A Refined Examination of the Facial Cues Contributing to Vicarious Effects on Self-Pain and Spinal Responses

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Abstract: Vicarious pain has been shown to enhance observers’ nociceptive reactivity and pain perception. We exposed healthy participants to specific parts of facial pain expressions in order to investigate which components are required to induce this modulation. We created 2 classes of stimuli: one containing the most useful information for identification of pain expressions (diagnostic) and one containing the least useful information (antidiagnostic). Twenty-eight normal volunteers received electrical stimulation of the sural nerve immediately after they viewed these stimuli. Subjective ratings (intensity and unpleasantness) as well as the nociceptive flexion reflex (NFR) evoked by the shock were recorded. Results show that diagnostic stimuli lead to higher subjective ratings of shock pain than the antidiagnostic stimuli, but the stimuli classes had no significant impact on the NFR. A control experiment showed that our facial stimuli were given very low valence and arousal ratings compared to stimuli previously used to demonstrate the effect of emotional pictures on pain. Thus, the results are unlikely to be explained by emotions felt by the observer and suggest a vicarious facilitation of supraspinal pain processing induced by key features underlying pain expressions recognition. Results provide further support to the perception-action model of empathy.

Perspective: This study demonstrates that visual features that are efficiently used for the recognition of pain expressions are sufficient to induce a vicarious facilitation of self-pain. Supraspinal pain responses were modulated by the informativeness of the areas of the pain expressions that participants viewed prior to the painful stimulations.

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Key words: Nociceptive flexion reflex (RIII reflex), facial expression, pain modulation, Bubbles technique, intensity, unpleasantness.

Beyond its sensory component, pain is a complex subjective and emotional experience. It is associated with the mobilization of resources for the protection of physical integrity by signaling a threat and evoking appropriate protective reactions. Many factors have been shown to influence the interpretation of the bottom-up signal originating from potential tissue damage. Attention, distraction, emotions, mood, expectation and anticipation can modulate the experience of pain and the corresponding brain activation.1 Several imaging studies have also demonstrated that brain activation produced by the perception of pain in others partly overlaps with the response to self-pain (reviewed in Jackson et al8,9; also see11). This vicarious pain priming effect is believed to reflect the automatic activation of a mental representation of pain and to constitute an adaptive response induced by the detection of a potential threat in one’s environment.40

Vicarious facilitation of self-pain and spinal motor responses (nociceptive flexion reflex [NFR]) induced by acute electrical stimulations has recently been examined in response to static images of limbs in nociceptive situations and to pictures of facial expressions of pain.16 In a separate study, we examined the spinal
(NFR), subjective (ratings), and expressive (corrugator contraction) responses to the painful shocks following dynamic clips of pain expressions. These studies confirmed the vicarious facilitation of pain responses when observing pain in others, although the magnitude of this effect was influenced by the pain process measured (spinal vs supraspinal), the information signaling pain in others (sensory vs emotional and static vs dynamic expression), and the dispositional empathy reported by the participants.

Facial expression of pain is the cue observers believe to be the most reliable when it comes to judging someone else’s pain, thus giving this output channel a special status in the pain communication process. The specific information subtending the recognition of a facial expression of pain has been explored using the Bubbles technique. Participants were shown randomly sampled regions of faces expressing pain, neutrality, or 1 of the 6 basic expressions of emotion (anger, disgust, fear, happy, sadness, and surprise) and were asked to identify the emotion. The correlation between performance accuracy and the location of the facial regions revealed on each pain trial showed which regions participants used to discriminate pain from the other facial emotions efficiently (see Fig 1).

