The role of inhibitory mechanisms in the regulation of facial expressiveness during pain

Anna J. Karmann *, Stefan Lautenbacher, Miriam Kunz

Physiological Psychology, Otto-Friedrich University Bamberg, Germany

A R T I C L E   I N F O

Article history:
Received 29 April 2014
Accepted 30 November 2014
Available online 8 December 2014

Keywords:
Facial expression
Inhibition
Pain
EMG
FACS

A B S T R A C T

Although it is assumed that inhibitory control plays a role in regulating the degree of facial expressiveness, so far the specific type of inhibitory mechanism involved has not been identified. The present study was designed to investigate the association between different types of inhibitory mechanisms and the degree of facial expressiveness.

Facial expressiveness during experimental pain was assessed using the Facial Action Coding System and facial electromyography (criterion variables). Different aspects of inhibitory functioning (Antisaccade task, Stroop task, questionnaire) were used as predictor variables.

The degree of facial expressiveness was significantly predicted by the performance in the Antisaccade, but not the Stroop task or the questionnaire. The higher the ability to inhibit saccadic eye movements, the lower was the degree of facial expressiveness.

This data suggests that the degree of facial expressiveness is not regulated by inhibitory control in general, but specifically depends on inhibitory mechanisms regulating automatic motor responses.

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1. Introduction

Facial expressions provide the possibility of conveying a large amount of information about inner affective states, thoughts and even motives of an individual to an observer (Craig, 2009; Craig, Prkachin, & Grunau, 2011; Ekman, 1999). Hence, they play an essential role in interpersonal relationships and social communication (Darwin, 1872; Halberstadt, Denham, & Dunsmore, 2001). In the context of distress (e.g., pain or fear), the function of facial expressions might even be fundamental. By signaling the experience to others, facial responses may elicit empathy and helping behavior, which may be crucial for survival (Botvinick et al., 2005; Williams, 2002).

Although facial expressions are known to be elicited rather automatically (Blair, 2003; Darwin, 1872), the degree to which inner affective states are actually displayed via the face varies substantially between individuals. Thus, even during strong emotional experiences, the degree of facial expressiveness ranges from stoicism, with almost no facial expressions shown, to high expressiveness (Gross & Levenson, 1993; Richards & Gross, 2000).

Although these variations have been reported solidly, so far little is known about what kind of mechanisms are responsible for regulating the degree of facial expressiveness.

Interestingly, a recent fMRI study of our group on the neural regulation of facial expressiveness (Kunz, Chen, Lautenbacher, Vachon-Presseau, & Rainville, 2011) has created results that point toward one possible underlying mechanism. Here, it was shown that the degree of facial expressiveness during painful heat stimulation was associated with the activity of fronto-striatal circuits. The higher the activation was in medial prefrontal areas and in the caudate nucleus, the less strongly individuals displayed their pain via the face. Since these areas are known to be involved in motor inhibition (Aron et al., 2007; Ridderinkhof, van den Wildenberg, Segalowitz, & Carter, 2004), the most reasonable interpretation seems to be that motor inhibition plays a major role in the (down-)regulation of facial expressiveness; with an active inhibition resulting in a reduced degree of facial expressiveness.

The relevance of inhibition for the regulation of facial expressiveness can also be derived from developmental observations. Young children seem to have unfiltered access to the full repertoire of facial activities early in life (Ekman, 1999), but just gradually learn to selectively control and adjust their facial expressiveness according to situational demands (Craig, Verslloo, Goubert, Vervoort, & Crombez, 2010; Izard, 1971). The fact that this acquired control is usually associated with a decrease in facial expressiveness (especially with regard to the display of negative affective...
states) (Izard, 1971) favors the idea of an active inhibition of facial responses. Thus, overtly displaying one's affective state seems to be the “default”, which is learnt to be inhibited throughout socialization.

In the current study we have now tried to elaborate these ideas further by investigating whether the degree to which an individual expresses his/her pain is associated with measures of inhibitory functioning. In other words, if the regulation of facial expressiveness was indeed strongly influenced by inhibitory processes, the degree of facial expressiveness should be associated with other indicators of inhibition.

Before addressing the association of inhibitory functioning and facial expressiveness, it seems reasonable to first differentiate between different types of inhibitory mechanisms that might be relevant in this context. A common differentiation is made between “behavioral” and “cognitive” inhibition (Harnishfeger, 1995; Nigg, 2000). While “cognitive inhibition” refers to the action of controlling mental processes like memory or attention, “behavioral inhibition” corresponds to the ability of controlling or inhibiting pre-potent behavioral motor responses or impulses. This “behavioral inhibition” seems to be the most relevant category in our context, given that facial expressions are behavioral motor responses (Craig et al., 2011) and thus, seem likely to be governed by behavioral/motor inhibitory mechanisms. In order to determine variables that are hypothetically more or less closely related to facial expressiveness, “behavioral inhibition” was assessed using different approaches. Firstly, the Antisaccade task (Hallett, 1978) was chosen to determine the ability to inhibit a response which is very similar to facial expressions. Namely, the Antisaccade task measures oculomotor inhibition of an automatic orientation response/reflex (Nigg, 2000). The Stroop task (Stroop, 1935) was selected as a second, more distantly related measure. Although involving motor inhibition as well, the Stroop task additionally demands the higher cognitive process of decoding the word meaning (Nigg, 2000). Due to its cognitive components and to ease further understanding, the Stroop task will further be referred to as an indicator of “behavioral and cognitive inhibition”. The choice of the Antisaccade and Stroop task was additionally founded in the fact that both represent well-established measures that indisputably belong to the battery commonly used to assess inhibitory functioning (Friedman & Miyake, 2004; Miyake et al., 2000). In order to assess “behavioral inhibition” on the level of dispositional personality variables and introduce this as a third, hypothetically most distantly related measure, the Impulsive Behavior Scale (UPPS; Whiteside & Lynam, 2001) was included in the current set of variables. The UPPS represents a well-elaborated tool for assessing impulsivity, which first established a subdivision of the trait into four distinct, but related factors. Its different dimensions have also been shown to be related to other indicators of inhibitory functioning (Gay, Rochat, Billieux, d’Acremont, & van der Linden, 2008; Roberts, Fillmore, & Milich, 2011). By precisely measuring all facets of impulsivity, this questionnaire, thus, seems to be best suited to identify a possible relationship between impulsivity and facial expressiveness.

