Effects of social presence on behavioural, neural and physiological aspects of empathy for pain

Abbreviated title: Effects of social presence on empathy for pain

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Abstract

Social interactions are rich in cues about others' mental and emotional states, and these cues have been shown to facilitate empathy. As more and more social interactions shift from direct to mediated interactions with reduced social cues, it's possible that empathy is affected. We tested whether behavioural, neural and physiological aspects of empathy for pain are reduced in a video-mediated interaction. To this end, 30 human participants (23 females, 7 males) observed one of 5 targets (all female) undergoing painful electric stimulation, once in a direct interaction and once in a live, video-mediated interaction (within-subject design) while EEG was measured. On a behavioural level, we found that observers were as accurate in judging others' pain via video as in a direct encounter and reported the same level of distress. On the neural and physiological levels, the theta response to others' pain and skin conductance coupling in the dyad were reduced in the mediated condition. Other measures, including mu suppression (a common marker of pain empathy), were not affected by condition. To conclude, a video-mediated interaction did not impair the cognitive aspects of empathy for pain, i.e., understanding the other accurately. However, the reduced theta response and reduced skin conductance coupling suggest that physical proximity with its rich social cues is important for other stimulus-driven physiological responses that may be related to resonance with the other's experience. Our results encourage more research on the role of social presence for different empathy components.

Key words: social presence, pain empathy, empathic accuracy, mu suppression, physiological coupling

Significance Statement

In mediated interactions (e.g. video calls), less information is available about the other. However, no study so far has investigated how this affects our empathy for one another. Here we show in human dyads that while some cognitive and affective aspects of pain empathy are unchanged in a video-mediated compared to direct interaction, some neural and physiological aspects of pain empathy are reduced. These results imply that there are neurocognitive consequences to remote social interactions, warranting future studies to confirm these results and to understand their behavioural significance.

Introduction

Over the last decades, many social interactions in private life and at work, including medical and psychotherapeutic contexts, have shifted from personal encounters to mediated interactions such as video calls. These mediums provide less detailed social cues and information channels and limit opportunities for immediate, reciprocal interaction compared to personal interactions. These factors are sometimes referred to as intimacy and immediacy, and together they contribute to the degree of social presence offered within social interactions (Cui et al., 2013; Short et al., 1976; see Biocca et al., 2003, for alternative accounts of social presence). Given the reduced social presence of mediated interactions, the question arises of how this changes our ability to share and understand others' affective states, i.e., our ability to empathize (Decety & Jackson, 2004; Shamay-Tsoory, 2011). Focusing on intimacy, we asked how the reduction of social cues – using a video call versus a direct interaction – affects empathy. We used empathy for pain as a well-established model to investigate the behavioural, neural and physiological aspects of empathy (Singer & Lamm, 2009).

Empathy for pain is a multifaceted process, including an affective response, feelings of distress and empathic care towards the person suffering (Goubert et al., 2009; Lamm et al., 2007; Singer & Lamm, 2009), as well as cognitive processes. The latter are sometimes measured as empathic accuracy, which is the accuracy of one's perception of the other's pain (Laursen et al., 2014; Zaki et al., 2009b).

On the neural level, empathy for pain has been associated with mu suppression in the EEG: reduced power between 8 and 13 Hz over the somatosensory cortex (Cheng et al., 2008; Gallo et al., 2018; Peled-Avron et al., 2018; Peng et al., 2021; Perry et al., 2010a). Mu suppression has been linked to higher empathic accuracy (Goldstein et al., 2018) and might aid empathy by representing the other's bodily state in one's own somatosensory system (Riečanský & Lamm, 2019). Other studies examined mid-frontal theta activity (4–8 Hz) as a possible electrophysiological component of empathy (Mu et al., 2008; Peng et al., 2021) and own pain experience (Misra et al., 2017; Peng et al., 2021; Ploner et al., 2017). Mid-frontal theta is thought to indicate activity in the anterior cingulate cortex (ACC, Mitchell et al., 2008; van der Molen et al., 2017), which is often reported in fMRI studies on empathy for pain and is associated with the negative affect during own and others' pain (Fallon et al., 2020).

Finally, several studies suggest that physiological "coupling" (in cardiac activity or skin conductance), i.e., aligning to the physiological state of someone in pain, might reflect empathic sharing and facilitate understanding of the other (Goldstein et al., 2017; Jospe et al., 2020; Reddan et al., 2020; Zerwas et al., 2021).

Humans respond to social cues such as facial expressions or eye contact by sharing the other's emotional state (Ensenberg et al., 2017; Hess, 2021; Perry et al., 2010b). Reducing the availability of social cues and thereby the intimacy of the interaction may thus reduce affective resonance and impair understanding of others. To test the effect of intimacy on empathy for pain in the current study, pairs of participants underwent an empathy-for-pain paradigm in two conditions: one direct, face-to-face interaction and one interaction mediated via real-time video transfer. In both conditions, one participant (the target) received painful electrical stimulation while the other (the observer) was watching. We measured EEG from the observer and behavioural, cardiac and skin conductance responses from both members of the dyad. We hypothesized that the reduced level of intimacy in mediated compared to direct interactions diminishes empathic accuracy (Agahi & Wanic, 2020; Jospe et al., 2020; Zaki et al., 2009a) and affective empathy (Bogdanova et al., 2022; Ionta et al., 2020). We also expected it to reduce neural and physiological responses to another's pain and physiological coupling within the dyad (Murata et al., 2020).

Methods

Participants

Five female psychology students were recruited as targets. Only females were recruited for the targets to reduce possible gender effects over the dyads. They were on average 19.8 years old (SD = 0.75). Thirty psychology students (7 males, 23 females, mean age(SD) = 24.07(4.68) years) were recruited as observers. A priori power analyses with data simulations using the *simr* package (Green & MacLeod, 2016) in R showed that this sample size is sufficient to detect a small effect (f_2 = 0.02) of condition on empathic accuracy (measured via an interaction effect between shock intensity and direct vs. mediated condition; see below for statistical analyses) with a power of at least 0.98 (depending on exact model structure). For both targets and observers, exclusion criteria were current psychiatric or cardiovascular and past or current neurological disorders, current or chronic pain conditions or current pain-medication intake. For one observer, all physiological data from the own-pain condition had to be excluded because of missing stimulus triggers. Skin conductance and electrocardiogram (ECG) data from one target (mediated-interaction condition) were missing due to a technical error. Skin conductance data from two observers could not be analysed due to poor data quality. These dyads were excluded from all analyses of the missing outcome variable. Targets received €10 per hour; observers received course credit or €10 per hour. All participants provided written informed consent prior to taking part in the study, including consent for video-recording them and showing the videos to other work group members, and in case of the targets, showing the videos to other participants in future studies. The experiment was carried out according to the Declaration of Helsinki and was approved by the Ethics Committee of the University of Lübeck.

Experimental design

Each target interacted with six different observers on six different study days. Observers came to the lab once for one session (two interactions) with one target. For the observers, there were three conditions (within-subject design, see Fig. 1A). In the “own pain” condition, the observer was alone in the laboratory and received electric shocks. This ensured that observers knew what the electric shocks felt like. In the “direct interaction” condition, observer and target sat opposite each other at a table, and the target received electric shocks while the observer watched. In the “mediated interaction” condition, target and observer sat in adjacent rooms and saw each other over a real-time video transmission. Observers watched the same target in the latter two conditions and rated the observed pain experience of the target. Targets rated their own pain experience. In all conditions, skin conductance and ECG were recorded from both participants, and EEG was recorded from the observer. The “own pain” condition was always carried out first, and the order of “direct interaction” and “mediated interaction” conditions was pseudo-randomized over participants. In the latter two conditions, targets’ and observers’ behaviour was video-recorded.

Stimulus calibration

Prior to the pain task, pain stimuli were calibrated to the subjective pain thresholds of the participants (the observers in the “own pain” condition, and the targets prior to the first interaction condition). To this end, participants received electric shocks starting from 0 mA, increasing in amplitude in steps of 0.5 mA. They were required to rate each stimulus on a scale from 0 (“not perceivable”) to 8 (“strongest pain imaginable”). As soon as they rated a stimulus with “7” (“unbearably painful”), stimuli were decreased in amplitude (again in steps of 0.5 mA) until participants rated the stimulus as “0” or an amplitude of 0 mA was reached. The procedure was then repeated once more with increasing stimulus intensity. The stimulus intensity that was rated as 1 (“noticeable”) in this last round was used as the lower limit for the stimuli presented during the task, with the stimulus intensity rated as 6 (“extremely painful”) used as the upper limit.

