Typical pain experience but underestimation of others’ pain: emotion perception in self and others in autism spectrum disorder

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Abstract
Difficulties in emotion perception are commonly observed in autism spectrum disorder (ASD). However, it is unclear whether these difficulties can be attributed to a general problem of relating to emotional states, or whether they specifically concern the perception of others’ expressions. This study addressed this question in the context of pain, a sensory and emotional state with strong social relevance. We investigated pain evaluation in self and others in sixteen male individuals with ASD and sixteen age- and gender-matched individuals without ASD. Both groups had at least average intelligence and comparable levels of alexithymia and pain catastrophizing. We assessed pain reactivity by administering suprathreshold electrical pain stimulation at four intensity levels. Pain evaluation in others was investigated using dynamic facial expressions of shoulder patients experiencing pain at the same four intensity levels. Participants with ASD evaluated their own pain as being more intense than the pain of others, showing an underestimation bias for others’ pain at all intensity levels. Conversely, in the control group, self- and other-evaluations of pain intensity were comparable and positively associated. Results indicate that emotion perception difficulties in ASD concern the evaluation of others’ emotional expressions, with no evidence for atypical experience of own emotional states.

Keywords
Emotion, face perception, pain, social cognition and social behaviour, alexithymia, sensory features, autism spectrum disorder
Individuals with autism spectrum disorder (ASD) have long emphasized the significance of sensory and perceptual alterations for characterizing the condition (e.g. Grandin, 1995). Recently, these have also been added to formal criteria for the diagnosis (Diagnostic and Statistical Manual of Mental Disorders, DSM-5; American Psychiatric Association, 2013). This addition is supported by more recent findings, which have revealed complex patterns of hypo- and hypersensitivity to sensory stimuli in ASD (e.g. Ben-Sasson et al., 2009; Rogers and Ozonoff, 2005). This research also includes important efforts to understand the relationships between the sensory and social symptoms of ASD – or the ways in which difficulties in processing sensory information are related to difficulties in communication and social cognition (Frith, 1989). A topic of particular importance in this context is the experience and expression of pain sensations. Pain is a sensory and emotional experience which has strong social relevance. The ability to evaluate and express one’s own pain experiences, and the ability to evaluate the pain expressions of others, can have meaningful consequences for one’s physical and social wellbeing.

According to one prominent explanation, difficulties in emotion perception seen in ASD – such as the ability to make inferences about others’ emotions based on their facial expressions – are driven by difficulties in categorizing and relating to emotional experiences more generally. The key idea here is that people with ASD have trouble evaluating others’ emotional expressions to the same extent as they struggle with
evaluating their own emotional states. Several studies have observed links between social symptoms in ASD and atypical perception of own sensory states (e.g. Duerden et al., 2015; Hilton et al., 2010). Moreover, it has been demonstrated that some of the social impairments seen in ASD can be attributed to alexithymic traits (Bird and Cook, 2013). Alexithymia is characterized by difficulties in detecting and describing emotional experiences in the self, but also in recognizing others’ emotions (Bagby et al., 1994a). Its prevalence in the general population has been reported to be around 13% (Salminen et al., 1999). Studies in people with ASD report rates in the range of 48 to 63% (Hill and Berthoz, 2006; Hill et al., 2004; Milosavljevic et al., 2015; Samson et al., 2012). While these figures stem from relatively small samples (n= 27 to 56), they consistently indicate that alexithymia is highly prevalent in ASD. This suggests that a majority of people with ASD have difficulties evaluating their own emotional experiences.

An alternative explanation is that people with ASD are able to recognize and make inferences about emotional states, but have trouble decoding such information from (for example) visual cues conveyed in others’ facial and bodily expressions. In support of this explanation, emerging evidence shows that people with ASD perceive faces differently, even when they meet attentional (Shah et al., 2016) or social-cognitive demands of face processing tasks (Walsh et al., 2016). The main aim of this study is to compare these explanations by investigating whether emotion perception
difficulties in ASD are specific to perceiving others’ expressions, or concern a general problem of relating to emotions, including own emotions. It thus focuses on the evaluation of emotional states in self and others, and the relationship between self- and other-perspectives.