In addition to inhibitory functioning, facial expressiveness was assessed using a standardized and experimentally controlled approach. As has successfully been done in our previous fMRI study (Kunz et al., 2011), experimental heat pain was used as method of affect-induction. This had the advantage of easily eliciting comparable levels of subjective experience across participants. In addition, two established methods to assess and quantify facial expressiveness were applied. On the one hand, we used the Facial Action Coding System (FACS; Ekman & Friesen, 1978), as has been done in most studies assessing the facial response to pain (for a review see Craig et al., 2011; Hadjistavropoulos et al., 2011). Moreover, electromyography (EMG) was conducted over the orbicularis oculi muscle (which is functionally the most prominent muscle in the response to pain; Prkachin, 1992) for a better resolution of the measurement than FACS can guarantee. Using these two complementary measures should enable a thorough assessment of the association between facial expressiveness and inhibitory functioning.

It has to be noted that participants were deliberately not instructed to inhibit facial expressiveness. The measurement, thus, differed from the measures of inhibitory functioning given that there was no clearly stated necessity to recruit inhibitory processes. However, as suggested by previous results (Karmann, Lautenbacher, Bauer, & Kunz, 2014; Kleck et al., 1976; Saarni, 1984; Underwood, Coie, & Herbsman, 1992; Vervoort et al., 2008, 2011), most social situations require the continuous inhibition of too frank expressions of (negative) internal states because otherwise social rules might be violated. Only in the presence of very intimate persons, this inhibitory control might be weakened (Karmann et al., 2014; Vervoort et al., 2008). Therefore, inhibition of facial expression was not subject to further instructions because inhibitory control was supposed to be sufficiently activated.

In summary, our aim was to investigate whether the degree of facial expressiveness (in response to painful stimulation) is related to inhibitory functioning (Antisaccade, Stroop task, UPPS). We hypothesized a substantial (negative) association between the degree of facial expressiveness and the performance in the Antisaccade task (hypothetically more closely related task). In contrast, the (negative) association with the performance in the Stroop task as well as with the UPPS was hypothesized to be more marginal; given that these two variables are hypothetically more distantly related to facial expressiveness.

2. Materials and methods

2.1. Participants

The 49 participants (24 females; M = 22.2 years; SD = 3.1) of the current study were recruited at the University of Bamberg by bulletins put up throughout campus. Individuals with chronic pain, psychological or physical illnesses or such taking psychotropic drugs or analgesics were excluded from participation. Furthermore, we only included participants who could do all tasks without depending on the use of some type of corrective lenses (e.g. glasses or contacts) to prevent insufficient infrared reflection during the eyetracking paradigm (Antisaccade task). Participants provided written informed consent before testing and either received course credit or monetary compensation for their participation. The study protocol was approved by the Ethics committee of the University of Bamberg.

2.2. Procedure

Upon arriving at the laboratory, participants were told that the experiment was designed to investigate the relationship between pain and inhibition. Participants were further told that this would be realized by assessing self-reported pain intensity, eye-movements via EMG- and video-recording and the performance in inhibitory tasks. Thus, by reducing the emphasis on our interest in facial expressions, the participants’ focus on their facial expressions was supposed to be attenuated.

The experiment consisted of two blocks. During the first block the degree of facial expressiveness was assessed (facial expressiveness block). This was done by applying a set of painful and non-painful thermal stimuli and recording the degree of facial expressiveness in response to these stimuli via video and electromyography (EMG). In the second block of the experiment the ability to inhibit was determined (inhibitory functioning block). This was realized by conducting the Antisaccade task (testing motor inhibition), the Stroop task (testing behavioral and cognitive inhibition) and the German version of the UPPS Impulsive behavior scale (testing impulse control via self-report).

2.3. Facial expressiveness block

2.3.1. Pain induction

Following a previous protocol that has been shown to successfully elicit facial responses to pain (Kunz et al., 2011; Kunz, Scharmann, Hemmeter, Schepelmann, & Lautenbacher, 2007), thermal stimulation was applied on the tibia of the left leg (centrally in between knee and ankle) by a Peltier based contact stimulation device (Medoc, TSA-2001, Ramat Yishai, Israel) with a 30 mm × 30 mm contact thermode. To ensure that all participants perceived the pre-set stimulus as similarly painful during the assessment of facial expressiveness, temperature intensities were tailored
to the individual pain threshold. As a result of this, it was guaranteed that individual differences in the degree of facial expressiveness were not simply due to percentage pain intensities but that pain thresholds were therefore determined first, using the method of adjustment. Participants were asked to adjust a temperature starting from 38 °C, using heating and cooling buttons, until they obtained a level which was barely painful. A constant press of the buttons produced a heating or cooling rate of 0.5 °C/s. Following this, there were 4 trials and the average of these trials was used to constitute the threshold estimate.