Pain task

The pain task itself was adapted from Rutgen and colleagues (2015). Electric shocks were delivered using a DS 5 isolated bipolar constant current stimulator (Digitimer) and a bar electrode (Digitimer, two electrodes with 9-mm diameter, 30 mm apart) attached to the back of the right hand. The skin under the electrode was treated with an abrasive paste and conductive gel to reduce the electric resistance of the skin.

Each trial started with an auditory cue lasting 500 ms that did not predict the shock stimulus intensity. At 1000 ms after the cue, the electric shock was delivered for 500 ms (series of 2-ms electric pulses, interspersed with approximately 20-ms breaks). After a randomly varying interval (6000–9000 ms), the next trial or the rating followed. In 50% of the trials, participants were prompted to rate the stimulus by a vocal recording saying, “Please rate”. We included the rating only in 50% of the trials to keep the duration of the experiment feasible. The rating was given on a tablet computer. Targets rated how painful the electric shock was for themselves on a visual analogue scale ranging from “not at all painful” to “extremely painful”. Observers rated how painful they thought the electric shock was for the target (on the same visual analogue scale) as well as how unpleasant it was for them to watch the target receive the electric shock (on a visual analogue scale ranging from “not at all unpleasant” to “extremely unpleasant”). The latter rating served as a measure of affective empathy. Electric shocks varied in intensity in 20 steps from the intensity the targets had rated as “noticeable” (intensity level 1) to the intensity they had rated as “extremely painful” (intensity level 20) during the calibration. There were 80 trials in each condition, and each intensity occurred four times. The order of intensities was pseudo-random (with no more than four shocks with intensity level higher than 10 or lower than 11 in a row) but fixed for all participants and conditions. The order of trials that had to be rated was fixed as well. In the “own pain” condition, the task was the same except that observers rated their own pain experience on the visual analogue scale. Targets and observers were instructed not to talk or move excessively during the task but were otherwise allowed to express their emotions freely. Observers were instructed to rate the pain of the other as accurately as possible.

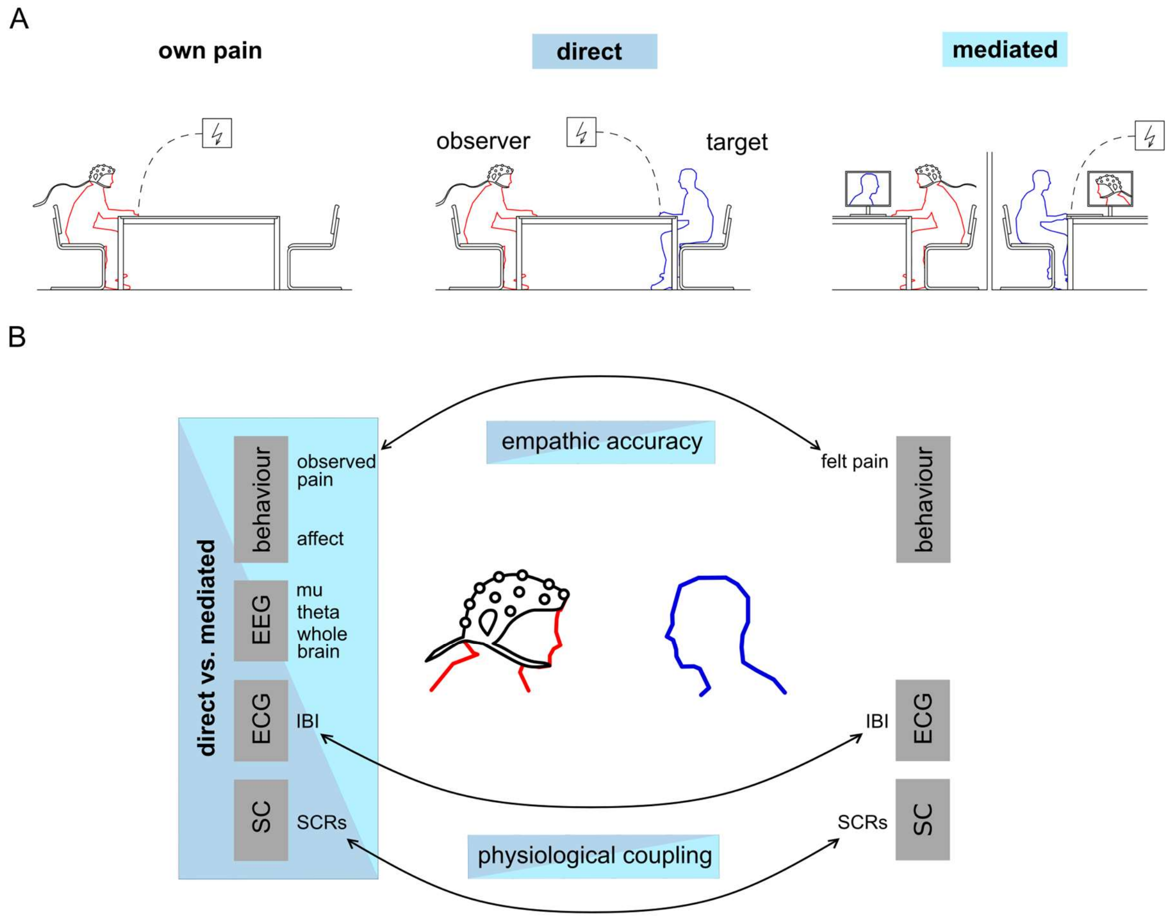

Figure 1. (A) Schematic overview of experimental conditions. (B) Overview of the analysed outcomes.

Experimental procedure

Target selection

Before targets interacted with observers, they came to the laboratory alone to familiarize themselves with the procedures and the stimuli. In this first session, they did the same pain task as in the main experiment, but with no other person in the room. As in the main experiment, skin conductance and ECG were recorded. After the first session, targets decided whether they wanted to participate in the main experiment. Moreover, we used this first session for target selection, as we aimed to recruit only targets who set the intensity limits of the stimuli during stimulus calibration to a level that was actually painful. This was important to ensure that we measured actual pain empathy during the main experiment. We therefore defined the minimum upper intensity limit that targets had to reach during the first session to be eligible for the full experiment as within +/-1 standard deviation of the mean upper intensity limit of a pilot study (resulting in a minimum upper intensity limit of 2.5 mA). We invited seven potential targets to this first session. Due to the criteria, we had to exclude one target, and one participant dropped out after the first session, which left five targets for the main experiment.

Main experiment

For the main experiment, the observer arrived first in the laboratory. After the informed-consent form was signed, the EEG, skin conductance and ECG measurement equipment as well as the stimulus electrode were prepared. The pain-calibration procedure was carried out. After five practice trials to familiarize themselves with the ratings on the tablet computer, participants did the pain task in the “own pain” condition. Meanwhile, the target arrived in a different room, responded to questionnaires and was equipped with the electrodes for physiological measurement. As soon as the “own pain” condition was finished, the stimulus electrode was attached to the target’s right hand, and the target underwent the pain-calibration procedure. Meanwhile, the observer completed questionnaires. When both were finished, the experimental tasks started (see “pain task” above; either “direct interaction” or “mediated interaction” first). Afterwards, target and observer were seated in different rooms again and replied to post-experimental questionnaires. Finally, observers were debriefed about the aim of the study. Targets were debriefed only after completing all six sessions.

Questionnaires

We assessed participants’ age, gender, body weight and height, educational degree, and habits regarding smoking, caffeine consumption and physical activity. After the experiment, we obtained participants’ subjective evaluation of the experiment and observers’ evaluation of the target. They also filled out two personality questionnaires: the Interpersonal Reactivity Index (Davis, 1983; German version: Paulus, 2009) and the Emotion Regulation Questionnaire (Gross & John, 2003; German version: Abler & Kessler, 2009). These data are not further evaluated here.