Existing research paints a complex picture of pain reactivity in ASD (see reviews by Allely, 2013 and Moore, 2015). A weight of anecdotal self-reports and caregiver reports suggest that people with ASD experience pain as less intense and respond less strongly in painful situations (e.g. Rutherford, 2005; reviewed in Allely, 2013). However, experimental research has found that compared to typically developing participants, people with ASD have at least comparable (e.g. Bird et al., 2010; Duerden et al., 2015), if not higher pain sensitivity (e.g. Cascio et al., 2008; Fan et al., 2014; Riquelme et al., 2016). Pain reactivity in ASD has mostly been assessed by determining pain threshold, i.e. the lowest level of stimulation at which an individual feels pain. Less is known about how people with ASD evaluate suprathreshold pain, i.e. pain of intensities that go beyond this lowest level. This is important because there is more to pain experience than the threshold at which pain is detected. For instance, it is not clear that someone who detects pain earlier, i.e. at lower stimulation intensity, will consistently evaluate suprathreshold stimulation as more intense. By covering a wider stimulus range, this study captures pain reactivity beyond pain detection. Another important aspect that has been overlooked in research so far is the negative valence that
people with ASD attribute to pain, i.e. how unpleasant they experience a painful stimulus to be. This study looks at both of these two components of pain reactivity: how people with ASD evaluate pain stimulation in terms of its intensity and unpleasantness.

Importantly related to this is the question of how people with ASD interpret others’ pain expressions. Research in ASD has traditionally focused more on the ability to correctly label emotions rather than on the judging of emotion intensity (reviewed in Harms et al., 2010). There is some evidence suggesting that when people with ASD are asked to rate the strength of emotional stimuli, they tend to provide more moderate (i.e. less intense) ratings than neurotypical controls (e.g. Gebauer et al., 2014). People with ASD also seem to perform worse at recognizing emotions in facial expressions when the expressed emotional intensity is lower (e.g. Doi et al., 2013; Law Smith et al., 2010; Wong et al., 2012). Pain provides an excellent example of an emotion which can often be understood and labelled from context, e.g. by seeing how another person’s body is harmed. Yet, to our knowledge, no study has investigated how people with ASD evaluate the intensity of others’ pain from bodily or facial expressions. By focusing on how others’ pain expressions are perceived and experienced when context is known, this study aims to investigate pain perception independently from the interpretation of context and the ability to label emotions. Observing others’ painful expressions may also trigger feelings of unpleasantness. It is unclear whether emotional
responses to others’ expressions are different in people with ASD. This study thus investigates two components of the perception of pain in others: how people evaluate others’ pain in terms of intensity, and how they respond to it emotionally in terms of unpleasantness.

Taken together, this study relates participants’ own pain reactivity, in the form of perceived intensity and unpleasantness, to how participants perceive and experience pain conveyed in the facial expressions of others. It compares two explanations for emotion perception challenges in ASD, tackling the question of whether these stem from a general difficulty in perceiving emotions, including in oneself, or are limited to the perception of others’ emotions. Based on current findings, we expect participants with ASD to perform worse in estimating others’ pain intensity. Considering known difficulties with facial expressions of low emotional intensity, it is also possible that those with ASD experience others’ painful expression as less unpleasant. If emotion perception difficulties include the self, participants with ASD should show lower reactivity to pain stimulation, i.e. lower intensity ratings and less unpleasantness during stimulation. In this case, reactivity to pain and pain evaluation in others should also be associated with alexithymic traits. In contrast, if difficulties are limited to perceiving others’ pain in others, responses to own pain should be at least comparable to the control group. By looking at different suprathreshold pain intensity levels and by distinguishing perceived intensity from experienced unpleasantness, we investigate
different aspects of pain reactivity in people with ASD. Relating self-evaluation, i.e. individual pain experience, to other-evaluation, i.e. the way individuals evaluate and experience pain in others, could provide important insights into the connection of sensory and social symptoms.

Methods

Participants

Thirty-two male adults (mean age = 25.0 years, range = 20 to 36 years) participated in the study. Out of these, sixteen had a formal diagnosis of Autism or Asperger syndrome (International Classification of Diseases, 10th revision, ICD-10; World Health Organization, 1992). Participants without ASD signed up through a web-based participant recruitment system of Aarhus University. Participants with ASD were recruited through the national autism and Asperger’s association, assisted living services for young people with ASD, and specialized educational facilities. People are referred to these associations on the basis of receiving a formal diagnosis of ASD by a specialized psychiatrist; hence they were expected to meet the diagnostic requirements for this study. The verbal and general intelligence of the ASD group were within the normal range (see Table I). All participants were right-handed and Danish native speakers. Sample size was limited by the availability of individuals with a diagnosis of
ASD but no comorbidities or intake of medication, as we aimed to compare relatively homogeneous groups. Exclusion criteria were: intake of medication, presence of pain disorders, presence of other psychological or physiological conditions (other than ASD), and any history of brain damage or neurosurgery. Participants read about these criteria before they signed up for the study. During the first contact (via e-mail or phone) they were asked about medication, neurological conditions and other psychiatric diagnoses. Those who reported that they did take medication or had another relevant diagnosis were not included in the study. Also, some did not reply to the email, which might indicate that they did not meet the inclusion criteria or that they did not want to participate for some other reason. Finally, to confirm that participants met all criteria they attended a screening interview with one of the researchers on the day of participation in the study. To ensure that all participants have at least average intelligence (IQ > 70), all participants underwent testing with the Danish version of the Wechsler’s Adult Intelligence Scale 4 (WAIS-IV, Wechsler et al., 2008). Participants signed an informed consent form and received 300 Danish Crowns (DKK) as compensation for taking part in the study. The study was approved by the local ethics committee and carried out in accordance with the ethical standards of the Declaration of Helsinki.