Following the assessment of pain thresholds, the intensities for the two types of stimuli were determined: non-painful (−1 °C below the pain threshold) and painful stimulation (+3 °C above the pain threshold). Participants received ten painful and ten non-painful stimuli in random order. Applying also non-painful intensities allows a determination of the degree to which facial responses during thermal stimulation are indeed specific for painful experiences. Due to individual differences in pain threshold, target temperatures varied between 41.3 °C and 47.7 °C for non-painful stimuli and between 45.3 °C and 51.0 °C for painful stimuli. Each phase of heat stimulus (painful/non-painful) had the same characteristics (5 s (plateau); rate of change: 4 °C/s; baseline temperature: 38 °C; inter-stimulus-intervals of 15–20 s). The trapeze-shaped stimulus, thus, consisted of a phase with temperature rise (variable length between 83.3 s and 3.25 s depending on the type of stimulus (painful/non-painful) and individual target temperature, 5 s of constant target temperature and a decrease of temperature toward baseline (same duration as the phase of rise). Following each stimulus, perceived pain intensity was assessed using self-report ratings. This was realized by using a visual analog scale (VAS; 100 mm) on an electronic shift register, which was placed on the table in front of the participant. Participants were informed that the left and right sides of the scale corresponded to “no sensation” and to “extremely strong pain”, respectively. In addition, the scale was labeled with a verbal anchor of “faintly painful” in the center so that all non-painful sensations should be rated to the left and all painful ones to the right of the center. Participants were instructed to rate the intensity of their pain by matching their perceived pain intensity with a certain distance on the scale. This was achieved by moving the cursor (which was set to the middle of the scale before each trial) to the right or left.

2.3.2. Degree of facial expressiveness (criterion variable)

2.3.2.1. Facial Action Coding System (FACS). The face of the participants was videotaped throughout the pain induction procedures. The camera was located approximately 3.0 m from the participant. In order to mark the plateau phase of the stimuli, a LED visible to the camera, but not to the participant, was lit concomitantly with the 5 s (plateau), starting value of the target temperature, and the LEDs reached. During stimulation, participants were instructed not to talk and to look at an emotionally neutral painting on the wall behind the camera to ensure that the face would always be recorded in an upright and frontal view.

Facial expressions were coded from the video recordings using the Facial Action Coding System (FACS; Ekman & Friesen, 1978), which is based on anatomical analysis of facial movements and distinguishes 44 different Action Units (AUs) produced by single muscles or combinations of muscles. One of the authors, a certified FACS coder (qualified by passing an examination given by the developers of the system) identified the frequency, thermosensitivity, starting value (of the different Action Units) and duration (of the different Action Units). To guarantee reliability of the coding, another certified FACS coder re-coded 10% of the data; with an interrater reliability of .86 as calculated using the Ekman–Friesen formula (Ekman & Friesen, 1978). A software designed for the analysis of observational data, The Observer X-PRO; Noldus Information Technology was used to segment the videos and to enter the FACS codes into a time-related database. Time segments of 7 s beginning just after stimulus had reached the target temperature were selected for scoring. In total, 20 segments of thermal stimulation (10 non-painful and 10 painful segments) were analyzed in each subject. For the purpose of necessary data reduction, we combined similar facial responses as has been done in preceding studies without any loss of information (Hale & Hadjistavropoulos, 1997; Karmann et al., 2014; Kunz, Mylius, Schepelman, & Lautenbacher, 2004; Kunz et al., 2007; Prkachin, 1992). Those combinations include AU 1/2, AU 6/7, AU 9/10 and AU 25/26/27.

Pain-relevant AUs were selected based on the procedure developed in previous studies (e.g. Kunz, Mylius, Schepelman, & Lautenbacher, 2008; Kunz et al., 2007) using the following steps: (1) AUs had to occur in more than 5% of the painful segments recorded and (2) AUs had to be more frequent during painful than during non-painful trials (effect size d > 0.5; these AUs are marked in bold in Table 1). This chosen subset of pain-relevant AUs is consistent with previous findings regarding facial responses to pain (Craig et al., 2011; Karmann et al., 2014; Kunz et al., 2008) and consists of the following AUs: AU 4 (towering of the brows), AU 6/7 (orbit tightening) and AU 9/10 (levator contraction). Following, mean AU-frequency and mean AU-intensity values of the selected AUs were combined (product terms) and averaged across all selected AUs to form a composite score of pain-relevant facial responses. Due to the fact that the composite scores were not distributed normally (Kolmogorov–Smirnov Z = 1.545, p < 0.5), square root transformed composite scores were used for further statistical analyses as has also been done in previous studies (Karmann et al., 2014; Kunz et al., 2011; Kunz, Faltermeier, & Lautenbacher, 2012).