Physiological data acquisition

Participants were asked to refrain from smoking, exercise, alcohol and caffeine for at least six hours before the experiment to prevent these factors from impacting the physiological measurements. EEG data were recorded with 59 Ag/AgCl electrodes placed on an elastic cap according to the international 10-20-system (using a BrainAmp MR plus amplifier, BrainProducts GmbH,). An online reference electrode was placed on the left earlobe, while an offline reference electrode was placed on the right earlobe. Horizontal and vertical EOG were recorded with four electrodes placed next to the outer corners of the eyes and above and below the left eye, respectively. Sampling rate was 500 Hz, and data were recorded with an online high pass filter of 0.016 Hz, a low pass filter of 48 Hz and a notch filter at 50 Hz. Impedances were kept below 5kΩ.

ECG data were recorded with bipolar Ag/AgCl recording electrodes and one reference electrode, using a 50-Hz notch filter. One of the recording electrodes was placed on the right forearm, the other one on the left lower calf of the participant (following Einthoven lead II configuration, Einthoven et al., 1950). Skin conductance was measured with two electrodes placed on the thenar and hypothenar of the left hand, using a 50-Hz notch filter. An electrode attached to the left forearm served as ground for both ECG and skin conductance, which were recorded with the same amplifier (BrainAmp ExG, BrainProducts *GmbH*). In the “direct interaction” and “mediated interaction” conditions, data from observer and target were recorded synchronously by connecting all amplifiers to the same USB adapter feeding the data into BrainVisionRecorder (version 1.21.0102, BrainProducts GmbH).

Physiological data processing

EEG data

All pre-processing was done in EEGLAB, version 2020.0 (Delorme & Makeig, 2004), implemented in MATLAB R2019b (The Mathworks*)*. Data were re-referenced to the right earlobe, and bipolar horizontal and vertical EOG channels were computed. Consistently bad channels (mean number = 2.01, range = 0 to 8 channels per participant and condition) and data segments with large artefacts were removed from the data (resulting in on average 1.6% of removed trials per participant and condition in the final epoched data). A bandpass filter was applied (finite impulse response filter, lower limit: 1 Hz, upper limit: 40 Hz, filterorder: 16500). Next, independent component analysis (ICA; implemented with the *runica* function in EEGLAB) was used for ocular artefact correction. Independent components that were clearly related to eye blinks or horizontal eye movements based on topography and time course were visually detected and removed (ranging from 2 to 6 components per participant). Afterwards, the weights of the remaining components were projected onto the original unfiltered data (Stropahl et al., 2018). Channels that had been removed before the ICA were interpolated (spherical interpolation); For some participants additional bad channels (mean number = 0.44, range = 0 to 3 channels per participant and condition) had to be interpolated. Data were then filtered with a bandpass filter with a lower limit of 0.2 Hz and an upper limit of 40 Hz (Finite impulse response filter, filter order = 16500, hamming window). Afterwards, data were segmented into stimulus-locked epochs of 4500-ms lengths (1000 ms before and 3500 ms after stimulus onset) and baseline-corrected to 1000 ms before stimulus onset. A voltage threshold (between -70/70 µV and -100/100 µV) was manually set for each participant in a way that all trials with non-ocular artefacts were removed. The number of rejected trials varied from 0 to 31% per participant and condition and did not differ between conditions (M = 13% in all conditions).

For the time-frequency analysis, single-trial data of all electrodes were convolved with a complex Morlet wavelet as implemented in MATLAB (function *cwt* with parameter specification 'cmor1-1.5'):

$$\omega(t) = (\pi f_b)^{-0.5} e^{-2\pi i f_c t} e^{-\frac{t^2}{f_b}},$$

where f_b = 1 is the bandwidth parameter, and f_c = 1.5 is the wavelet center frequency. Specifically, for each participant, changes in time-varying energy were computed (square of the convolution between wavelet and signal) in the frequencies (1–40 Hz, linear increase) for the 1500 ms after shock onset. Power values were converted to decibels with respect to an average baseline from 500 to 50 ms before stimulus onset (Cohen, 2014). For analyses of peak-frequency power (see next paragraph), we subtracted the averaged data from 500 to 50 ms before stimulus onset as a baseline correction.

To analyse mu suppression, we determined the individual peak frequency of mu power for each participant by using the *restingIAF* toolbox (Corcoran et al., 2018). Frequency peaks in the range from 8 to 13 Hz were detected in the baseline data from 1000 to 0 ms before shock onset in the "own pain" condition at central electrodes (C1, C2, C3, C4, C5, C6, CP1, CP2, CP3, CP4, CP5, CP6, FC1, FC2, FC3, FC4, FC5, FC6). For each detected frequency peak, the difference in average power between baseline (-1000 to 0 ms) and stimulation (0 to 1000 ms after shock onset) was calculated. The electrode and corresponding peak frequency with the strongest shock-related desynchronization for each participant was chosen for all further analyses. In 16 participants, a clear peak frequency was detectable. Peaks occurred at all possible frequencies except 9 Hz, and at the following electrodes: C1, C4, C3, CP2, CP3, C6, CP6, FC6, and FC2. The remaining 13 participants did not show a peak frequency, and for them data from the most frequent peak frequency and electrode were used (11 Hz at C4).

ECG data

ECG data were loaded into EEGLAB, filtered with a bandpass filter (lower: 1 Hz, upper: 30 Hz, finite impulse response filter, filterorder = 8250) and segmented into epochs of 2 s before and 8 s after stimulus onset for the stimulus-locked analyses. The MATLAB function *findpeaks* was used to detect the r-peaks in the segmented as well as the continuous data (for additional analyses of physiological coupling). Afterwards, data were visually screened for wrongly assigned or missing r-peaks. Data sections containing extrasystoles or otherwise undetectable R-peaks were treated as missing values. The interbeat interval (IBI) in ms was calculated for every pair of heartbeats and used as IBI value for each original data point in between the two heartbeats. In this way, the IBI trace had the same time resolution as the original data. For the analysis of condition differences in IBI responses to shocks, the mean IBI from 2 to 5 s after cue onset (Sperl et al., 2016) was computed and baseline-corrected to the mean of the 2 s before cue onset.

Skin conductance data

Skin conductance responses (SCRs) were analysed using the Ledalab-Toolbox (Benedek & Kaernbach, 2010, version 3.4.8) in Matlab. Data were downsampled to 50 Hz, smoothed and visually screened for strong artefacts, which were spline-interpolated. Afterwards, a continuous deconvolution analysis was conducted to separate phasic from tonic skin conductance activity (Benedek & Kaernbach, 2010). In the following, the mean phasic driver activity 1–4 s after shock onset was used for analyses (for targets' SCR in physiological coupling). For the observer data (condition differences and physiological coupling), four different time lags (1–4 s) were considered to account for a time lag in the observer's response to the target's pain expression.

Statistical analyses

We first outline the general statistical analysis approach before specifying the details. One set of statistical analyses examined the observers' responses to the targets' pain (empathic accuracy, unpleasantness ratings, neural and physiological responses; see Fig. 1B, left side). In these analyses, the predictors, shock intensity, condition (direct vs. mediated) and their interaction were tested. A significant effect of shock intensity indicates that the observer's responses are influenced by the other's pain and are therefore interpreted as empathic. A significant effect of condition indicates that social presence generally changes the observers' behaviour and physiology, whereas an interaction between shock intensity and condition indicates that social presence alters the sensitivity to another's pain. The second set of analyses examined physiological coupling between observer and target responses (see Fig. 1B, bottom). In these analyses, the targets' responses, the condition (direct vs. mediated) and their interaction served as predictors. A significant effect of targets' responses indicates that observers' and targets' responses are generally coupled, whereas an interaction with condition indicates stronger coupling in one condition compared to the other.

In both sets of analyses, (generalized) linear mixed models with single trials (Level 1) nested in observers (Level 2) and observers nested in targets (Level 3) were calculated. For theta activity, responses were averaged over trials. For the peak-mu analysis, permutation tests over the whole time course were conducted, as there was no predefined time window. To test for effects of intensity, condition and their interaction beyond mu suppression, exploratory permutation tests on the whole EEG data space were conducted.