**Questionnaire measures**
The 20-item Toronto Alexithymia Scale (TAS-20) is a self-report measure assessing alexithymic traits. Sample items from three respective subscales are: “I am often confused about what emotion I am feeling” (subscale: Difficulty identifying feelings), “It is difficult for me to find the right words for my feelings” (subscale: Difficulty describing feelings) and “I prefer talking to people about their daily activities rather than their feelings” (subscale: Externally oriented thinking). The TAS-20 has good internal (Cronbach’s alpha = 0.81) and test-retest (r = 0.77) reliability (Bagby et al., 1994a; Bagby et al., 1994b). In samples with ASD, the TAS-20 correlates highly with scores in the Bermond Vorst Alexithymia Questionnaire (BVAQ-B; Vorst & Bermond, 2001) (e.g. Berthoz and Hill, 2005; Bird et al., 2010; Cook et al., 2013). Compared to the BVAQ-B, the TAS-20 appears to have superior re-test reliability and discriminant validity (Berthoz and Hill, 2005).

The Interpersonal Reactivity Index (IRI) is a measure of self-reported empathy that focuses on four distinct aspects. The perspective taking (PT) subscale assesses the tendency of adopting others’ point of view in everyday life. The empathic concern (EC) subscale assesses feelings of warmth and compassion for other people. The personal distress (PD) subscale investigates the tendency to feel unease and discomfort in emotional interpersonal settings. The fantasy (F) subscale measures the tendency of imagining oneself in the place of fictional characters in movies or books. Sample items are: “I try to look at everybody's side of a disagreement before I make a decision” (PT)
and “I often have tender, concerned feelings for people less fortunate than me” (EC). Subscales have good internal (Cronbach’s alpha = 0.70-0.78) and test-retest (r = 0.61-0.81) reliability (Davis, 1980). For young adults with ASD, an internal reliability (Cronbach’s alpha) of 0.85 was found for a Dutch version (Demurie et al., 2011). The IRI subscales EC and PT correlate with the Empathy Quotient, indicating adequate convergent validity (Lawrence et al., 2004). Further, two studies involving adults with ASD found a negative correlation between scores of the IRI and the TAS-20 (Bird et al., 2010, Silani et al., 2008).

The Pain Catastrophizing Scale (PCS) assesses self-reported catastrophic thinking in connection with pain. It consists of three subscales focusing on magnification of pain, rumination about pain, and helplessness in response to pain. Sample items are: “When I’m in pain, I keep thinking about how much it hurts” (subscale: Rumination), and “When I’m in pain, I feel I can’t go on” (subscale: Helplessness). Subscales show good to excellent internal reliability (Cronbach’s alpha = 0.66-0.87) (Sullivan et al., 1995). Reliability estimates have been confirmed in further studies (e.g. Meyer et al., 2008), which have also provided evidence for adequate concurrent and discriminant validity in clinical and non-clinical samples (Osman et al., 2000).

Experimental paradigm
Pain stimuli were delivered with a constant current stimulator (Model DS7A, Digitimer, Hertfordshire, UK) and a concentric electrode (WASP, Speciality Developments, Kent, United Kingdom). Pulses were delivered in square waveform pulse trains of 500 $\mu$s = 0.5 ms duration to the back of the left hand. A random inter-stimulus interval of 300-500 ms with a mean of 400 ms and 10 stimulations for the total stimulation time of 3 s was chosen in order to minimize habituation. To calibrate stimuli to subjective pain intensity levels based on individual sensitivity, we used the method of limits. Participants rated test impulses on a scale of 0 to 100, where 0 refers to ‘no pain’ and 100 refers to the ‘worst pain imaginable’. Test impulses started at an amplitude of 0 milliamperes (mA) and increased with increments of around 0.1 mA until the participant rated a stimulus as painful. Then the intensity of test impulses was decreased again, until the participant rated a stimulus as not painful. The two amplitude levels obtained by these increasing and decreasing application series were then averaged to determine level 0, i.e. the highest stimulation intensity that a participant did not experience as painful. The same procedure of ascending and descending stimulus series, and averaging of resultant amplitude levels, was repeated to determine levels 20, 40, and 60. Then the entire procedure was repeated and intensity levels obtained in the second run were used for pain stimulation.

