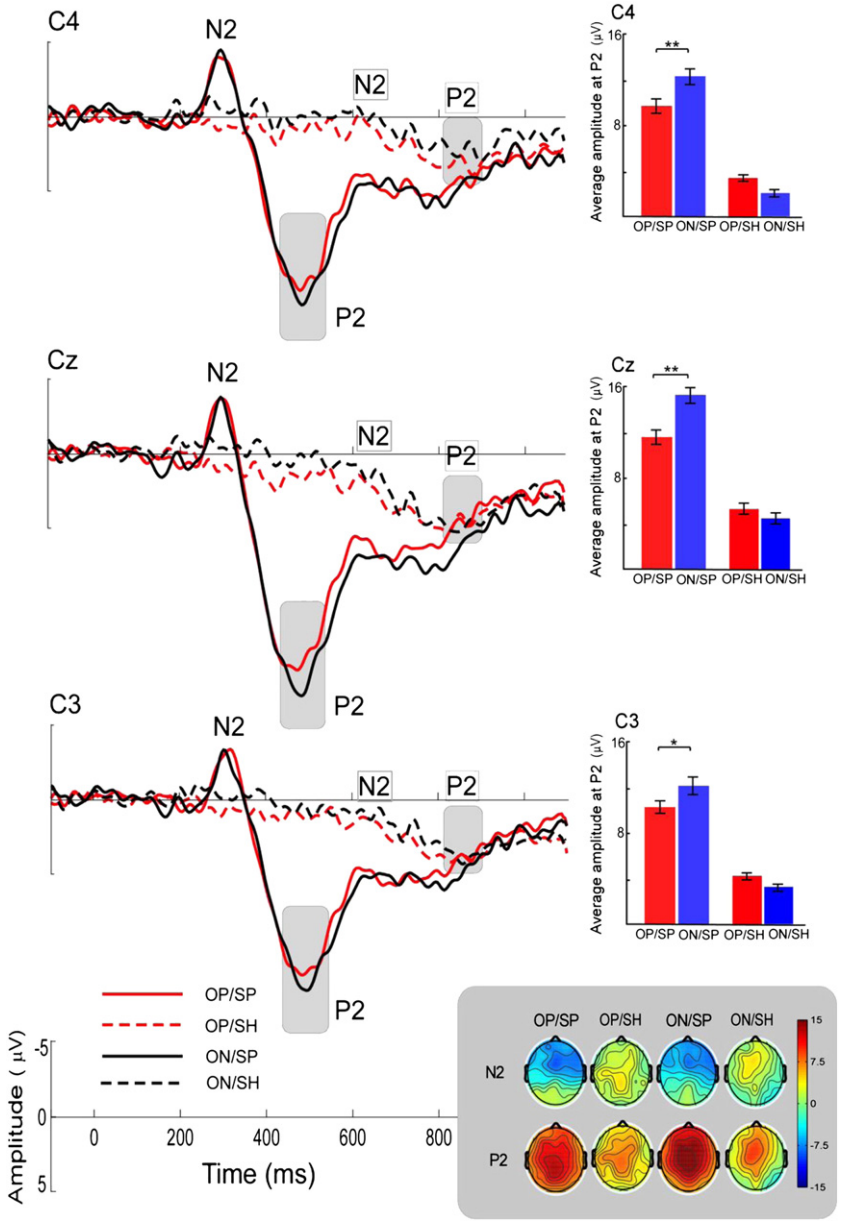

Fig. 3. Cortical responses to self-pain and self-heat targets primed by other-pain or other non-painful pictures. ERPs elicited by the other-pain prime/self-pain target condition (OP/SP, red solid line) were significantly decreased relative to those elicited by the other non-painful prime/self-pain target condition (ON/SP, black solid line) in the P2 time window. No significant difference was observed in ERP amplitudes elicited by other-pain or other non-painful primes to self-heat targets (OP/SH, red dashed line and ON/SH, black dashed line). The bar chart (right panel) illustrates averaged amplitudes within the P2 time window in each condition. At the bottom, voltage scalp maps are shown for N2 and P2 in each condition.

than self-heat targets (2.98 ± 0.20). No visual prime × sensory target interaction was observed for pain intensity [$F(1,19) = 1.13$, $p = 0.299$, $\eta^2 = 0.057$].