To assess the robustness of the findings, the analyses of mu, IBI and SCRs were repeated with data averaged over trials. In these analyses, the factor intensity was dichotomized into low and high intensity (low: 1 to 10; high: 11 to 20). The results of these analyses are not reported, as they did not differ from the single-trial results. In the figures, data are dichotomized into low and high intensities for display purposes only. Permutation tests were carried out in MATLAB (version R2019b, The Mathworks*)*, and all other statistical analyses were carried out in R (version 4.0.2, *R core team*).

Behavioural data

To test for condition differences in empathic accuracy, we conducted a negative binomial generalized linear mixed model (using the function *glmer.nb* in the *lme4* package in R, Bates et al., 2020) on observers' single-trial ratings of the other's pain (Lawless, 1987). To find the best random slopes structure, first models with the full fixed-effects structure and different random slopes were compared using the Akaike Information Criterion (AIC, Akaike, 1998). An AIC difference greater than 2 was set as the threshold for a significant difference (Burnham & Anderson, 2004). Then one fixed predictor after the other was added to the model with the optimized random-effects structure, and only predictors that significantly improved the model were kept. The same procedure was used to test for differences in unpleasantness ratings between the conditions.

EEG data

To test whether mu suppression was modulated by shock intensity (20 levels), condition (direct vs. mediated) or their interaction, permutation tests were conducted on the mu peak-frequency power within the window from -500 to 1500 ms after stimulus onset (see Cohen, 2014). To test for effects of shock intensity on mu suppression, Spearman correlations between normalized single-trial power and single-trial shock intensity were calculated for each data point and across participants. This yielded a time course of correlation coefficients between peak-frequency power and shock intensity. Correlation values were z-transformed by comparing them to a permutation-based null-hypothesis distribution (based on randomly shuffling over trials 1000 times). To correct for multiple comparisons, a maximum value correction was used (Cohen, 2014).

To test for condition differences in mu suppression regardless of shock intensity, mu power was averaged over trials and compared between conditions. For the null-hypothesis distribution, the assignment of condition was randomly shuffled over participants (1000 permutations). To test for condition differences in the correlation between shock intensity and power (whether power tracked shock intensity to a larger degree in the direct condition), the condition difference between correlation coefficients was calculated for each data point and participant and compared to a random distribution (shuffled between conditions in 1000 permutations). For a comparison, we also analysed power in the traditional mu band (8–13 Hz, electrode C4, 500–1000 ms after shock onset). We chose the time window according to previous literature and to be comparable to the time window in which the response to own pain occurred (Zebarjadi et al., 2021).

For the effects of condition, shock intensity and their interaction on theta responses, we analysed power in the traditional theta band (4–8 Hz, electrode Fz, 0–500 ms after shock onset). We chose the time window according to previous literature (Mu et al., 2008; Peng et al., 2021). In the latter two analyses, we used a linear mixed model with condition averages nested in observers and observers nested in targets and dichotomized shock intensities.

Finally, in an exploratory analysis, using the same permutation test method as described above for peak-frequency mu, we looked for main effects of shock intensity (20 levels), condition and their interaction on power across the whole time-frequency-electrode space (1–20 Hz, all electrodes). For the effect of shock intensity, a time window from 0 to 1500 ms was used, for the other analyses a time window from -500 to 1500 ms. For the effect of shock intensity data were downsampled to 125 Hz to reduce computation time. Again, a maximum value correction and additionally a cluster size correction (Cohen, 2014) were used to correct for multiple comparisons.

IBI and SCRs

To test for condition and shock intensity (20 levels) effects on the observers' IBI, linear mixed models on the single-trial IBI averages were conducted (using the *lmer* function in *lme4* in R). The same procedure was used for the skin conductance data. Here, first the time lag with the greatest SCR over all conditions was selected for further analyses. To account for interindividual variation in SCR levels, SCR means were normalized by dividing them by participants' individual standard deviation. As SCR data were not normally distributed, generalized linear mixed models (using the *glmer* function in *lme4*, Gamma family, log-link function) were used for these data. To assess the robustness of the findings, all analyses were also carried out with data averaged over trials (with dichotomized shock intensities).

To test for coupling between targets' and observers' IBI and SCRs, similar (generalized) linear mixed models were used, but this time the targets' IBI response or SCR was entered as a fixed predictor instead of the stimulus intensity. For all (generalized) linear mixed models, fitting of random- and fixed-effects structure was carried out in the same way as for the single-trial behavioural data. To assess the robustness of the findings, skin conductance coupling was also analysed by calculating Spearman's correlation coefficients between targets' and observers' SCRs and comparing them between conditions using a linear mixed model. Correlations were calculated for the four time lags, and the time lag with the highest correlation coefficients across both conditions was chosen for the linear mixed model. For the robustness analysis of the IBI coupling, the Spearman's correlation between the targets' and the observers' continuous IBI over the whole task was calculated. For this, IBI data were smoothed with a moving average function of 2 seconds to reduce the influence of strong outliers. The correlation coefficients were calculated for 20 different time lags (steps of 0.5 s) between target and observer data. For testing condition effects, the lag with the highest correlation over all conditions was used.

Correlation coefficients were transformed using the Fisher's z-transformation to obtain normally distributed data. They were then compared between conditions using linear mixed models with correlation coefficients from different conditions (Level 1) nested in observers (Level 2) and targets (Level 3). The results of these analyses are reported in the results section when they diverge from the results of the single-trial analyses.

Control analysis of target expressivity

Empathic accuracy depends on the expressivity of the other (Zaki et al., 2008), and differences between direct and mediated interactions might result from altered expressivity of the targets in either condition. To test whether the targets show systematic differences in their pain expression between the conditions, a control experiment with a different sample was conducted. Thirty-one participants (25 females, 6 males, mean age(SD) = 23.51(4.70)) were shown 100 segments from the video recordings of both direct and mediated interaction without being aware of that manipulation. Video segments included four seconds before and four seconds after an electric shock and were chosen such that there was an equal number of videos from each original session, condition and target pain rating (summarized in 5 bins of 20 rating points each). Videos were shown in random order. After each video the participants had 10 seconds to rate how painful the stimulus was for the target in the video on a visual analogue scale ranging from "not painful at all" to "extremely painful". If targets expressed their pain differently in the two conditions, we would expect condition differences in the mean pain ratings or in the empathic accuracy of the control participants. Mean pain ratings and mean empathic accuracy scores (Spearman's correlations) were then compared between the conditions using t-tests.

Pre-registration and data availability

A pilot study using a similar design was pre-registered at OSF: https://osf.io/gcyqs. Behavioural, EEG and physiological raw data and main analysis code are available at: https://osf.io/pqmra/?view_only=df6b8e7cd19743d481d86ef7cb83cb83. Further data and code are available upon request from the first author.

Results

Empathic accuracy and unpleasantness ratings

Data from 30 dyads were included in these analyses. The best generalized linear mixed model for empathic accuracy had a random-effects structure containing random slopes for *condition* and *intensity* (AIC difference to the next best model during fitting of random effects: -63). Moreover, it contained a fixed effect of *intensity* (b(SE) = 0.07(0.01), z = 10.24, p < 0.0001, AIC difference to a model without the fixed effect of intensity: -14). This shows that the intensity of the shocks received by the targets predicted the observers' pain ratings (Fig. 2A & B), indicating meaningful empathic accuracy. However, this effect did not differ between conditions.

The best generalized linear mixed model for unpleasantness ratings had a random-effects structure containing random slopes for the interaction between *condition* and *intensity* (AIC difference to the next best model during fitting of random effects: -29). It contained a fixed effect of *intensity* (b(SE) = 0.07(0.01), z = 8.07, p < 0.0001) and a fixed effect of *condition* (b(SE) = -0.14(0.05), z = -2.63, p = 0.009, AIC difference to a model without the fixed effect of *condition*: -4). This shows that the intensity of the shocks received by the targets predicted the observers' unpleasantness ratings (Fig. 2C), but equally so in both conditions. However, the observers rated the pain of the target as slightly more unpleasant in the direct than in the mediated interaction (mean difference (SD) = 1.47(6.45)).