In the present study, we investigated the vicarious pain modulation effect further using pain expression stimuli specifically showing or masking pain-discriminative facial features, as determined by our previous results. We masked our stimuli in order to minimize or maximize their “diagnosticity”: the quantity of discriminative information they revealed. We examined pain perception and the NFR of participants who viewed these partially masked expressions of pain in order to test whether the facial cues most important for the discrimination of the facial expression of pain (“diagnostic features”) from other facial expression of emotions were sufficient to modulate pain perception. Half of the stimuli were partly masked to reveal only the areas of the face that contributed most to the recognition of pain expressions whereas the other half showed only those associated with the lowest recognition accuracy (see Fig 2). Immediately after viewing a visual stimulus, participants received a brief electrical stimulation of the sural nerve and were asked to rate its intensity and unpleasantness. In a follow-up experiment, we measured the subjective valence and arousal perceived and induced by the diagnostic and antidiagnostic facial stimuli in order to assess the possibility that vicarious pain effects could reflect emotional modulation.

Methods

Participants

The target sample size of the study was set to 24 based on a previous study using a similar method. Thirty-seven Caucasian participants (15 males) between 19 and 32 years old (mean ± standard deviation [SD] = 24.12 ± 3.28) were recruited to participate in the study using advertisement posted around the campus of the Université de Montréal. All the participants were healthy and did not suffer from chronic pain or psychiatric or neurologic disorder. None of the subjects took any analgesic medication in the 24 hours preceding the experiment. Of the 37 subjects, 8 were excluded during the reflex assessment procedure (unstable or undetectable reflex response) and 5 more were excluded from the analysis of the NFR because of habituation observed during the experiment. The final sample therefore comprised 29 subjects for the analysis of pain ratings and 24 for the analysis of the NFR. All the experimental procedures used conformed to the standards set by the latest revision of the Declaration of Helsinki and were approved by the Research Ethics Board of the Research Centre of the Institut universitaire de gériatrie de Montréal. All the participants gave written informed consent,
acknowledging their right to withdraw from the experiment without prejudice, and received compensation of $15/hour for their travel expenses, time, and commitment.

**Apparatus**

A computer running E-Prime 1.1 (Psychology Software Tools, Inc, Sharpsburg, PA) controlled the presentation of the visual stimuli, triggered the electrical stimulations, and recorded the participants’ pain ratings (intensity and unpleasantness) after each shock. Electrical stimulations were produced by a Grass stimulator (model S48) and delivered by a bipolar stimulating electrode connected through an optical isolation unit. Physiological signals were amplified, filtered, and sampled at 1,000 Hz using a BIOPAC MP150 system (BIOPAC Systems, Inc, Goleta, CA).
Visual Stimuli

Facial expressions of 10 actors (5 males) were selected from a standardized and validated database based on the strong intensity and the high discriminability of the pain expression (ie, minimal confusion with other emotions). The stimuli were spatially aligned on the average coordinates of the eyes and nose, and the luminance was calibrated. The final grayscale stimuli had a resolution of 256 x 256 pixels and spanned about 5.7 x 5.7 degrees of visual angle. The final stimuli were empirically validated through participants’ rating of the intensity of the emotions displayed by each stimulus on continuous scales.

In a previous study by Roy et al, the Bubbles technique was applied in order to determine the information used to recognize pain expressions (vs 6 basic emotions and neutral expression). This technique has proven to be a valid and powerful research tool when it comes to revealing which parts of a visual stimulus are responsible for the measurable performance of observers in a specific categorization task. On every trial, Roy et al asked subjects to identify an emotion from small random samples of a facial expression stimulus displayed at different bands of spatial frequencies. Then, for each pixel of the image and for each spatial frequency band, the correlation between facial discrimination accuracy and the amount of facial information displayed was calculated. This allowed Roy et al to determine the contribution of each region of the face to pain expression recognition (see Fig 1).

For the present study, 2 sets of stimuli were created based on those above-mentioned results: the “diagnostic” masks showed the most useful information for the identification of the facial expression of pain (ie, the pixels associated with the greatest positive correlations; ie, above the 80th percentile) and the “antidiagnostic” masks showed the most useful information for the categorization of pain expressions (ie, the pixels associated with the greatest negative correlations; ie, under the 20th percentile). The stimuli creation procedure is illustrated in Fig 2.