Participants saw videos of shoulder patients carrying out a standardized range-of-motion test used in physiotherapy assessment, which involved abduction of the
affected shoulder (Prkachin and Solomon, 2008). Before watching the videos, participants were made aware of this context; hence they knew that these patients were in a situation that was potentially painful for them. Moving the affected shoulder can elicit varying levels of pain intensity. Patients filmed in these test sequences were asked to repeatedly rate the intensity of their spontaneous pain experience on a scale ranging from 0 (‘no pain’) to 100 (‘worst pain imaginable’). We used these patient ratings to sample a subset of videos with ratings of 0, 20, 40, and 60, thus corresponding to the calibrated intensity levels of pain stimuli in this study. The final selection of videos presented in this study thus depicted seven patients from each of whom we could obtain pain expressions at the four respective intensity levels 0, 20, 40, and 60. All videos show the face and neck of patients during abduction of the affected shoulder. Videos had durations ranging from 4 to 7 seconds and depict an initially neutral facial expression that gradually changes over time.

**Measures**

After each pain stimulus, participants rated on visual analogue scales how intense their pain felt to them, and how unpleasant they perceived this pain to be. This type of visual analogue scale is widely used and is considered a reliable form of pain assessment (Hjermstad et al., 2011). It has a high test-retest reliability and good convergent validity (e.g. Bijur et al., 2001; Gallagher et al., 2002). Unlike the number scale used during
calibration, these scales had verbal labels on each end (‘no pain’ to ‘worst pain imaginable and ‘no unpleasantness’ to ‘worst unpleasantness imaginable). When watching others in pain, participants also used these scales. They were asked to rate how intense they thought the pain to be for the patient, and how unpleasant it was for them to observe the facial pain expression of the patient.

The experiment was thus a 2 (diagnosis = Control vs. ASD) × 2 (stimulus condition = SELF vs. OTHER) mixed factorial design with rated pain intensity and rated pain unpleasantness as two separate dependent variables. Importantly, stimulus condition referred to the person experiencing the pain, which was not always equivalent to the target of emotion inference. Specifically, in the OTHER condition of unpleasantness, participants rated their own unpleasantness, thus inferring their own emotional response to others’ pain. To further assess evaluation of others’ pain, intensity ratings were compared to respective pain intensity levels of stimuli (0, 20, 40, and 60).

Procedure

Upon arrival, participants received instructions in both written and oral form, signed the consent form and filled in questionnaires. The main part of the experiment was then completed in a different room where participants were seated comfortably in a chair in front of a computer. In the first part of the study participants underwent the calibration sequence described above and then received pain stimulation. Twenty-one individually
calibrated pain impulses were administered according to a pseudorandom protocol including each intensity level five times. The first stimulus was applied at intensity level 0 to establish a common starting level and was later discarded from analysis. To avoid systematic order effects, half of the participants received stimuli in the inverted order. A dividing wall separated participants from the researcher controlling the pain stimulator apparatus, in order to limit confounding effects of feeling observed. After each pain impulse, participants rated its intensity and unpleasantness. In the second part, participants watched the videos of others in pain while alone in the room. Twenty-eight videos were presented in a fixed, pseudorandom order and were each followed by ratings of intensity and unpleasantness.

Results

Data was analyzed using R software (version 3.2.2, R Core Team, 2015) and packages ez (version 4.3, Lawrence, 2015), car (Fox and Weisberg, 2011), effsize (version 0.6.1, Torchiano, 2016) and lsr (version 0.5, Navarro, 2015). Pain intensity and pain unpleasantness ratings were transformed from horizontal positions of mouse clicks to a scale between 0 and 100.

Participant data
Demographic characteristics and mean questionnaire scores for different groups are displayed in Table I. Independent t-tests showed that groups were comparable in terms of age, self-reported empathy on the IRI, alexithymia, and pain catastrophizing. There was an almost significant group difference in mean intelligence. However, with the lowest IQ score being 78, all IQ scores were above the lower cut-off of what is considered average intelligence (IQ > 70).