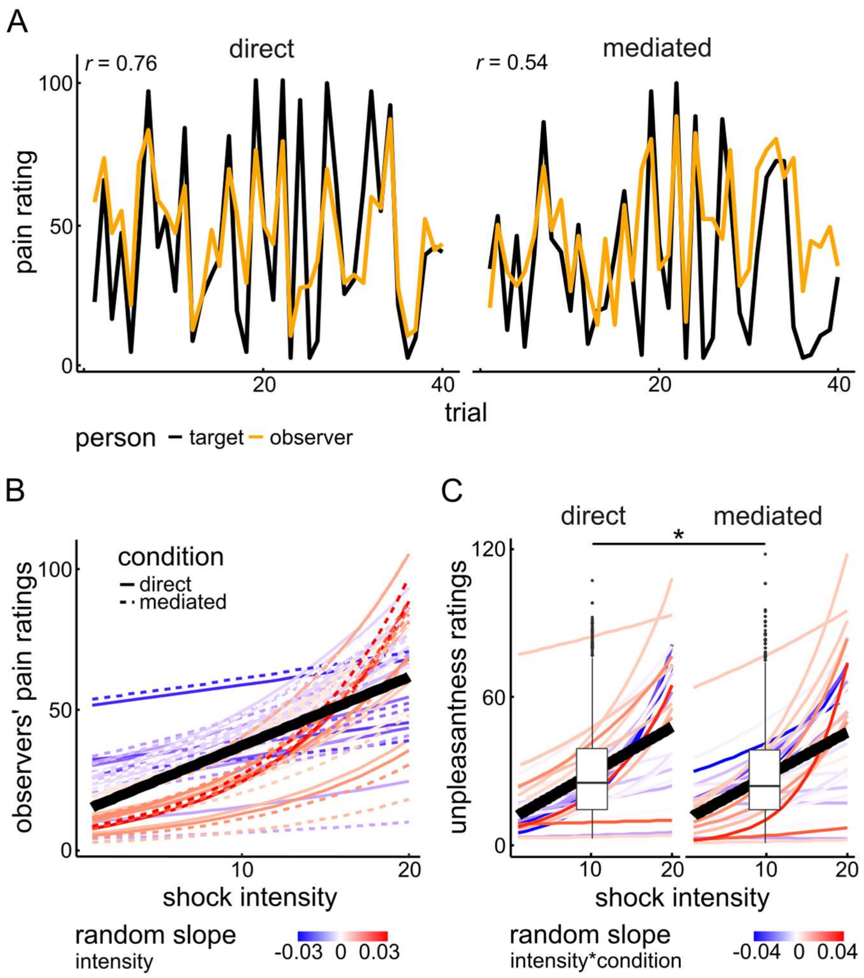

Figure 2. (A) Example pain rating data over trials from one randomly chosen sample dyad. (B) Predicted observer pain ratings for direct- and mediated-interaction conditions. The black thick line represents the fixed effect of shock intensity; single coloured lines represent predicted data from single participants. The colour shading from blue to red represents the value of the random slope for intensity of each participant, and the solid and dotted lines show predicted ratings for direct and mediated condition, respectively. (C) Predicted observer valence ratings in direct and mediated interaction. The black thick line represents the fixed effect of shock intensity; single coloured lines represent predicted data from single participants. The colour shading from blue to red represents the value of the random slope for the interaction between intensity and condition of each participant. The boxplots represent summary statistics for each condition. * = $p < 0$.

Mu suppression

Data from 29 dyads were included in these analyses. The analyses of peak mu suppression in the "direct" and "mediated interaction" conditions showed no effect of *intensity* or *condition,* nor an interaction that was significant after maximum value correction (see Fig. 3A & 3B middle and right). This shows that mu suppression was not sensitive to others' pain intensity in either condition. Similarly, analysing averaged power over the canonical mu band (8–13 Hz) yielded no significant effect of either factor (*intensity*: b(SE) = 0.34(0.30), t(df) = 1.13(87), p = 0.26; *condition*: b(SE) = -0.04(0.30), t(df) = -0.14(87), p = 0.88; *interaction intensity*condition*: b(SE) = -0.02(0.42), t(df) = -0.04(87), p = 0.97).

However, peak mu significantly differed between pain levels when participants experienced pain themselves (Fig. 3A left).

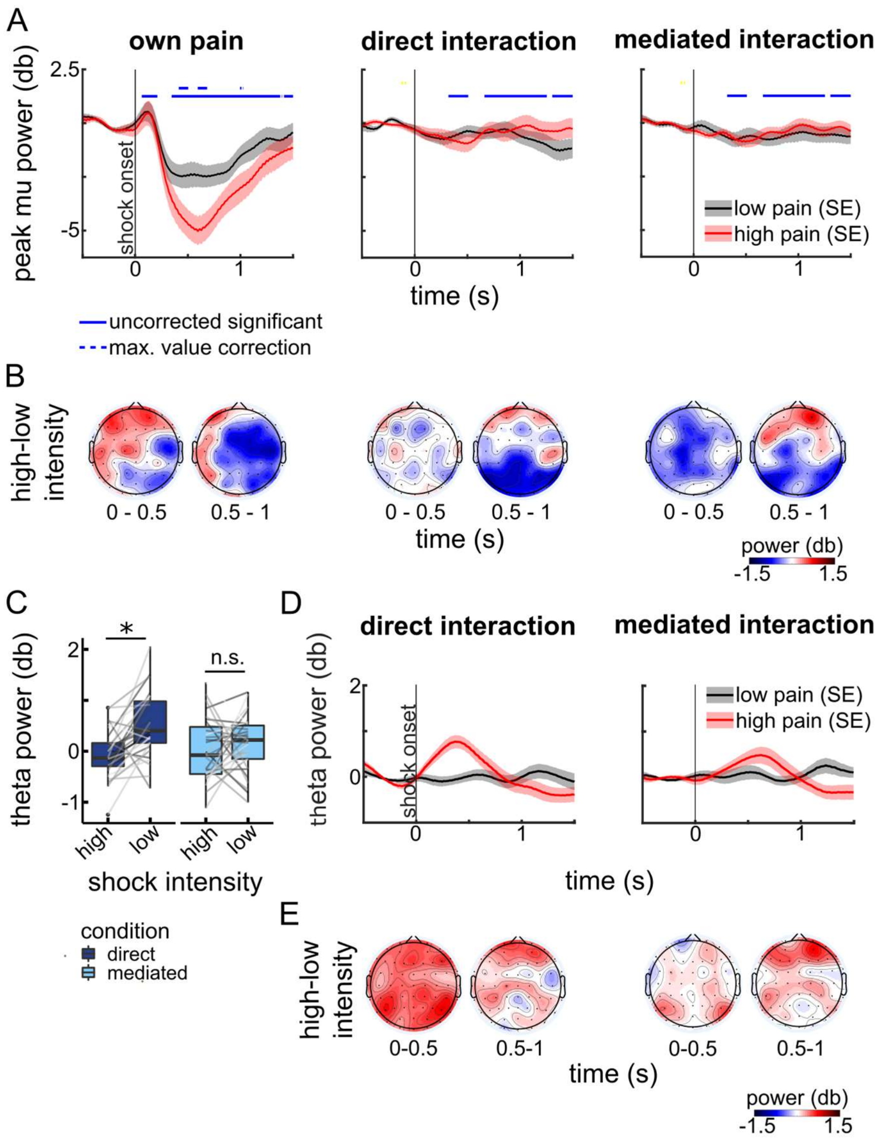

Figure 3. (A) Shown is peak mu power averaged over participants, dichotomized into low- and high-intensity shocks for display purposes only. Blue solid lines indicate time windows where the main effect of intensity reached significance (uncorrected level); blue dotted lines indicate time windows where the intensity effect survived maximum value correction. The main effect of condition and the interaction of condition x intensity did not survive maximum value correction, at any time-point. On the left side, the clear effect of pain intensity on mu power in the own-pain condition can be assessed. (B) Shown is the topography of averaged peak mu power differences between high- and low-intensity shocks in the three conditions. (C) Shown are boxplots for theta power averaged over 4–8 Hz and 0–500 ms after shock onset at electrode Fz for the direct and mediated conditions. Grey lines depict means from single participants. Significance asterisks refer to post-hoc tests from linear mixed models. * = $p < 0.001$. (D) Shown is the time course of averaged theta power (4–8 Hz) after stimulus onset. (E) Shown is the topography of averaged theta power differences between high- and low-intensity shocks in the two conditions.