ERPs

Averaged ERP waveforms and scalp topographies related to each target type within each priming condition are shown in Fig. 3. Self-pain targets displayed a negative component (N2) from 275 ms to 325 ms maximal over the frontal-central area and bilateral temporal regions, as well as a positive deflection (P2) between 450 and 550 ms over the central area. Self-heat targets evoked a negative wave (N2) between 550 and 650 ms and a late positive component (P2) from 800 ms to 900 ms.

N2. The main effect for sensory target [$F(1,19) = 7.57$, $p = 0.013$, $\eta^2 = 0.321$] indicated that self-pain targets elicited a larger negative amplitude than did self-heat targets. For region [$F(2,38) = 6.89$, $p = 0.004$, $\eta^2 = 0.201$], amplitudes were larger in the right brain region than the left ($p < 0.001$) and central ($p < 0.001$) regions, which did not differ from one another ($p = 0.356$). The main effect for electrode [$F(4,76) = 11.34$, $p = 0.002$, $\eta^2 = 0.429$] revealed an N2 response over the frontal-central and bilateral temporal regions (see Fig. 3). None of the two-way, three-way, or four-way interaction was significant (all p-values > 0.05).

P2. For sensory target [$F(1,19) = 55.15$, $p < 0.001$, $\eta^2 = 0.785$], self-pain targets elicited larger amplitudes than did self-heat targets. For region [$F(2,38) = 29.79$, $p < 0.001$, $\eta^2 = 0.611$], amplitudes were larger in the central than the left ($p < 0.001$) and right ($p < 0.001$) regions, which did not differ from each other ($p = 0.742$). Simple effects analyses of the prime × target interaction [$F(1,19) = 6.73$, $p = 0.018$, $\eta^2 = 0.101$] confirmed that self-pain targets elicited smaller amplitudes when they followed other-pain primes rather than other non-painful primes [$F(1,19) = 10.29$, $p = 0.005$, $\eta^2 = 0.268$], while P2 amplitudes for self-heat targets were not affected by the visual prime types [$F(1,19) = 1.26$, $p = 0.276$, $\eta^2 = 0.001$]. No other main effect or interaction effect was significant (all p-values > 0.05).

Study 2

Behavioral data

Table 2 displays descriptive statistics for RTs, pain intensity ratings, and ERP amplitudes for visual targets in all conditions.

For RTs, the main effect for visual target [$F(1,19) = 11.54$, $p = 0.003$, $\eta^2 = 0.378$], indicated that participants responded faster in rating other non-painful targets (950.57 ± 62.68 ms) than other-pain targets (1146.82 ± 90.23 ms). While the main effect for sensory prime was not significant [$F(1,19) = 0.30$, $p = 0.588$, $\eta^2 = 0.016$], the sensory prime × visual target interaction [$F(1,19) = 7.26$, $p = 0.014$, $\eta^2 = 0.276$] indicated that RTs for rating other-pain targets were marginally faster if they followed self-pain rather than self-heat primes [$F(1,19) = 3.59$, $p = 0.073$, $\eta^2 = 0.159$]. RTs for other non-painful targets did not differ as a result of sensory primes [$F(1,19) = 2.58$, $p = 0.124$, $\eta^2 = 0.120$].

For pain intensity, the effect for sensory prime [$F(1,19) = 5.38$, $p = 0.032$, $\eta^2 = 0.221$] indicated that target pictures were rated more painful when they followed self-pain (4.29 ± 0.23) than self-heat primes (4.01 ± 0.17). For visual target [$F(1,19) = 102.76$, $p < 0.001$, $\eta^2 = 0.844$], other-pain targets (6.04 ± 0.19) were rated as more painful than other non-painful targets (2.26 ± 0.32). The associated sensory prime × visual target interaction was not significant [$F(1,19) = 0.55$, $p = 0.467$, $\eta^2 = 0.028$].