2.3.2.2. Electromyographic (EMG) recording and analysis. Facial surface EMG was recorded over the region of orbicularis oculi (one of the most activated muscles in the response to pain; Prkachin, 1992) below the left eye using two Ag/AgCl electrodes filled with electrode paste. The electrodes were placed according to the guidelines for standard electrode placement by Frieland and Cacioppo (1986). Prior to application of the electrodes, skin was cleaned with an alcoholic skin detergent to reduce electrode resistance. The acquisition of EMG raw signal was carried out by the device SIGMA Plpro/Type Databox DB 36 including a 16 bit AD-converter with a dynamic range from 0.5 µV to 2 mV. The recording bandwidth of the EMG signal was between 0.2 Hz and 300 Hz; input resistance was above 20 MΩ. The signal was sampled at 512 Hz.

Analysis of the raw EMG signal was done offline by the program “Vision Analyzer” (Brain Products, Munich). In the first step, the signal was cut into 7-s segments which contained the EMG response starting when the stimulus had reached the target temperature (painful/non-painful). The signal of each segment was subsequently filtered (50Hz notch filter, 20Hz high-pass filter, 250Hz low-pass filter), rectified and integrated (100-ms time constant). Following, a sum-score was calculated for the signal of the 7-s period creating a score that covered the same time period as the FACS score. For baseline correction, the trials with non-painful stimulation (reference situation; Hess, 2012) were considered. The EMG data of each subject was thus z-transformed across painful and non-painful trials.

When comparing the two measures of facial expressiveness, one notices that the EMG measure reflected the activity of a single muscle (orbicularis oculi) whereas the FACS measure was less regionally specific and can potentially include all facial movements. Despite this difference, these two measures were chosen for the following reason. The EMG signal – as a measure with high intensity resolution – might even be adequate when only using a single recording site with particular relevance for the facial expression of pain. A single Action Unit (AU) of the FACS on the other hand might not be as sensitive given that one single muscle movement does not become visible in every trial. Furthermore, the chosen composite score has been shown to be a more valid measure of the facial response to pain than any single AU (Prkachin & Solomon, 2008). The better sensitivity of the FACS composite score compared to the single AU covering orbicularis oculi activity (AU 6/7) can also be seen when correlating the two measures with the EMG score (see Section 3.1).

2.4. Inhibitory functioning block (assessment of predictor variables)

2.4.1. The Antisaccade task. To assess the ability to inhibit an automatic motor response, we conducted the Antisaccade task by replicating the experimental set-up of Derakshan, et al. (Derakshan, Ansari, Hansard, Shoker, & Eysenck, 2009). Stimulus presentation and tracking of eye movements were conducted by a system distributed by the company Interactive Minds, Dresden. This system consisted of a 19 inch Samsung LCD-screen (resolution 1280 x 1024 pixels; 60 cm viewing distance) and the eyetracking system Eyezage Edge™ by LC Technologies Inc and was driven by the software NYAN 2XT (version 2.3.3). In order to measure the pupil’s orientation, this eyetracking system uses the corneal reflection of an infrared light source (corneal reflex method; Mason, 1989). It features a sampling rate of 60 Hz and a fidelity of 0.4. Each trial began with the presentation of a fixation cross (12 mm x 12 mm, 1.15 x 1.15) in the center of the screen which was to be fixated until it disappeared (2000 ms). In addition, two dark gray rectangular frames were presented left and right of the fixation cross (60 mm x 60 mm, 5.73 x 5.73; located at 8.29° horizontally from the fixation cross) to indicate the destination of the later saccade. These two frames were then constantly present in all following images. Next, a circle (diameter = 35 mm, 3.34”) which represented the “cue” appeared for 600 ms. The cue was either presented within in the frame in the left or right side of the screen (1° from horizontally from the fixation cross) with both locations being equally probable. Depending on the instruction given at the beginning of each block, participants were asked to look either toward the cue (Prosacade block) or away from it (Anti-saccade block), as quickly as possible. Afterwards, an arrow pointing up or down (1° horizontally from the fixation cross) was presented for 100 ms. The target either appeared in the same location as the cue (Prosacade block) or on the opposite side of the screen (Antisaccade block). The participants had to identify the direction of the arrow via pressing the up or down key of a regular computer.

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Table 1

<table>
<thead>
<tr>
<th>Action Unit</th>
<th>Percent Change</th>
<th>Effect Size</th>
</tr>
</thead>
<tbody>
<tr>
<td>AU 1/2</td>
<td>7.8</td>
<td>0.25</td>
</tr>
<tr>
<td>AU 6/7</td>
<td>44.3</td>
<td>0.88</td>
</tr>
<tr>
<td>AU 9/10</td>
<td>9.8</td>
<td>1.07</td>
</tr>
<tr>
<td>AU 12</td>
<td>5.5</td>
<td>0.38</td>
</tr>
<tr>
<td>AU 14</td>
<td>9.8</td>
<td>0.21</td>
</tr>
<tr>
<td>AU 25</td>
<td>7.1</td>
<td>0.14</td>
</tr>
<tr>
<td>AU 26/27</td>
<td>14.7</td>
<td>0.49</td>
</tr>
</tbody>
</table>

Effect sizes for frequency differences between "non-painful" and "painful" segments are given. Medium and strong effect sizes (≥ 0.5) are marked in bold.
keyboard, as fast and accurate as possible. This approach served the purpose of maintaining the participant’s vigilance and given that our main focus lay on the inhibition of automatic ocularmotor reactions, key-press reaction time data was not further analyzed. The task consisted of six blocks (three Prossaccade and three Antisaccade blocks; alternating), with 12 trials each, which resulted in a total of 72 trials. One half of the participants started the test with a Prossaccade block, the other half with an Antisaccade block. Ahead of testing, participants practiced the task in a short training block consisting of 8 trials.