Theta band

Data from 30 dyads were included in these analyses. The best-fitting linear mixed model for the power averaged over the canonical theta band (4–8 Hz) yielded a significant main effect of *intensity* (b(SE) = 0.57(0.14), t(df) = 3.99(90), $p < 0.001$) and a significant interaction between *condition* and *intensity* (b(SE) = -0.46(0.20), t(df) = -2.30(90), $p = 0.024$, AIC difference to next best model: 2.9). Follow-up models on the interaction showed a significant effect of *intensity* in the direct condition (b(SE) = 0.57(0.13), t(df) = 4.46(28.99), $p < 0.001$), but not in the mediated condition (b(SE) = 0.10(0.15), t(df) = 0.70(28.99), $p = 0.49$). This indicated that frontal theta was more responsive to the other's shock intensity in the direct condition than in the mediated condition (see Fig. 3C, D, E).

Exploratory analyses of whole time-frequency-electrode space

Data from 30 dyads were included in these analyses. During both direct and mediated interaction, others' pain *intensity* was positively related to 2 to 6 Hz power between 56 and 840 ms at the frontal and central electrodes (cluster 1, maximal at F4, see Fig. 4A left, B and E top left). Others' pain *intensity* was also negatively related to 12- to 20-Hz power between 680 and 1040 ms over parietal and occipital electrodes (cluster 2, maximal at P2, see Fig. 4A left, B and E bottom left).

Neither the permutation test on the *condition* effect (mediated vs. direct) nor the interaction between *condition* and *intensity* yielded any cluster that survived the cluster size correction or the maximum value correction. However, due to the exploratory nature of the analyses, we further examined the biggest cluster, which was significant on an uncorrected level. In direct compared to mediated interactions (main effect of condition; see Fig. 4A middle, C and E middle), theta/alpha (5–12 Hz) power at the frontal electrodes and lower beta (13–20 Hz) power at the frontal, central and centro-parietal electrodes was enhanced in an early time window (-256–644 ms). In a later time window (511–1500 ms), alpha/lower beta (8–20 Hz) power at the centro-parietal electrodes was enhanced during direct interactions. For the interaction between *condition* and *intensity*, the biggest uncorrected significant cluster showed a stronger positive effect of *intensity* in the "direct" than in the "mediated" condition. This cluster spanned 3–20 Hz between -220 and 1380 ms and included most left-hemisphere and central electrodes (see Fig. 4A right, D). Its time course is displayed separately for low (3–9 Hz, electrode with maximal interaction: Cz, Fig. 4E top right) and high frequencies (10–20 Hz, electrode with maximal interaction: CP1, Fig. 4E bottom right).

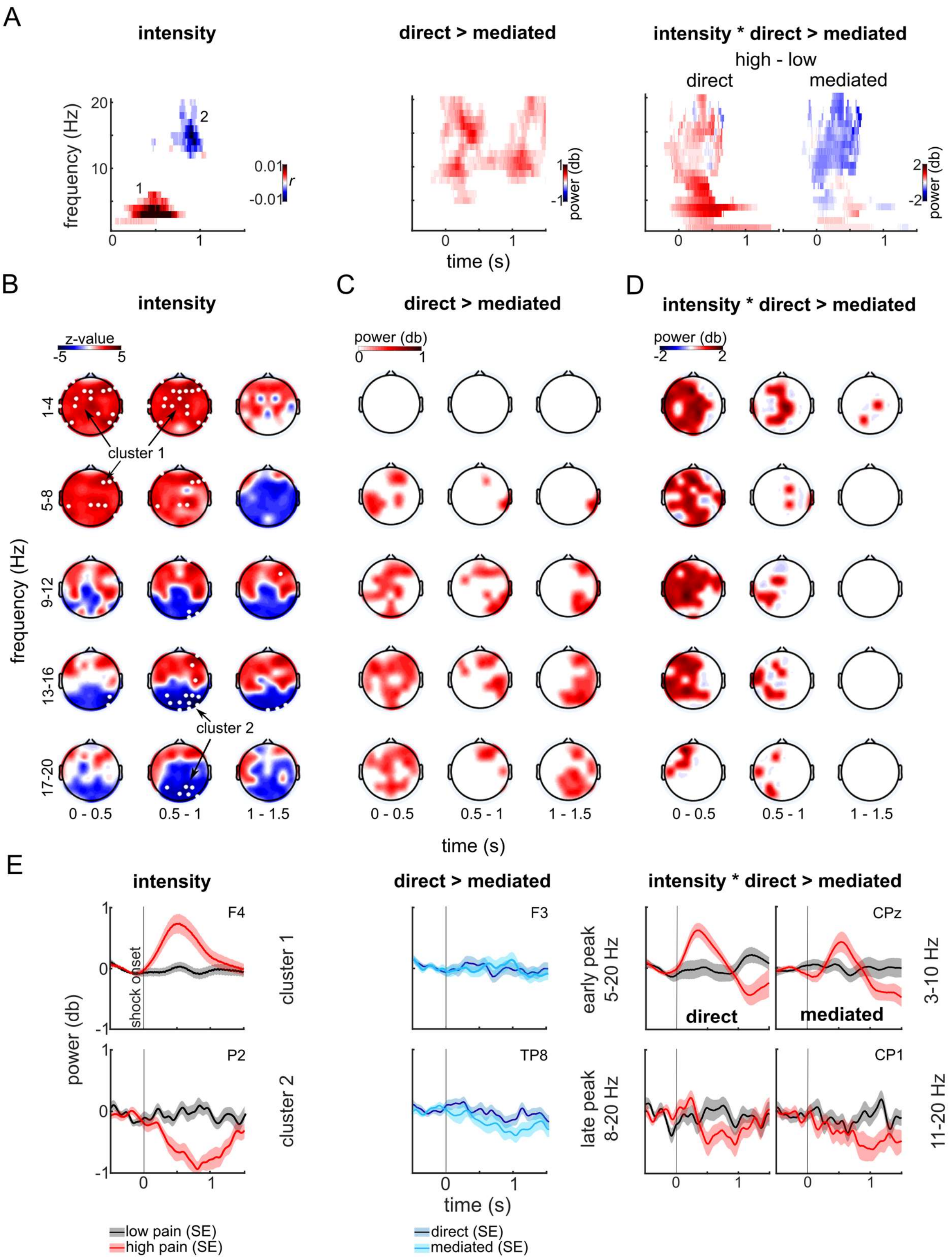

Figure 4: (A) Clusters found in the permutation tests on the correlation between shock intensity and EEG power across both conditions (left), on the main effect of condition (middle) and on the interaction between shock intensity

(dichotomized for display purposes only) and condition (right), averaged over electrodes. (B) Results of the permutation test on the correlation between shock intensity and EEG power across direct and mediated conditions. Data points that were significant after cluster size correction are displayed in colour; data points that were significant after maximum value correction are marked in white. (C) Results of the permutation tests on the condition effect. Data points of the largest cluster (uncorrected significant) are displayed in colour. (D) Results of the permutation tests on the interaction between condition and intensity (dichotomized for display purposes only). Data points of the largest cluster (uncorrected significant) are displayed in colour. (E) Power time course of cluster 1 (top left) and cluster 2 (bottom left), and power time course for the early cluster (5–20 Hz, F3, middle top) and the late cluster (8–20 Hz, TP8, middle bottom) showing a condition effect and power time course of lower frequencies (3–9 Hz; top, right), and higher frequencies (10–20 Hz; bottom, right) for interaction effects between condition and intensity. Data are dichotomized into low and high intensities for display purposes only.

Condition effects on observers' physiological responses

The best single-trial linear mixed model testing the effect of "direct" vs. "mediated" condition on observers' IBI responses to the observed shocks contained random slopes for *condition* and *intensity* but no fixed effects. These results indicate that observers' IBI responses were not sensitive to the observed shock intensity on the sample level (see Fig. 5A).

Observers' SCRs were greatest in the time window from 2 to 5 seconds after shock onset, hence this time window was used for all further analyses. The best single-trial linear mixed model on observers' SCR to the observed shocks contained random slopes for *condition* and *intensity*, a non-significant fixed effect of *condition* and a fixed positive effect of *intensity*. These results indicate that observers' SCRs were sensitive to the shock intensity, but equally so in direct and mediated conditions (Fig. 5C and 5D). All model parameters are listed in Table 1. Models with dichotomized intensities yielded the same results.