ERPs

Averaged ERP waveforms and scalp topographies of each prime-target condition are summarized in Fig. 4. ERPs for visual targets displayed a negative component from 120 ms to 170 ms (N1) over the frontal-central area, a positive deflection between 180 and 230 ms (P2) over the central area, and a negative deflection from 240 ms to 290 ms (N2) over the frontal region, followed by a positive component from 350 ms to 450 ms (P3) and a late positive deflection from 500 ms to 700 ms (LPC) over the central and parietal area. Significant effects were observed at P3 and LPC.

P3. For visual target [$F(1,19) = 12.80$, $p = 0.002$, $\eta^2 = 0.402$], other-pain targets elicited a more positive ERP deflection than did other non-painful targets. For region [$F(2,38) = 6.19$, $p = 0.011$, $\eta^2 = 0.246$], larger amplitudes were observed over the left than the right brain region ($p = 0.004$) but not the central region ($p = 0.125$). The effect for electrode [$F(14,76) = 31.32$, $p < 0.001$, $\eta^2 = 0.622$] revealed the presence of a P3 component over the central and parietal areas (see Fig. 4).

The sensory prime × visual target × region interaction [$F(2,38) = 5.70$, $p = 0.009$, $\eta^2 = 0.276$] indicated that sensory prime × visual target effects varied by region. To clarify localization of the effect, comparisons were made in each region. For the right region, the effect for sensory prime × visual target [$F(1,19) = 4.79$, $p = 0.041$, $\eta^2 = 0.201$] indicated that other-pain targets elicited smaller amplitudes when they followed self-pain rather than self-heat primes [$F(1,19) = 43.27$, $p < 0.001$, $\eta^2 = 0.695$]; amplitudes for other non-painful targets did not differ as a result of the sensory primes [$F(1,19) = 4.31$, $p = 0.052$, $\eta^2 = 0.185$]. Sensory prime × visual target interactions were not significant for the central [$F(1,19) = 0.745$, $p = 0.399$, $\eta^2 = 0.038$] or left [$F(1,19) = 0.190$, $p = 0.668$, $\eta^2 = 0.010$] brain regions. No other main effect or interaction effect was observed (all p-values > 0.05).

LPC. The main effect for sensory prime was not significant [$F(1,19) = 2.01$, $p = 0.172$, $\eta^2 = 0.096$], but for visual target [$F(1,19) = 24.51$, $p < 0.001$, $\eta^2 = 0.563$], other-pain targets elicited larger amplitudes than other non-painful targets did. For electrode [$F(14,76) = 25.03$, $p < 0.001$, $\eta^2 = 0.483$], an LPC response was present over the central and parietal areas (see Fig. 4). The sensory prime × visual target interaction was not significant [$F(1,19) = 0.17$, $p = 0.688$, $\eta^2 = 0.009$] nor were three-way or four-way interactions (all p-values > 0.05).

Discussion

This research assessed the effects of other-pain and self-pain primes on behavior and ERP responses to complementary (1) self-pain targets

Table 2
Descriptive statistics for behavioral and ERP data in Study 2.

Condition	RT (ms)	Pain intensity	P3 window (μV)			LPC window (μV)		
			Right	Middle	Left	Right	Middle	Left
SP/OP	1117.02 (95.91)	6.16 (0.19)	6.64 (1.25)	6.75 (1.56)	8.91 (1.14)	6.43 (1.27)	6.23 (1.50)	5.65 (1.27)
SH/OP	1176.61 (87.49)	5.92 (0.21)	8.00 (1.27)	8.48 (1.62)	9.88 (1.26)	7.29 (1.28)	7.36 (1.56)	6.03 (1.30)
SP/ON	968.87 (64.88)	2.43 (0.38)	5.74 (1.21)	5.72 (1.56)	7.73 (1.18)	3.39 (1.45)	3.25 (1.68)	3.23 (1.40)
SH/ON	932.26 (62.50)	2.09 (0.27)	6.31 (1.31)	7.02 (1.73)	8.93 (1.34)	4.55 (1.34)	4.58 (1.58)	4.53 (1.28)

SP/OP, self-pain prime/other-pain target; SH/OP, self-heat prime/other-pain target; SP/ON, self-pain prime/other non-painful target; SH/ON, self-heat prime/other non-painful target. Means and (SD) are shown.