Measures

Nociceptive Flexion Reflex

The NFR was elicited and measured using a standard procedure (as reviewed in Sandrini et al). Transcutaneous electrical stimulations were induced using a bipolar surface electrode placed on the skin of the left ankle over the retromalleolar path of the sural nerve. Stimulation consisted of ten 1-ms rectangular wave pulses given in 30 ms at 333 Hz. The NFR was recorded using 2 Ag-AgCl electrodes placed on the brevis head of the left biceps femoris above the popliteal fossa once the skin was cleaned with alcohol and gently abraded with NuPrep (Weaver and Co, Aurora, CO) to obtain an impedance <10 kΩ. A third electrode placed over the medial side of the tibial tuberosity served as the ground. Participants sat on an inclined chair with a pillow under their knees to ensure lower limb relaxation. The angle of the knee was maintained at 120 degrees. The NFR threshold was determined for each participant using a staircase method. Stimulations were delivered every 6 seconds with gradually increasing intensity until the stimulus intensity evoking a clear NFR was found. Stimulus intensity was then slowly decreased until the NFR completely disappeared. This increase-decrease cycle was repeated until a stable threshold was found (at least 3 times). Finally, series of stimulations were administered at 120% of the threshold to test the reflex stability and to ensure that the participant tolerated this intensity. Eight participants did not show a clear and reliable reflex (undetected or unstable) in this phase and were excluded from the study. The mean intensity of the shock used to induce the reflex in the other participants was 18.0 ± 8.0 mA.

Interpersonal Reactivity Index (IRI)

The IRI, a measure of empathy, was administered. This 28-item questionnaire includes 4 subscales: fantasy, perspective taking, empathic concern, and personal distress. This instrument was chosen for its known valid assessment of the different aspects of empathy.

Subjective Ratings

During the experiment, participants rated the perceived intensity and the unpleasantness of the electrical stimulations. Following each shock, a visual analog scale appeared on the screen. Using a computer mouse, participants were instructed to move the cursor on the screen up to the level corresponding to their experience. On the first scale, they rated the intensity of the sensation from 0 (no sensation) to 100 (pain threshold), and the intensity of pain from 100 to 200 (extremely intense pain). On the second scale, they rated the unpleasantness of the experience from 0 (not unpleasant at all) to 100 (extremely unpleasant).

Procedure

All the participants were provided with an overview of the procedure before they read and signed the consent form. Electrodes were installed and subjects were explained the rating scales with a written description of the intensity and unpleasantness dimensions. They were asked to stay still and quiet during the experiment. Participants were informed that the experiment comprised 4 parts. In the first part, the pain threshold was determined with stimulations of various intensities given every 6 seconds. The second part was the main experiment. Participants sat approximately 70 cm away from a computer monitor. All trials comprised the following sequence of events (Fig 3): “Ready?” was shown for 1 second and a fixation cross appeared at the center of the screen for a duration of 5 to 8 seconds; then, a stimulus displaying either the
diagnostic or the antidiagnostic regions of faces expressing pain was presented at the center of the screen for a duration of 1 second; immediately after, the screen became homogenously black and a 30-ms electrical stimulation was administered; this was followed by the intensity and unpleasantness visual analog scales appearing on the screen. In order to prevent habituation, we introduced some uncertainty relative to the intensity of the shock: the intensity of the stimulus was varied pseudo-randomly between high intensity (120% of the reflex threshold intensity) and low intensity (60% of the threshold). Participants were not told that there were only 2 stimulus intensities and were asked to rate the perceived intensity and unpleasantness of the electric shock as accurately as possible. In total, 90 electrical stimulations were administered, 60 of high intensity and 30 of low intensity, for an approximate duration of half an hour with a pause of 5 minutes in the middle. The 20 visual stimuli were pseudo-randomly presented 4 or 5 times to each participant. To ensure that participants were paying attention to the stimuli, the subjects were told that in the third part of the study they would take part in a recognition task to evaluate their recall of the visual stimuli. In the fourth part, participants were asked to categorize the valence (positive, negative, or neutral) of the 20 stimuli. Participants were then debriefed and thanked for their participation.