The measures chosen for further analyses were – based on previous work (Deraedt et al., 2009) – the following: Latency of the first correct saccade (latency of the first saccade (time interval between cue onset and start of the first fixation) in the right direction) and percentage of incorrect saccades (percentage of first saccades in the wrong direction). Inhibitory functioning was then determined by calculating the differences between Anti- and Pro-saccade trials for the two variables (in the following called Δ). High scores, resulting from big differences between Anti- and Pro-saccade trials, thus indicated a poor ability to inhibit (inhibitory functioning), whereas low scores indicated a high degree of inhibitory functioning.

2.4.2. The Stroop task

In order to additionally assess a variable which is hypothetically more distinctly related to facial expressiveness, the Stroop task (Stroop, 1935) was conducted. For this purpose, a computerized version of the Stroop task was created with E-Prime Version 2.0. There were four blocks consisting of the classical stroop conditions. In two of the blocks the presented color word (“red,” “green,” “blue” or “yellow”) had to be named; once in a neutral condition where the color word was printed in black, and once in a congruent condition (CNincg – CNneu) that was presented in either red, green, blue or yellow) color naming – incongruent; WNincg ). In the other two blocks the color of the stimulus had to be named: once again in a neutral condition where the stimuli consisted of a row of X’s (XXXXX) that were presented in either red, green, blue or yellow (color naming – neutral; CNneu) and once when the color words were again incongruently colored in one of the other three colors (word naming – incongruent; WNincg ). The order of the blocks was randomized across participants. All stimuli were presented centrally on a 19 inch EIZO LCD-screen (resolution 1280 x 1024 pixels; 60 cm viewing distance) in Arial, font size 30. Instructions explaining the task appeared on the screen ahead of each block. Each stimulus was presented until the subject responded. All responses were given via one of four color-coded keys on a regular computer keyboard (“L”, “R”, “P”, “G”; located within a range of 12 cm) using the index finger of the dominant hand. As indicators of inhibitory functioning, scores for the stroop-effect (reaction time of CNincg–CNneu) and the reverse stroop-effect (reaction times of WNneu–WNincg ) were calculated neglecting erroneous trials. Again, high scores indicated a low degree of inhibitory functioning whereas low scores indicated a high degree of inhibitory functioning.

2.4.3. Self-evaluation of impulse control

In order to assess the self-evaluation of impulse control, we used the UPPS Impulsive Behavior Scale (Whiteside & Lyamam, 2001) which was developed to measure four dimensions of impulsive behavior, namely Urgency, Lack of Premeditation, Lack of Perseverance and Sensation Seeking. The inventory contains 45 items that are rated on a four-point scale with the end points “agree strongly” and “disagree strongly”. The UPPS has been widely used in research and the German version has been shown to be a robust and valid measure for impulsive behavior (Kimpfe & Mitte, 2009; Schmidt, Gay, d’Acremont, & van der Linden, 2008).

2.5. Statistical analysis

First, descriptive statistics (Mean, standard deviation) were calculated for the Antisaccade and Stroop task, as well as the UPPS. In order to demonstrate that inhibition prevailed, t-tests for independent samples were further calculated to determine whether variables differed between the Anti- and Pro-saccade blocks and for the stroop and reverse stroop effect. In order to screen for multicollinearity (in the following regression analyses), Pearson’s correlations were additionally conducted for relationships amongst and between the criteria (facial expressiveness) and predictor variables (inhibitory functioning).

To test our main hypotheses, sequential multiple regression analyses were conducted in order to find out whether inhibitory functioning can predict the degree of facial expressiveness. For this purpose the measures of inhibitory functioning were entered into an analysis blockwise. The order of entrance was thereby determined by the relevance of the measure; starting with the least and ending with the most relevant. This way the more relevant measures of inhibitory functioning were evaluated for their additional predictive value after controlling for variations explained by less relevant variables (conservative approach). Accordingly, the model consisted of three regression blocks: The UPPS variables (Urgency, Lack of Premeditation, Lack of Perseverance and Sensation Seeking) were entered in the first block, the Stroop task variables (stroop effect, reverse stroop effect) in the second and the Antisaccade task variables (Δ latency of the first correct saccade, Δ percentage of incorrect saccades) in the third block. In order to assess the association of inhibitory functioning and facial expressiveness measured by FACS as well as EMG, two separate regression analyses were conducted. In one of them, the FACS-composite score served as criterion, whereas in the other one the EMG-signal of orbicularis oculi was used.

Given that directed hypotheses existed, one-way tests were conducted for the main (regression) analyses. For all analyses, findings were considered to be statistically significant at α < 0.05.