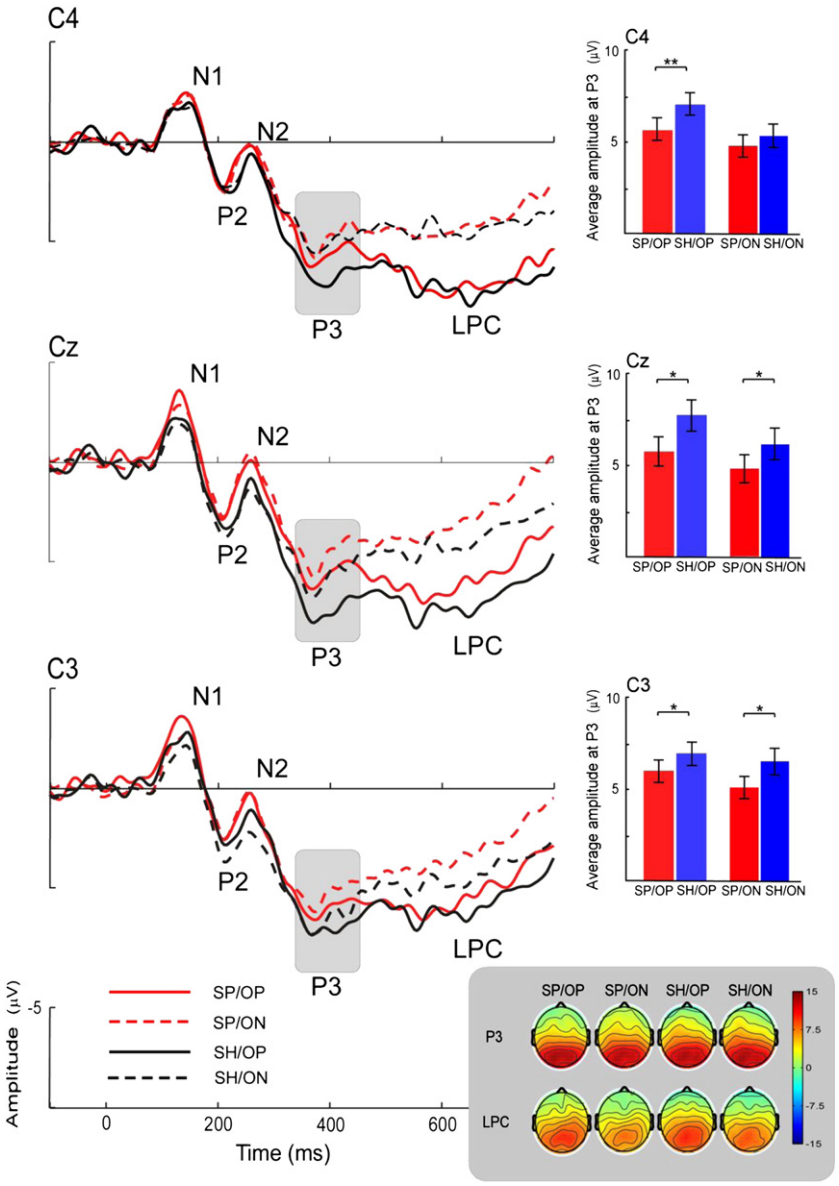

Fig. 4. Cortical responses for each sensory prime–visual target condition. ERPs elicited by the self-pain prime/other-pain target condition (SP/OP, red solid line) were significantly decreased relative to those elicited by the self-heat prime/other-pain target condition (SH/OP, black solid line) in the P3 time window within right lateral region (see C4). No significant effect was observed in ERP amplitudes elicited by self-pain or self-heat primes related to other non-painful targets (SP/ON, red dashed line and SH/ON, black dashed line). The bar chart (right panel) illustrates averaged amplitudes in the P3 time window for each condition. Voltage scalp maps of P3 and LPC in each condition are shown at the bottom.

and (2) other-pain targets. With notable exceptions, pain-related primes facilitated responsiveness to congruent pain-related targets, while responses to non-pain-related targets were not affected by prime types. As such, findings offered stronger support for assumptions of the "shared-representation" model than the "threat value of pain" hypothesis.