Condition effects on physiological coupling

The best single-trial linear mixed model testing effects of "direct" vs. "mediated" interaction on IBI coupling – predicting observers' IBI responses from targets' IBI responses – contained a random slope for *condition* and a fixed but not significant effect of *target IBI*. These results indicate that there was only minimal coupling between targets' and observers' IBI responses, which did not differ between conditions (Fig. 5B). Comparing the correlations between the continuous IBI traces in the two conditions showed the same results (correlations were highest for a lag of 2 seconds).

The best single-trial linear mixed model on SCR coupling – predicting observers' SCR from targets' SCR – contained a random slope for *condition* and *target SCR* and fixed effects of *target SCR*, *condition* and their interaction (for model parameters, see Table 1). Follow-up models on the interaction between *condition* and *target SCR* showed a significant positive effect of *target SCR* on observers' SCR in the "direct" condition (b(SE) = 0.12(0.03), t = 4.15, p < 0.0001), but not in the "mediated" condition (b(SE) = 0.03(0.02), t = 1.64, p = 0.1). When comparing the correlation coefficients of targets' and observers' SCRs between conditions, there was only a marginally significant effect of *condition* (b(SE) = -0.07(0.04), t(df) = -1.81(26), p = 0.082). These results indicate that coupling between targets' and observers' SCRs was greater in the direct than the mediated interaction, but the effect was rather small (see Fig. 5E).

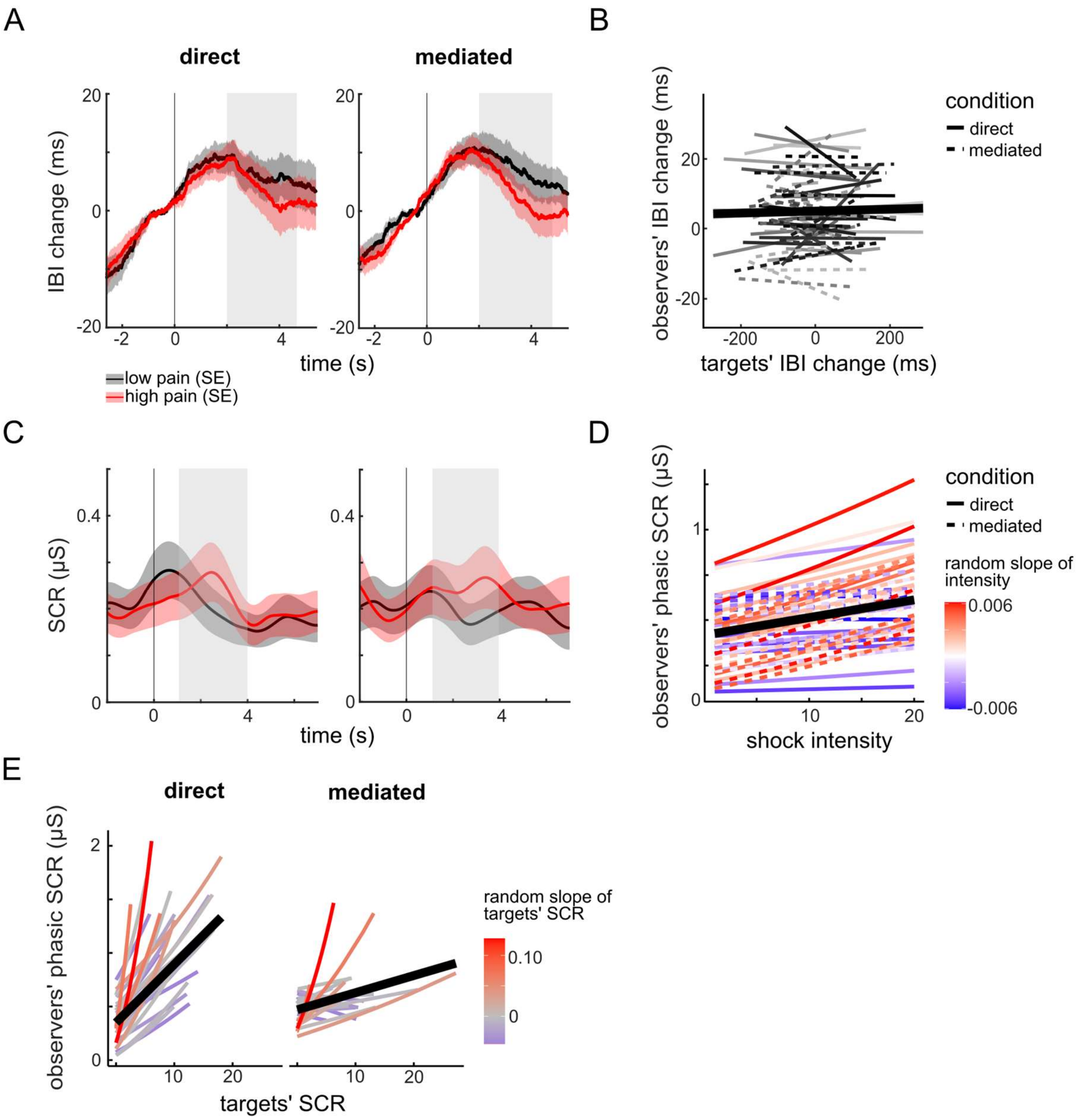

Figure 5: (A) Grand averages of observers' IBI responses in direct and mediated interactions, dichotomized into low- and high-intensity trials for display purposes only. (B) Observer IBI responses predicted from the single-trial model on physiological coupling. The thick black line represents the fixed effect of target IBI; the thin lines represent predicted data for single participants. (C) Grand averages of observers' SCRs in direct and mediated interactions, dichotomized into low- and high-intensity trials for display purposes only. (D) Observer SCRs predicted from shock intensity in the generalized linear mixed model. The black thick line represents the fixed effect of intensity; the

coloured lines display predicted values for single participants. The colour shading from blue to red represents the value of the random slopes of shock intensity for each participant. Solid and dotted lines show predicted values for direct and mediated conditions, respectively. (E) Observer SCRs predicted from target SCRs in the generalized linear mixed model on physiological coupling. The black thick line represents the fixed effect of targets' SCRs; the coloured lines display predicted values for single participants. The colour shading from blue to red represents the value of the random slopes of shock intensity for each participant.

Control analysis: Target expressivity

The means of the pain ratings from the video control experiment did not differ significantly between conditions (mean difference = 0.79, $t(30) = 1.66$, $p = 0.11$, Cohen's d = 0.3). The mean Spearman's correlations between video observers' ratings and shock intensity also did not differ between videos from direct and mediated interaction ($mean_{direct}$(SD) = 0.36(0.16), $mean_{mediated}$(SD) = 0.32(0.13), mean difference = 0.040, $t(30) = 1.36$, $p = 0.18$). These results indicate that the targets did not express their pain significantly differently in the direct versus mediated interaction.

Discussion

Although mediated social interactions through video calls are becoming the new norm, the impacts on understanding others and their feelings have not yet been researched thoroughly (Grondin et al., 2019), especially in social neuroscience. In the current study, we explored how a video-mediated interaction affects empathy for pain on behavioural, physiological and neural levels. We expected that less availability of social cues in a mediated interaction would hamper empathizing with the other. However, we found that observers were just as accurate in judging the other's pain in the mediated interaction as in the direct interaction. Moreover, participants experienced the other's suffering as only slightly less unpleasant in the mediated interaction. On the neural level, mu suppression over the somatosensory cortex was not sensitive to the other's pain in either condition. However, mid-frontal theta tracked the other's pain intensity more in the direct than in the mediated interaction. Exploratory analyses of the whole time-frequency-electrode space showed no additional differences between direct and mediated conditions after correcting for multiple comparisons. On a physiological level, observers' SCRs were coupled to targets' SCRs to a stronger degree in the direct compared to the mediated interaction. In sum, behavioural empathy was not reduced in the mediated interaction, whereas some neural and physiological aspects of empathy were dampened.