**Data Analysis**

**Nociceptive Flexion Reflex**

The magnitude of the NFR produced by the 60 high-intensity stimuli was scored following standard methods. The integral of the rectified electromyographic signal was calculated and the 90-ms prestimulation baseline was subtracted from the 90- to 180-ms poststimulation. The data were then transformed into z-scores within each participant to account for individual differences in the absolute magnitude of responses. Twenty-four of the 29 participants tested were included in the NFR analyses; 5 were excluded because of an important habituation effect during the experiment, resulting in the absence of the NFR in the majority of trials.

**Statistical Analysis**

The z-normalized NFR as well as the intensity and unpleasantness ratings were averaged within each participant for each condition (diagnostic and antidiagnostic). Note that NFR responses were obtained only at supra-threshold intensity (120% of NFR threshold), so only the high-intensity condition was considered for this variable. A paired t-test was used to compare the mean of each condition for NFR. The impact of both stimulation intensity and diagnosticity condition on the ratings were tested using 2 × 2 repeated measures analyses of variance with the shock intensity (60% or 120% of the NFR threshold) and stimuli condition (diagnostic or antidiagnostic) as the within-subject factors. Partial eta-squares (Ω²p) were calculated to evaluate the effect sizes. All analyses were performed with SPSS 16.0 (SPSS Inc, Chicago, IL).

**Analysis of Stimuli Valence Categorization**

Categorical ratings of valence obtained in the postexperimental phase were compiled across subjects for each stimulus category. The percentages of diagnostic and antidiagnostic stimuli assigned to the negative and positive valence categories were compared using chi-square.

**Postexperimental Assessment of the Valence and Arousal of the Visual Stimuli in an Independent Sample**

Following the results of the categorical valence assessment performed at the end of the main experiment, a second group of 10 healthy participants (5 men and 5 women; mean ± SD age = 22.5 ± 4.0) was recruited to document the affect associated with the masked pain expressions more precisely using parametric ratings of the perceived and felt valence and arousal (perceived: “rate the valence/arousal evoked by each image”; felt: “rate the valence/arousal you experience when looking at each image”). In order to compare the affect associated with our 20 stimuli (described in section “Visual Stimuli”) relative to that induced by the stimuli typically used in studies investigating the effects of emotions on pain, we also included the set of 8 positive, 8 negative, and 8 neutral images used by Rhudy et al, taken from the International Affective Picture System (IAPS). The full set of stimuli therefore included 2 stimulus sets (masked expressions and IAPS pictures) in the following categories: 1) diagnostic mask of pain face, 2) antidiagnostic mask of pain face, 3) IAPS–negative emotion, 4) IAPS–positive emotion, and 5) IAPS–neutral emotion. Stimuli were presented for 1 second as in the main experiment (but without electric shocks) and following a pseudo-random order balancing categories. Each stimulus was presented twice, in separate blocks with the block order counterbalanced across subjects. In half of the blocks, subjects were asked to provide ratings of
the valence and arousal depicted in each image (perceived valence and arousal). In the other half of the blocks, they were asked to indicate the emotional valence and arousal they subjectively felt while looking at each image (induced valence and arousal). Ratings were performed using an unbounded magnitude-estimation scale\(^7,35\) and without time pressure. Subjects were specifically told that they should use their own numerical scale, with the only constraint being that neutral should be given a value of 0 on the valence scale and that arousal could not be given negative values. This rating method was chosen because it is less vulnerable to ceiling effects and thereby ensures a greater sensitivity to differences in the relative level of valence and arousal of each category. In order to compare the diagnostic and antidiagnostic conditions of the present study to the negative and positive valence conditions of IAPS pictures, we performed 2-way repeated measures analyses of variance contrasting stimuli sets (masked faces vs IAPS stimuli) and categories (diagnostic masks and IAPS images with negative valence vs antidiagnostic masks and IAPS images with positive valence).