Study 1

Dovetailing with general assumptions about effects of primes on congruent versus incongruent targets (e.g., Fazio, 2001), and behavioral evidence specific to self-pain stimuli (Meng et al., 2012b), other-pain primes predicted faster RTs in rating self-pain intensities relative to other non-painful primes, while visual prime types were not differentially-related to RTs in rating self-heat stimuli. Following the shared representation model (Decety and Jackson, 2004), other-pain cues may have triggered representations of pain that facilitated rapid appraisals of congruent self-pain targets relative to less congruent self-heat targets.

Consistent with other works (Godinho et al., 2006, 2012; Kirwilliam and Derbyshire, 2008; Loggia et al., 2008; Meng et al., 2012b), other-pain primes also predicted higher pain intensity ratings for self-pain targets than did other non-painful primes. Self-heat targets were rated as non-painful regardless of prime type. However, counter to predictions, other-pain primes also had comparatively stronger effects on intensity ratings of self-heat targets. This finding was in line with research suggesting that activation of the pain matrix influences pain perception as well as non-painful heat perception (Kirwilliam and Derbyshire, 2008). Perhaps lingering representations, associations or memories of immediately preceding other-pain primes contributed to higher pain ratings for both noxious and non-noxious sensory targets relative to other non-painful primes which should not activate pain representations (e.g., Koster et al., 2005, 2006; Van Damme et al., 2006).

More generally, compared to other non-painful depictions, other-pain images elicit higher subjective and sympathetic arousal levels

(Loggia et al., 2008; Rainville et al., 2005), potentially contributing to increases in intensity ratings across sensory target types. Similarly, motivational priming theory (Lang, 1995) predicts that negatively-valenced primes increase pain perception via activation of an aversive system (Villemure and Bushnell, 2002). However, negative valences might not explain such effect fully, given that non-specific negative affective images do not affect self-pain to the extent that other-pain images do (see de Wied and Verbaten, 2001; Godinho et al., 2006).

In relation to ERP responses, paralleling Granovsky et al.'s (2005) study, during pain stimulation, an effect emerged for N2 over the frontal-central and bilateral temporal regions (275–325 ms) and a widespread central positive waveform (P2) was found (450–550 ms). This pattern may have reflected activation of Aδ-fibers. Heat stimulation corresponded to N2 activation (550–650 ms) and a long latency positive deflection (P2, 800–900 ms), possibly reflecting activation of C-fibers.

P2 amplitudes decreased for self-pain targets that followed other-pain primes rather than other non-painful primes. In conceptually-related work, Valeriani et al. (2008) reported reductions of N1/P1 during laser pain stimulation coinciding with viewing other-pain video clips compared to non-painful video clips while Valentini et al. (2012) found no amplitude differences elicited by pain stimulation corresponding to other-pain versus non-painful clips. Methodological differences between studies may have contributed to discrepancies. In the studies above, attention may have been divided during simultaneous presentations of video clips and self-pain stimuli. Perhaps because the capacity to detect painful environmental cues enhances survival, other-pain video clips diverted attention from coincidental self-pain to a greater degree than non-painful clips did (Villemure and Bushnell, 2002). As well, since laser stimulus onsets occurred at different points during the video clips, accompanying ERP waves may have been affected by the timing of events within the depictions. To reduce possible interpretive challenges associated with simultaneous presentations of visual and sensory stimuli, the present study used a priming paradigm in which cortical responses to sensory targets were disentangled from extraneous visual images.

Furthermore, decreased P2 amplitudes were contrary to evidence of (1) higher pain intensity ratings after other-pain than other non-painful primes and (2) substantial positive correlations between P2 and pain intensity (Iannetti et al., 2005; Ohara et al., 2004). Regardless, because pain intensities can remain constant while P2 amplitudes decrease when pain stimuli are repeated at short and constant inter-stimulus intervals, the initial presentation of pain stimulation may increase temporal expectancy for ensuing pain stimuli (see Iannetti et al., 2008). Following this logic, other-pain primes may have activated pain representations, increasing expectations that target stimuli would be painful. Hence, other-pain prime/self-pain target trials may not have elicited surprise or perceived novelty to the extent that violated expectations in other non-painful prime/self-pain target trials did, resulting in facilitated processing of self-pain targets based on both decreases in P2 amplitudes and faster RTs.