3. Results

In the present study the mean pain threshold amounted to 46.0°C (SD = 1.55). Participants on average rated the painful stimulation (+3°C above threshold) clearly above 50 (M = 80.8; SD = 9.6) whereas the non-painful stimulation (–1°C below threshold) was on average rated as clearly below 50 (M = 18.1; SD = 13.2) on the VAS. The ratings of the two stimulus intensities differed significantly from each other (t(48) = 32.0; p < 0.001). Descriptive data of the predictor variables is given in Table 2. As expected, the latency of the first correct saccade and the percentage of incorrect saccades in the Antisaccade task were significantly higher in the Antisaccade compared to the Prossaccade trials – t(48) = 17.98; t(48) = 8.72; all p < 0.001. With regard to the Stroop task, the present data showed a stroop effect, as well as the reverse stroop effect; with the reaction times of color naming – incongruent (CNincg) trials being significantly longer than those of color naming – neutral (CNneu) trials (t(48) = 5.71; p < 0.001) and the reaction times of word naming – incongruent (WNincg) trials being significantly longer than those of word naming – neutral (WNneu) trials (t(48) = 9.79; p < 0.001). The findings of the present study were thus, within normal limits and the proper use of the tests, indicating inhibitory functioning, can be assumed.

3.1. Intercorrelations (within criteria and predictor variables)

Facial measurements (criteria): Although the EMG recording was only assessed over the orbicularis oculi muscle, we did not want to restrict the FACS criterion to the activity of orbicularis oculi, but rather use a score which represents the activity of all pain-relevant muscles in the face and, thus, used the composite score (including AU 4, AU 6/7, AU 9/10) as the FACS criterion variable. As can be seen in Table 3, FACS composite score of the whole face and EMG recording of the orbicularis oculi muscle were strongly correlated (r = 0.702; p < 0.001).1

Measures of Inhibitory functioning (predictors): As can be seen in detail in Table 3, the UPPS variables were correlated so that Lack of Premeditation was significantly related to Urgency, as well as Sensation Seeking. The UPPS variable Lack of Perseverance was significantly related to both Antisaccade measures; the Δ latency of the first correct saccade as well as the Δ percentage of incorrect saccades. Besides that, none of the UPPS variables were significantly related to each other or any of the Antisaccade or Stroop variables (all p > .05). Surprisingly, the Stroop measures were neither significantly related to each other nor to the Antisaccade measures (all p > .05). The Antisaccade variables Δ latency of the first correct saccade and Δ percentage of incorrect saccades were significantly related to each other. Thus, none of the predictors correlated highly with predictors from the other predictor groups.

3.2. Prediction of the degree of facial expressiveness by inhibitory functioning

3.2.1. FACS composite score as criterion

As illustrated in Table 4, the sequential multiple regression revealed that the UPPS (β-weights ranging between –.01 and .15)
Table 2

<table>
<thead>
<tr>
<th>Category</th>
<th>Measure</th>
<th>Mean</th>
<th>SD</th>
<th>t</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anti-saccade task</td>
<td>Latency of first correct saccade – pro</td>
<td>250 ms</td>
<td>32.6</td>
<td>17.98</td>
<td>&lt;.001</td>
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<tr>
<td></td>
<td>Latency of first correct saccade – anti</td>
<td>379 ms</td>
<td>45.0</td>
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<td></td>
<td>Antisaccade – Δ Latency correct (Δ latency of first correct saccade)</td>
<td>129 ms</td>
<td>50.1</td>
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<tr>
<td></td>
<td>Percentage of incorrect saccades – pro</td>
<td>8.16%</td>
<td>7.0</td>
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</tr>
<tr>
<td></td>
<td>Percentage of incorrect saccades – anti</td>
<td>25.25%</td>
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<tr>
<td></td>
<td>Antisaccade – Δ Percentage incorrect (Δ percentage of incorrect saccades)</td>
<td>17.10%</td>
<td>13.7</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stroop</td>
<td>WNincg</td>
<td>915 ms</td>
<td>112.8</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>CNneu</td>
<td>1083 ms</td>
<td>242.9</td>
<td>8.71</td>
<td>&lt;.001</td>
</tr>
<tr>
<td></td>
<td>Stroop effect</td>
<td>169 ms</td>
<td>206.8</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>WNneu</td>
<td>951 ms</td>
<td>151.2</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>WNeug</td>
<td>1227 ms</td>
<td>248.8</td>
<td>9.79</td>
<td>&lt;.001</td>
</tr>
<tr>
<td></td>
<td>Reverse stroop effect</td>
<td>276 ms</td>
<td>197.5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>UPPS</td>
<td>Urgency</td>
<td>27.06</td>
<td>5.18</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Lack of Premeditation</td>
<td>24.08</td>
<td>4.28</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Lack of Perseverance</td>
<td>19.65</td>
<td>3.99</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Sensation Seeking</td>
<td>34.78</td>
<td>6.73</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

CNneu = color naming (non-word stimulus); CNneu = color naming of incongruously color-words; WNneu = word naming of black color-words; WNincg = word naming of incongruously colored color-words; UPPS = Impulsive Behavior Scale.

could not significantly contribute to explaining the variations in facial expressiveness measured by FACS. Entering the Stroop variables (β-weights ranging between −.21 and .12) into the model did not also add significantly to explaining the variations in facial expressiveness. Not until the Antisaccade variables were entered into the model (see Table 4), the predictive value of the model changed significantly (ΔR² = .183; p < .01). It showed that the Antisaccade variables (Δ latency of the first correct saccade, Δ percentage of incorrect saccades) were positively related to the FACS composite score (β-weights ranging between .08 and .44). Thus, the lower the level of inhibition was during the Antisaccade task (indicated by higher Δ-values), the stronger the participants expressed their pain via the face.