### Results

#### Subjective Ratings

Both effects of shock intensity and visual stimulus condition reached significance on shock-intensity ratings \(F[1, 28] = 61.7, P < .001, \eta^2_p = .7,\) and \(F[1, 28] = 329.4, P < .001, \eta^2_p = .9,\) respectively). There was also a significant interaction between the intensity of the electrical stimulation and the visual stimulus condition on perceived intensity \(F[1, 28] = 154.9, P < .001, \eta^2_p = .9.\) For the high-intensity stimulations, the diagnostic visual stimuli led to higher-intensity ratings than the antidiagnostic ones \(t[28] = 24.1, P < .001.\) The same effect was observed within the low shock intensity condition \(t[28] = 2.4, P = .022;\) but the amplitude of the difference was much smaller than for the high-intensity shocks.

There was a main effect of the shock intensity on shock-unpleasantness ratings \(F[1, 28] = 8.3, P = .000, \eta^2_p = .8\) with high-intensity stimulations being rated as more unpleasant. There was also a significant main effect of the visual stimulus conditions \(F[1, 28] = 5.4, P = .028, \eta^2_p = .2\) with the diagnostic visual condition leading to higher shock unpleasantness than the antidiagnostic condition (Fig 4B). The interaction between shock intensity and diagnosticity was not significant on unpleasantness \(F[1, 28] = .0, P = .997\).

No significant correlation was found between subscales of the IRI and changes in ratings of intensity or unpleasantness induced by the diagnostic vs the antidiagnostic stimuli.

#### Nociceptive Flexion Reflex

A paired t-test revealed that there was no significant difference \(t[23] = 1.4, P = .170\) between the z-scores mean of the diagnostic and antidiagnostic conditions for the painful (120% of the NFR threshold) trials although the means were in the expected direction (Fig 4C). No significant correlation was found between subscales of the IRI and the individual difference in the amplitude of the NFR induced by the diagnostic vs the antidiagnostic stimuli.

#### Stimuli Valence

At the end of the experiment, participants judged the diagnostic stimuli as presenting a negative emotional valence in 93.7% of the trials (positive valence in 1.48% of the trials and neutral in 4.81%) whereas antidiagnostic stimuli were judged as expressing a positive emotional valence for 67.0% of the stimuli (15.19% negative and 17.78% neutral). The difference

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**Figure 4.** Effects of diagnosticity for the mean (SEM) subjective ratings (A, B) and NFR (C) for painful (120% of the NFR threshold) and nonpainful (60% of the NFR threshold) electrical stimulations. Significant contrasts between the diagnostic and antidiagnostic conditions: ***\(P \leq .05;\) ****\(P \leq .001.\) Note that error bars reflect between-subject variance and that statistical tests are based on within-subject effects between conditions (see text).
between the percentages of the diagnostic and antidiagnostic assigned to the positive versus negative valence categories was highly significant ($\chi^2[1, 479] = 321.4, P = .000$).