Study 2

Study 2 indicated that self-pain primes modulated select responses to other-pain targets. For RTs, Meng et al. (2012b) recently found that participants (n = 30) were significantly faster in judging other-pain targets that followed self-pain primes rather than self-heat primes; conversely, RTs for judging other non-painful targets did not differ on the basis of sensory primes. This study replicated the above pattern for RTs to a marginally-significant extent, despite having lower power based on a smaller sample (n = 20). Firm conclusions cannot be drawn in the absence of additional replications but, together, the two studies suggest that relative to self-heat stimuli, self-pain increases speed in judging severity of other-pain. Because perspective-taking enhances affective and brain responsiveness to other-pain (Lamm et al., 2007, 2008; Li and Han, 2010), perceived similarity and congruence in representations of self-pain and other-pain experience (Decety and Jackson, 2004) may have facilitated speed of judging pain intensities in this condition.

Also of note, the main effect for target reflected faster RTs in judging other non-painful than other-pain target pictures while RTs were faster in rating self-pain sensory targets than self-heat targets in Study 1. Regarding Study 2, complexity differences in rating other-pain versus non-painful targets may have been an influence, given that RTs in decision-making tasks are longer as the number of response choices increases (e.g., Maylor et al., 1992). Specifically, intensities were rated on a 9-point scale wherein "4" defined the pain threshold. Consequently, the range of options for judging other non-painful targets (1–3) was one-half the number of options used to judge other-pain targets (4–9). Response complexity should have also affected RTs of intensity judgments since the same 9-point rating scale was used in Study 1. However, the rate at which target stimuli were transmitted may have exerted an even stronger effect on response latencies for Study 1. In contrast to Study 2 wherein other-pain and other non-painful target pictures were transmitted at an identical (rapid) rate, self-pain targets were transmitted by Aδ-fibers at a conduction velocity of 10 m/s while self-heat targets were transmitted by C-fibers at a rate of 1.0 m/s (see Study 1 ERP results and Fig. 3). Because sensory pain stimuli are immanently more threatening to physical integrity, they may be processed with greater efficiency.

Findings for intensity ratings closely paralleled those observed in Study 1. Compared to self-heat primes, self-pain primes resulted in higher intensity ratings of other-pain targets, similar to work linking self-pain to higher intensity and empathic emotional response ratings of other-pain (Preis and Kroener-Herwig, 2012). However, facilitative effects of self-pain primes on intensity ratings were not specific to pain-congruent targets and extended to ratings of other non-painful depictions, perhaps due to mechanisms discussed in relation to the associated pain intensity findings from Study 1.

Consistent with other ERP studies (e.g., Decety et al., 2010; Fan and Han, 2008), an early negative component (N1) in the frontal area, a positive deflection (P2) over the central area, a negative component (N2) over the frontal region, and long-latency positive deflections (P3 and LPC) over the posterior parietal area emerged in Study 2. Regarding differences, other research reported differences in early versus late components (Decety et al., 2010; Fan and Han, 2008), while ERP differences were observed only after 300 ms in Study 2. Compared to use of painful or non-painful pictures without primes in the above accounts, our paradigm included sensory primes as well as other-pain or other non-painful targets. Hence, sensory primes may have occupied early attention, resulting in delayed ERP amplitude differences in relation to subsequent targets.

As hypothesized, P3 amplitudes for other-pain targets were significantly decreased when they followed self-pain primes rather than self-heat primes while sensory primes did not differentially influence P3 in response to other non-painful targets. P3 amplitudes over the posterior parietal area have been linked to stimulus evaluation processes that are, to a certain degree, independent of response selection and execution (McCarthy and Donchin, 1981; Olofsson et al., 2008). Hence, the pattern for P3 might reflect use of fewer cognitive resources in evaluating other-pain targets preceded by congruent self-pain primes than self-heat primes.