3.2.2. EMG-activity as criterion

When using the orbicularis oculi EMG-activity as measure of facial expressiveness, similar results evolved (see Table 4). Again, neither the UPPS (β-weights ranging between −.06 and .19) nor the Stroop task variables (β-weights ranging between −.19 and .02) could significantly contribute to explaining the variations in facial expressiveness. However, entering the Antisaccade variables (see Table 4) changed the predictive value of the model significantly (ΔR² = .119; p < .05). It showed that the Antisaccade variables (Δ latency of the first correct saccade, Δ percentage of incorrect saccades) were again positively related to the EMG activity of orbicularis oculi (β-weights ranging between .11 and .33). Thus, the lower inhibitory functioning was in the Antisaccade task (indicated by higher Δ-values), the more expressive were the individuals.

As can be seen in Sections 3.2.1 and 3.2.2, the same pattern of significant and non-significant associations was found when FACS composite scores or EMG activity of orbicularis oculi were used as measures of facial expressiveness (criteria). Whether the two criterion variables can statistically substitute each other, however, cannot be drawn from these results. In order to test the mutual substitutability, we conducted partial correlations eliminating either the influence of EMG or FACS when correlating the performance in the Antisaccade task with FACS or EMG, respectively. In order to avoid multiple testing (due to multiple Antisaccade parameters), the performance in the Antisaccade task was in this context represented by the sum of the z-standardized Antisaccade parameters (which was possible since both parameters acted in the same direction and were highly correlated). The results show that the correlation of the performance in the Antisaccade task and the FACS score (r = .380, p < .01) does not remain significant when controlling for the EMG score (r = .195, p = .184). Likewise, the significant correlation between the performance in the Antisaccade task and the EMG score (r = .356, p < .05) disappears when controlling for the FACS score (r = .136, p = .357). These findings indicate that FACS and EMG can indeed statistically substitute each other in the present study.

4. Discussion

The main purpose of the present study was to investigate whether the degree to which individuals facially express their inner affective state, namely pain, can be predicted by different aspects of inhibitory functioning. As expected, we found a strong negative relationship between motor inhibitory functioning assessed by the Antisaccade task and facial activity during painful stimulation (measured via FACS and EMG). The stronger the inhibitory control of motor responses was, the weaker an individual expressed his/her pain via the face. In contrast, the degree of facial expressiveness did neither depend on the inhibitory performance in the Stroop task nor on impulse control assessed by the UPPS. These findings will be discussed in detail below.

Although research on facial expressions of pain has had a long history, to date still little is known about the mechanisms regulating the degree of facial expressiveness. Our results now suggest that the degree of facial expressiveness during pain is related to the ability to inhibit automatic motor responses. More precisely, we found that the degree of facial expressiveness could be predicted by the performance in the Antisaccade task. Individuals who perform well in this task are highly able to inhibit the automatic orientation saccade toward an abruptly appearing peripheral stimulus. Interestingly, the stronger this ability was pronounced, the less facially expressive individuals were when experiencing moderately painful heat. These results seem to be well in line with the results of recent brain imaging findings investigating cerebral activation patterns during the facial expression of pain (Kunz et al., 2011) and disgust (Goldin, McRae, Ramel, & Gross, 2008). Both studies showed concordantly that areas known to be involved in motor inhibition – such as the medial prefrontal cortex and the basal ganglia – were more active when facial expressiveness was low. Thus, combining these findings with our current results strongly suggests that
Table 4

Linear regression models for FACS composite score (criterion) and orbicularis oculi EMG activity (criterion). Predictors in step 1: Urgency, Lack of Premediation, Lack Perseverance and Sensation Seeking (UPPS); added predictors in step 2: stroop effect, reverse stroop effect (Stroop task); added predictors in step 3: Δ Latency of the first correct saccade, Δ percentage of incorrect saccades (Antisaccade task); significant results are marked in bold.

<table>
<thead>
<tr>
<th>Step</th>
<th>Variables added</th>
<th>Total R²</th>
<th>ΔR²</th>
<th>p (for Δ in R²)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>+ UPPS</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>+ Stroop</td>
<td>0.51</td>
<td>0.35</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>+ Antisaccade</td>
<td>0.28</td>
<td>0.15</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Orbicularis oculi EMG activity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>+ UPPS</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>+ Stroop</td>
<td>0.06</td>
<td>0.29</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>+ Antisaccade</td>
<td>0.21</td>
<td>0.30</td>
<td></td>
</tr>
</tbody>
</table>

Inhibitory mechanisms seem to play a crucial role in the (down-)regulation of facial expressiveness – at least when considering the expression of negative affective states such as pain and disgust.

The robustness of our findings is supported by the fact that the relationship between facial expressiveness and the performance in the Antisaccade task was found when using FACS scores as well as EMG activity as measures of facial expressiveness. This result is even more intriguing when considering the differences of the two measures. On the one hand, FACS quantifies visible muscle movements in the face, whereas the EMG signal reflects slight changes in muscular activity, which are not necessarily visible. Secondly, with FACS the activity of all muscles in the face was coded while the EMG signal was only derived from orbicularis oculi; single site recording was necessary to not interfere with facial expressiveness. Although orbicularis oculi – as one of the most active muscles in the response to pain (Prkachin, 1992) – represents the best possible single parameter of the facial response to pain, recording its activity can never cover all aspects of the facial pain response. Nevertheless, even when only represented by the EMG activity of one muscle, facial expressiveness of pain could be predicted by the Antisaccade task. The association between the ability to inhibit automatic motor responses and facial expressiveness, thus, seems to be robust independent of the measure of facial expressiveness.