**Postexperimental Independent Assessment of Valence and Arousal**

Results of the parametric assessment of the valence and arousal associated with the diagnostic and antidiagnostic masks are reported in Table 1, along with those of the IAPS pictures previously used to test the effect of emotions on pain. The analyses of variance performed on the postexperimental ratings confirmed the differences between the diagnostic and antidiagnostic facial stimuli and the positive and negative IAPS pictures. The same significant effects and interactions were found on induced/felt and perceived emotions (valence and arousal). Only the statistical results on felt emotions are reported in detail here. The main effect of picture category (positive vs negative) reached significance (F[1, 9] = 145.2, $P < .001$, $\eta^2_p = .9$), whereas the main effect of stimuli set (masked pain faces vs IAPS pictures) was not significant (F[1, 9] = 2.1, $P = .184$). The differences in valence were consistently much greater between the negative and positive IAPS picture categories than the diagnostic and antidiagnostic images of pain expression (interaction between stimulus set and category: F[1, 9] = 15.0, $P = .004$, $\eta^2_p = .6$). All pairwise comparisons of felt valence reached significance (all $P$'s < .05), including the contrast between diagnostic and antidiagnostic stimuli (t[9] = 4.9, $P = .001$), thus confirming that the more negative emotions are associated with the diagnostic compared to the antidiagnostic stimuli. However, the diagnostic pain expression stimuli induced much less negative valence than the negative IAPS stimuli (t[9] = 2.5, $P = .035$), and the antidiagnostic pain expression stimuli induced much less positive valence than the positive IAPS stimuli (t[9] = 4.8, $P = .001$). A contrast between the difference in valence between diagnostic and antidiagnostic stimuli ($\Delta$ masked expression = diagnostic – antidiagnostic) versus the negative and positive IAPS pictures ($\Delta$ IAPS = negative – positive) revealed a significant effect (t[9] = −3.87, $P = .004$), which is consistent with the finding of a much larger effect of IAPS (2.3 SD) than pain faces stimuli (1.0 SD) on valence (see Table 1).

Arousal also differed significantly between stimuli sets, with much higher values observed for the IAPS pictures than for the masked pain expressions (F[1, 9] = 23.1, $P = .001$, $\eta^2_p = .7$). No significant arousal difference was found between diagnostic and antidiagnostic masks of pain expressions or between negative and positive IAPS pictures (main effect of stimuli valence: F[1, 9] = .2, $P = .7$). The interaction was not significant (F[1, 9] = 4.0, $P = .08$).

**Discussion**

Showing parts of pain expressions that vary in their diagnosticity was sufficient to modulate the supraspinal

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**Table 1.** Mean (SD) Normalized Ratings (z-Scores) of Perceived and Felt Valence and Arousal Reported in Response to the Facial Stimuli Showing Pain Diagnostic and Antidiagnostic Information, and to Emotional Pictures (IAPS) Previously Used to Show an Effect of Emotion on Pain

<table>
<thead>
<tr>
<th>Stimulus Type</th>
<th>Perceived Valence</th>
<th>Felt Valence</th>
<th>Perceived Arousal</th>
<th>Felt Arousal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Masked expression</td>
<td>0.45 (0.32)</td>
<td>0.33 (0.32)</td>
<td>0.47 (0.64)</td>
<td>0.99 (0.64)</td>
</tr>
<tr>
<td>Antidiagnostic</td>
<td>0.45 (0.32)</td>
<td>0.33 (0.32)</td>
<td>0.47 (0.64)</td>
<td>0.99 (0.64)</td>
</tr>
<tr>
<td>Diagnostic</td>
<td>0.45 (0.32)</td>
<td>0.33 (0.32)</td>
<td>0.47 (0.64)</td>
<td>0.99 (0.64)</td>
</tr>
</tbody>
</table>

*Note: IAPS pictures selected based on Rhudy et al. 25"
processing of pain. When participants saw facial expressions of pain masked in order to reveal only the 20% most useful information for pain recognition, they rated the sensation induced by an electrical stimulation of the sural nerve as more intense and more unpleasant than while looking at the same face but masked to show only the 20% least diagnostic pixels to recognize pain (antidiagnostic). For the intensity ratings, the effect was not specific to painful electrical stimulation, but the magnitude of the effect was larger in response to high-intensity (120%) than to low-intensity (60%) shocks. Finally, the NFR was not modulated significantly.