Effects of social presence on behavioural aspects of empathy for pain

Surprisingly, among the many studies on empathy for pain, hardly any measured empathic accuracy, and none explored which type of information is necessary or sufficient to judge others' pain accurately (Gauthier et al., 2008; Laursen et al., 2014; Leonard et al., 2013). In story-based empathy studies, empathic accuracy for emotion was reduced when auditory linguistic information was completely removed, whereas missing visual information did not impact empathic accuracy (Jospe et al., 2020; Zaki et al., 2009a). Similarly, we reveal that participants could judge the targets' pain quite well, and this ability did not decline in the mediated interaction. In contrast to the story-based paradigm, our results imply that visual information (apparent in both the direct interaction and the video calls) is sufficient for empathic accuracy for others' pain. As our participants did not experience severe pain (expressed by moaning or crying), auditory information might have been less important than for example in empathic responses to the pain of hospital patients (Agahi & Wanic, 2020). As our control analysis showed no condition differences in target expressivity, we can be assured that these did not mask true condition differences in empathic accuracy.

Although observers showed more affective empathy in the direct than in the mediated condition, the effect size was so small that it was practically negligible. One reason for this might be that many of our participants remembered their own recent pain experience and so were empathic to the similar experience of another. This was also stated by many in the debriefing questionnaires. This strategy might have led to imagination of others' pain independent of the medium, causing similar affective empathy (Goubert et al., 2005).

Effects of social presence on neural aspects of empathy for pain

As most former studies on empathy for pain used abstract cues or static pictures, we expected to find even stronger mu suppression in our paradigm using real stimuli and focusing on individual peak-mu frequency (i.e., Perry et al., 2010a; Riečanský & Lamm, 2019; Zebarjadi et al., 2021). Instead, we did not find any mu suppression in response to others' pain. Speculatively, mu suppression is a compensatory mechanism that aids empathy for pain via somatosensory representation of others' pain if insufficient sensory cues are available. Alternatively, it may be a weak signal that requires many repetitions – and stronger pain signals – to find a significant effect. However, if mu suppression is a general empathy mechanism, our analysis should have been sensitive enough to detect it. Therefore, our null findings on mu suppression align with recent criticism of its robustness and validity as a mechanism underlying empathy in general (Hobson & Bishop, 2016).

The mid-frontal theta/delta response constitutes another neural component of empathy for pain that has so far been rarely examined in EEG studies (but see Mu et al., 2008; Peng et al., 2021). Mid-frontal theta has been related to the salience, unexpectedness and aversiveness of many different types of stimuli (Cavanagh & Shackman, 2015; González-Roldan et al., 2011; Güntekin & Başar, 2014). Therefore, the heightened sensitivity of theta to the other's pain level might indicate that the other's pain elicits more arousal and negative affect in the direct interaction (Balconi et al., 2009), although this did not result in measurable behavioural differences. Mid-frontal theta might stem from the ACC, which shows reliable activity to both own and others' pain in fMRI studies (Cavanagh & Frank, 2014; Fallon et al., 2020). Confirming this assumption with source analyses was beyond the scope of this paper but could be an important step in future studies. The exploratory single-trial permutation test confirmed on a trend level the interaction between others' pain intensity and direct versus mediated interaction.

Lastly, our exploratory analysis revealed stronger parietal beta suppression relating to stronger observed pain irrespective of the condition. Parietal beta decrease has been linked to attention to affective touch (von Mohr et al., 2018). Future EEG studies should clarify its role in empathy for pain.

Effects of social presence on physiological coupling

Observers' cardiac activity was not sensitive to others' pain and showed no coupling with targets' cardiac activity. In contrast, previous empathy studies found cardiac coupling in emotional empathy (Zerwas et al., 2021), especially when semantic and auditory information was missing (Jospe et al., 2020). One reason for these discrepancies might be that the shocks used in the current study elicited such strong cardiac responses in targets that they were not easily mimicked by observers' cardiac activity (Goldstein et al., 2017).

In contrast, we found coupling in skin conductance responses, and this was the one aspect of pain empathy that was markedly reduced in the mediated interaction. This indicates that the physiological-coupling component of empathy, specifically in SCR, might rely on physical proximity (Chatel-Goldman et al., 2014; Murata et al., 2020) and possibly olfactory cues that are missing in mediated interactions (Calvi et al., 2020; de Groot et al., 2014).

Limitations

The main limitation of this study is the small sample size, which was due to the complexity of the design. This is especially prominent when reporting mostly null results, as one might argue that our small sample size prevented us from detecting subtle effects of social presence. However, as power analyses showed, by analysing single trials and using a within-subject design, we had sufficient power to detect meaningful effects of social presence. Another limitation is the comparably low standardization of our laboratory task. Using a task with real live people, we aimed to capture real-life empathy for pain in the best way possible in the EEG laboratory. At the same time, by using a standardized pain-stimulation protocol, we maintained a high degree of standardization compared to studies using unstructured interactive paradigms (i.e., Levy et al., 2017). Our study therefore answers recent calls for more interactive and contextual experimental methods for researching social interaction (Dumas, 2011; Przyrembel et al., 2012; Sonkusare et al., 2019).

Conclusions and future directions

Many recent studies examined direct interactions between participants, claiming that this is necessary for understanding social cognition (Fan et al., 2021; Levy et al., 2021; Redcay & Schilbach, 2019). However, few studies have explicitly compared these new paradigms to similar tasks using mediated interactions (but see e.g. Hietanen et al., 2020). Hence, it remains unclear whether the degree of social presence affects social cognition and if these effects are due to the interactivity (here called "immediacy") or to the amount of information transferred and the shared physical space (called "intimacy") (Cui et al., 2013; Grondin et al., 2019). Therefore, by examining the impact of social presence on empathy for the first time, we add a potentially important dimension to the study of social cognition. We show that the effects are nuanced: Only the immediate mid-frontal theta response to others' pain, presumably relating to emotional arousal (Balconi et al., 2009), and SCR coupling were affected by the reduced intimacy. This could indicate that intimacy is especially important for more automatic, stimulus-driven empathy components. Future studies should address whether the immediacy within the interaction might have a stronger impact on all components of empathy (Hamilton & Lind, 2016; Hietanen et al., 2020).

To conclude, we do not find evidence that empathy for pain is markedly impaired in video-mediated interactions, although physiological and neural resonance with the other's pain was reduced, which implies that some level of synchronization with the other is impaired. This suggests that empathic abilities might be preserved in everyday mediated social interactions, which are becoming more common. By showing specific changes in empathy components in a mediated interaction, we start to fill the gap in knowledge about social presence in social neuroscience.

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Tables

Table 1: Results of the single-trial (generalized) linear mixed models on IBI responses and SCRs

model	random slopes	SD	fixed effects	b(SE)	t(df)	*p*	AIC Δ
observers' IBI							-13
(n=29)	intercept/obs	25.01	intercept	4.33(2.16)	2.00(27.6)	0.055	
	condition /obs	13.00	-				
	intensity/obs	0.80					
	intercept/targ	7.9					
	condition/targ	0.13					
	intensity/targ	0.36					
observers' IBI (coupling with target IBI)							-50
(n=29)	intercept/obs	24.38	intercept	5.50 (2.22)	2.47 (31.7)	<0.019	
	condition/obs	12.98	target IBI	0.03(0.02)	1.66 (4312.2)	0.096	
	intercept/targ	0.000002583					
	condition /targ	0.000003744					
observers' SCR							-6
	intercept/obs	0.16	intercept	0.35(0.04)	8.10	<0.0001	
(n=27)	condition/obs	0.21	condition	-0.02(0.06)	-0.32	0.74	
	intensity/obs	0.007	intensity	0.01(0.002)	3.24	0.001	
	intercept/targ	0.03					
	condition /targ	0.03					
	intensity/targ	0.0009					
observers' SCR (coupling with target SCR)							-28
	intercept/obs	0.10	intercept	0.17(0.05)	3.34	0.0008	
	condition /obs	0.19	target SCR	0.12(0.02)	5.85	<0.0001	

(n=27)	targ-SCR/obs	0.06	condition	0.18(0.07)	2.81	0.005
	intercept/targ	0.0000	target SCR * condition	-0.08(0.02)	-5.72	<0.0001
	condition/targ	0.05				
	targ SCR/targ	0.02				

Notes: *IBI = interbeat interval, SCR = skin conductance response, AIC Δ = difference in Akaike information criterion to the next best model. Models on IBI responses are linear mixed models, and models on SCRs are generalized linear mixed models (note that the latter do not provide degrees of freedom for fixed effects).*