The P3 effect was limited to the right lateral region, suggesting contra-lateral hemispheric dominance in the modulation of self-pain on perception of other-pain. Sensory stimulation was applied to the left forearm, so cortical electrophysiological response over the contra-lateral scalp (largely generated in right S1 or S2) would be expected (Garcia-Larreaa et al., 2003; Iannetti et al., 2005; Valentini et al., 2011). Given relations between observing other-pain and corresponding somatosensory cortical excitability in the self (Avenanti et al., 2006; Voisin et al., 2011), it is reasonable to assume that cortical activation elicited by painful stimulation in specific regions influences cortical processes related to observing other-pain in corresponding regions.

General discussion

Across two studies, pain processing was facilitated in pain-prime/pain-target conditions compared to non-painful-prime/non-painful target conditions. Specifically, in Study 1, other-pain primes predicted comparatively faster RTs in judging pain intensities, higher intensity ratings, and decreased P2 amplitudes in response to self-pain target. In Study 2, self-pain primes predicted marginally faster RTs as well as significantly higher intensity ratings and decreased P3 amplitudes in response to other-pain targets compared to effects of self-heat primes. Together, the pattern supports a premise central to the shared-representation model (Decety and Jackson, 2004; Jackson et al., 2005): observing other-pain and experiencing self-pain may activate pain representations that increase responsiveness to painful targets that share features of such representations. Conversely, results did not support the threat value of pain hypothesis which suggests that self-pain is a threat that can ultimately reduce responsiveness to other-pain (Coll et al., 2012).

The shared representation account implies that facilitative effects of pain-based primes are specific to congruent (pain)-targets and do not extend to non-pain-related stimuli. This tenet was partially supported by marginally to significantly faster RTs and significantly decreased late ERP amplitudes in pain-congruent prime-target conditions compared to conditions featuring pain-related primes and non-pain-related targets. However, at variance with the assumption of specificity, other-pain (Study 1) and self-pain (Study 2) primes resulted in higher intensity ratings of both pain-related and non-pain-related targets compared to target stimuli preceded by non-pain-related primes.

Despite its possible implications, this research has four main limitations that serve as foundations for further work. First, the sensory stimuli used were of short durations (432 ± 2 ms) that provided sharp sensory perceptions. Accordingly, it is not clear that similar modulation effects would be observable for sensory stimuli of longer durations or higher intensities. Indeed, when self-pain stimuli are intense and/or prolonged (i.e., more threatening), reduced responsiveness to other-pain in the form of slower RTs and increased late ERP amplitudes seems plausible. Extensions to varied stimulus durations may elucidate conditions under which shared representation and threat value of pain hypotheses are most applicable. Inclusion of longer stimulus durations within designs may allow for consideration of integrated accounts (e.g., initial increases in responsiveness to pain followed by escape/avoidance as threatening or noxious stimuli persist). Second, because behavior reactions and patterns of cerebral activation differ in response to other-pain images featuring body limbs versus those depicting facial expressions (see Vachon-Presseau et al., 2011, 2012), extensions are needed to evaluate whether patterns of effects from this research generalize to emotional-communicative pain stimuli.

Third, rather than providing pain intensity ratings immediately after target stimulus onsets, participants inhibited these responses until the intensity scale appeared onscreen 1 s after target offsets to reduce interference from response preparation and movements on EEG data. Because of this time-lag in responding and the unequal number of response options provided for judging stimuli as painful versus non-painful, the validity of the RT findings warrants scrutiny; extensions that assess speed of responding immediately after stimulus onsets and equate response options for judging painful and non-painful targets may clarify the veracity of RT effects.

Finally, sensory and visual stimuli were not strictly matched. That is, pain-related primes and targets shared key perceptual properties (i.e., noxiousness, negative affective valences) central to priming paradigms (e.g., Fazio, 2001) but thermal stimuli were applied to participants and mechanical or pressure stimuli were featured in corresponding visual images. Future research should examine whether pain-congruent prime-target pairs matched for type of stimulation as well as valence and noxiousness result in stronger or weaker congruence effects based on respective assumptions of shared representation and threat value of pain accounts.

Conclusion

In sum, compared to non-painful primes, other-pain and self-pain primes had generally facilitative effects on RTs, pain intensity ratings and ERP responses to complementary self-pain and other-pain targets. This pattern supported the shared-representation model premise that painful primes increase responsiveness to pain-congruent targets. However, the assumption of specificity regarding facilitative effects of painful primes on responses to pain-congruent targets garnered mixed support.

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