The same participants judged that the diagnostic masked faces were expressing a negative emotion, whereas the antidiagnostic masked faces were expressing a positive emotion. To further investigate the properties of these visual stimuli, another experiment was performed with different observers to compare the valence and arousal expressed and induced by our stimuli with stimuli typically used in emotional modulation experiments (IAPS stimuli). The difference between the diagnostic and antidiagnostic stimuli ratings was significant, but much smaller than the one between negative and positive IAPS pictures. For the arousal ratings, there was a difference only between the 2 stimuli groups, the IAPS stimuli leading to much higher arousal induction and perception.

**Potential Mechanisms: Is It Emotion? Attention?**

There are several potential mechanisms that might contribute to the observed effects. One possible explanation relies on the well-known effect of emotions on pain. Rhudy and colleagues26 have demonstrated that the valence of pictures used to modulate pain explains the direction of the modulation (negative valence enhancing pain responses and positive valence inhibiting pain responses), whereas the arousal of the same stimuli explains the amplitude of the modulation. (See an independent confirmation of the interaction between valence and arousal on pain and NFR modulation in Roy et al.28) Here, the facial stimuli showing the diagnostic information to recognize pain expression were rated as more negative than those showing the antidiagnostic information, which were perceived as positive. This is consistent with the emotion modulation model. However, in the postexperimental control experiment, participants rated our stimuli as expressing and inducing a significantly smaller valence and much less arousal activation than typical IAPS stimuli used for emotional modulation experiments. Therefore, the emotional induction produced by our stimuli was weaker than that typically produced in previous emotional pain modulation studies.28,29 Furthermore, the effect size of the supraspinal modulation was comparable to the ones typically found in similar previous studies on emotions.25 Therefore, without a confirmation that our stimuli elicited strong emotions, it appears very unlikely that the present results only reflect the emotional modulation of pain.

Another result that contrasts with the usual effects produced by emotions on pain is the modulation found here on both the perceived intensity and unpleasantness of both painful and nonpainful shocks. This suggests a generalized influence on sensory processes rather than a more specific influence on pain perception or, more specifically, on pain affect. Typically, when both intensity and unpleasantness measures are taken, the emotional modulation of pain is much stronger in, or is specific to, unpleasantness ratings.22,23,38 Considering all of the above, the emotional modulation explanation appears insufficient to explain the supraspinal modulation found here.

Directing attention away from pain is another very robust way to produce analgesic effects.17 Typically, these effects are found on intensity ratings and secondarily on unpleasantness.37,38 However, one would expect that negatively valenced pain expressions (diagnostic condition) might have a stronger distracting effect on shock pain. Here, the observed effects are in the opposite direction, so a simple distraction effect is unlikely.

**The Vicarious Modulation of Pain**

Besides emotion and attention, a third potential mechanism is provided by the perception-action model of empathy.19 This model postulates that the observation of actions or states (including pain; see Jackson et al8) activates the same neural structures implicated in the first-person experience. The pain communication mechanism is a complex phenomenon and includes pain expression, pain recognition, and, ultimately—according to the perception-action model—the mapping of the perceived expression on the observer’s own neural representations. It seems plausible that the resonance of pain communication on the observer’s own neural system induces a priming of the pain responses for the diagnostically masked stimuli.

The present results are congruent with a previous study demonstrating very convincingly that emotion modulation is insufficient to account for some vicarious pain facilitation effects. In this study, viewing images showing somatic cues of human pain, compared to aversive pictures with equivalent negative valence and arousal but without pain-evocative content, produced increases in both pain and in the late somatosensory brain evoked-potentials elicited by painful shocks.5 As previous studies examining the effects of negative emotions on pain have generally included images with and without pain-related content,29 it is possible that the effects associated with negative emotions in these studies might have been driven largely by vicarious pain processes produced by the subset of pain-related images. Consistent with this, Godinho5 suggested that the observer’s representation of someone else’s suffering might act through an automatic empathy-induced activation of pain circuitries that facilitate self-pain.