In contrast to the Antisaccade task, the performance in the Stroop task could not contribute significantly to explaining variance in the degree of facial expressiveness. Methodological shortcomings cannot account for this difference given that we found the usual stroop as well as the reverse stroop effect with sufficient strength (cf. Miyake et al., 2000). It thus can be assumed that the Stroop task was performed according to the state-of-the-art. Therefore, it is more likely that the Stroop and the Antisaccade task target different types of inhibitory functioning; which are differently related to the processes involved in the regulation of facial expressiveness. The Antisaccade task requires the inhibition of a reflex saccade (Hutton & Ettinger, 2006) while the Stroop task engages the inhibition of a discriminative operant (non-reflex) response based on semantic information processing (Nigg, 2000). Thus, two clearly distinguishable types of inhibition seem to be targeted which is also suggested by the lack of correlation between the Antisaccade and Stroop variables. Facial expressions are motor responses which can be both reflex and voluntary in nature (Craig et al., 2011). It can be assumed that facial responses to noxious stimuli in a socially deprived environment – as our experimental laboratory – are better explained by a reflex mechanism. Accordingly, it seems likely that the performance in the Antisaccade task (inhibition of a reflex saccade) and the degree of facial expressiveness were both regulated by similar reflex mechanisms and filtered through the same “inhibitory gate”. The performance in the Stroop task on the other hand might be regulated by another “inhibitory gate” given that higher cognitive processes are demanded in this
case. Thus, especially inhibitory motor control seems to be crucial for the (down-)regulation of facial expressiveness.

This assumption is corroborated by brain imaging findings. Brain activation during the Antisaccade task shows close resemblance to the activation involved in the regulation of facial expressiveness, overlapping e.g. in the prefrontal cortex and the basalganglia (Ford, Goltz, Brown, & Everling, 2005; Goldin et al., 2008; Kunz et al., 2011; Munoz & Everling, 2004). In contrast, inhibitory performance in the Stroop task was mainly associated with the activation of anterior cingulate cortex (ACC), insula, premotor and inferior frontal areas (Leung, Skudlarski, Gatenby, Peterson, & Gore, 2000), which were not found to be active during the inhibition of facial expressiveness (Goldin et al., 2008; Kunz et al., 2011).

The context, in which this close relationship between inhibitory motor control and facial expressiveness applies, however, might be limited. As mentioned before, facial expressions can also be characterized as voluntary. This is the case in socially enriched situations, where the voluntary programming of facial activity can become more relevant (e.g. social/affiliative smiling; Niedenthal, Mermillod, Maringer, & Hess, 2010). Here, the consequences of expressing inner affective states have to be considered and the reflex response can be overridden e.g. when individuals try to fake or aggravate facial expressions (Craig et al., 2011; Hadjistavropoulos et al., 2011). In such contexts, it seems less likely that the inhibitory regulation of reflex activity has the same relevance in determining the degree of facial expressiveness. In order to be able to quantify the impact of reflex and voluntary inhibitory regulation mechanisms, further studies including socially enriched contexts are, thus, necessary.

Our analyses did not indicate a significant association between self-evaluated impulse control (UPPS) and the degree of facial expressiveness. This was both the case for the FACS- as well as the EMG-scores. How can this be explained? On the one hand, the different measures might have accessed different levels of processing. The completion of self-report measures such as the UPPS rather demands the subject’s explicit and conscious processing (Kline, 2000), whereas the degree of facial expressiveness measured by objective, implicit measures (FACS; EMG) is not instructed to be monitored while it is assessed. On the other hand, there might just simply be no association. Even though both, impulse control (Gay et al., 2008; Roberts et al., 2011) and facial expressiveness (current study) have been shown to be associated with inhibitory functioning, the two do not necessarily have to be related. It, thus, seems possible that impulsive behavior and facial expressiveness are regulated by different inhibitory mechanisms.

4.1. Limitations

A limitation of the present study might be that we only tested facial expressiveness during painful stimulation. Our results now indicate that certain aspects of inhibitory functioning can predict the degree of facial expressiveness in response to pain whereas others cannot. Whether this pattern of significant and non-significant associations can also be found for other affective states has to be demonstrated in further studies.

4.2. Summary

Our findings clearly indicate that the degree of facial expressive- ness in response to pain can be predicted by the ability to inhibit automatic motor responses (Antisaccade task). Accordingly, the better an individual is able to inhibit automatic motor responses, the less facially expressive he/she seems to be. In contrast, there does not seem to be a similar relationship between inhibitory functioning demanding cognitive processing (Stroop task) or self-evaluated impulse control (UPPS) and facial expressiveness. These results suggest that the degree of facial expressiveness in response to pain is specifically regulated by motor inhibitory mechanisms, but is not regulated by inhibitory mechanisms in general.

This association applies to situations, in which the facial activity is triggered by preceding pain stimuli and has little instrument- al relevance for social interactions, in other words where facial activity is rather of reflex nature. Such situations are prevalent, comprising most of the everyday acute pains. However, it is still an open question whether the inhibition of automatic motor responses plays a similarly critical role when facial activity during negative affective states is more instrumental, aiming at social impact and reward.

Disclosures

The study was supported by a research grant from the Deutsche Forschungsgemeinschaft (DFG, Ku2294/6). There are no financial or other relationships that might lead to a conflict of interest.

References