Theoretical accounts of empathy based on the perception-action model generally include higher-order processes regulating self-other assimilation to allow...
for a more detached and adaptive response of the observer.\(^\text{6,19}\) Consistent with this, 2 previous studies conducted in our laboratory using a similar methodology demonstrated that the magnitude of pain facilitation effects assessed by self-ratings was reduced in individuals scoring higher on trait empathy scales.\(^\text{16,36}\) This was attributed to the engagement of higher-order empathic processes that might tune down the basic vicarious facilitation effects resulting from spontaneous self-other assimilation. However, the present study did not replicate this correlation between trait empathy, assessed using the IRI, and the modulation produced by the pain expressions on pain intensity and unpleasantness. The absence of such an association here could be due to the masking of the pain expressions, which likely directed higher-order cognitive resources toward the disambiguation of the expressions (ie, top-down regulation of perceptual processes) and may have thereby prevented the activation of self-regulatory empathic processes.

Finally, the perceived individual characteristics of the expresser (personality, trustworthiness, etc) may have a major impact on the pain response of the observer. Indeed, in addition to the vicarious facilitation generally produced by pain expression, Loggia et al\(^\text{15}\) showed an effect of the perceived characteristics of the expressers. In this study, participants experienced more pain while viewing an expresser toward whom they were led to experience compassion related to emotional suffering and independent from pain, as opposed to a negative socioaffective response (ie, antipathy) induced by the expressers’ description of a situation where he or she displayed socially reprehensible behavior. Here, we cannot exclude the possibility that masking of the faces might have affected these perceived individual characteristics of the expressers.

### Study Limitations

There are several issues raised by the present study in relation to prior literature that merit further investigation. First, we relied on a post hoc assessment of valence and arousal to show that the present stimuli elicited much weaker valence and arousal than the stimuli typically used to induce emotions. Ideally, a more direct comparison of the IAPS and the present masked facial expressions of pain would be necessary to determine the extent to which the emotional induction explanation could be discarded (ie, comparison of stimuli with same valence and arousal ratings). More importantly, pain expressions should be compared to other negative emotional faces with comparable valence and arousal (eg, fear, sadness, disgust) to test for the specificity of pain-related processes. Second, contrary to our expectations, the vicarious facilitation of the NFR was not confirmed here. Another study using unmasked stimuli failed to show a robust modulation of the NFR by facial expressions of pain, so it seems unlikely that this absence of spinal modulation is due to the visual masking.\(^\text{36}\) In contrast, the modulation of the NFR was robust in a more recent study using dynamic expressions.\(^\text{16}\) Therefore, there might be a necessary contribution of dynamic visual information to the priming of spinal responses. Some authors suggested that brain structures involved in facial expression processing show enhanced activation to dynamic compared to static facial expressions.\(^\text{10,32}\) The various conditions under which vicarious pain effects are induced must be further examined across a variety of communication conditions and pain measures to establish the minimal and the optimal conditions leading to self-pain modulation.

### Conclusion

The visual features used for the efficient recognition of pain expressions are sufficient to induce a vicarious facilitation of self-pain as shown by the higher reports of felt intensity and unpleasantness. This implies that pain communication may have an impact on the observer even when visual interference masks up to 80% of the face as long as key diagnostic information is available. The well-known emotion induction effect appears insufficient to explain the present modulation. Consistent with the perception-action model, these effects are thought to reflect a supraspinal vicarious priming of self-pain via the mapping of others’ pain states on the observer’s own pain system. This basic research on the key features of pain communication and the detailed assessment of the impact of those cues on the observer is fundamental to our understanding of pain communication and of its complex consequences.

### Acknowledgments

We thank Etienne Vachon-Presseau, Mathieu Roy, and Marianne Arsenault for their help in RIII measurements.

### References