**Table I.** Demographic characteristics and mean questionnaire scores in the whole sample, and separate for the control group and the group with autism spectrum disorder (ASD).

<table>
<thead>
<tr>
<th></th>
<th>Total (n=32): Mean (SD)</th>
<th>Control (n=16): Mean (SD)</th>
<th>ASD (n=16): Mean (SD)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>25.00 (4.24)</td>
<td>24.50 (2.73)</td>
<td>25.50 (5.40)</td>
<td>0.52</td>
</tr>
<tr>
<td>TAS-20</td>
<td>44.19 (11.77)</td>
<td>42.13 (10.30)</td>
<td>46.25 (13.09)</td>
<td>0.33</td>
</tr>
<tr>
<td>IRI</td>
<td>90.59 (10.64)</td>
<td>92.19 (9.25)</td>
<td>89.00 (11.96)</td>
<td>0.41</td>
</tr>
<tr>
<td>PCS</td>
<td>14.53 (8.68)</td>
<td>14.50 (6.80)</td>
<td>14.56 (10.46)</td>
<td>0.98</td>
</tr>
<tr>
<td>WAIS</td>
<td>107.63 (12.45)</td>
<td>111.75 (11.13)</td>
<td>103.50 (12.67)</td>
<td>0.06</td>
</tr>
<tr>
<td>Perspective-taking (IRI)</td>
<td>25.09 (4.01)</td>
<td>26.19 (3.37)</td>
<td>24.00 (4.40)</td>
<td>0.13</td>
</tr>
<tr>
<td>Fantasy (IRI)</td>
<td>24.03 (4.72)</td>
<td>24.38 (3.12)</td>
<td>23.69 (6.01)</td>
<td>0.69</td>
</tr>
<tr>
<td>Empathic concern (IRI)</td>
<td>22.44 (3.19)</td>
<td>23.13 (2.78)</td>
<td>21.75 (3.51)</td>
<td>0.23</td>
</tr>
<tr>
<td>Personal distress (IRI)</td>
<td>19.03 (4.71)</td>
<td>18.50 (4.49)</td>
<td>19.56 (5.02)</td>
<td>0.53</td>
</tr>
</tbody>
</table>

SD: standard deviation
Mean calibrated stimulation intensity levels for different groups are displayed in Table II. To test for differences in individually calibrated intensity levels, we entered stimulation intensity (i.e. mA at levels 0, 20, 40, and 60) in a mixed ANOVA, using intensity levels as within-subjects factor and diagnosis (Control vs. ASD) as between-subjects factor. There was a significant effect of intensity (Greenhouse-Geisser adjusted $p < 0.01$, generalized eta squared $= \eta^2_G = 0.37$), but no effect of diagnosis ($p = 0.62$).

**Table II.** Mean calibrated stimulation intensities (mA) in the whole sample, and separate for the control group and the group with autism spectrum disorder (ASD).

<table>
<thead>
<tr>
<th></th>
<th>Total (n=32): Mean (SD)</th>
<th>Control (n=16): Mean (SD)</th>
<th>ASD (n=16): Mean (SD)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pain intensity level 0</td>
<td>1.58 (0.91)</td>
<td>1.49 (1.04)</td>
<td>1.66 (0.79)</td>
<td>0.60</td>
</tr>
<tr>
<td>Pain intensity level 20</td>
<td>5.27 (3.33)</td>
<td>4.65 (2.08)</td>
<td>5.89 (4.21)</td>
<td>0.30</td>
</tr>
<tr>
<td>Pain intensity level 40</td>
<td>7.40 (4.44)</td>
<td>6.68 (3.15)</td>
<td>8.13 (5.45)</td>
<td>0.37</td>
</tr>
<tr>
<td>Pain intensity level 60</td>
<td>11.23 (7.54)</td>
<td>11.34 (7.71)</td>
<td>11.13 (7.63)</td>
<td>0.94</td>
</tr>
</tbody>
</table>

SD: standard deviation

**Rated pain intensity in self and others**
To test for differences in perceived pain intensity, we entered intensity ratings in a mixed ANOVA, using stimulus condition (SELF vs. OTHER) as within-subjects factor and diagnosis (Control vs. ASD) as between-subjects factor. Mean intensity ratings for own pain at different intensity levels are depicted in Figure 1(a). There was a significant effect of stimulus condition \( (p < 0.01, \eta^2_G = 0.09) \) and an interaction effect of stimulus condition and diagnosis \( (p < 0.01, \eta^2_G = 0.07) \). Post-hoc independent t-tests detected no significant differences between diagnostic groups in intensity ratings in the SELF condition or in the OTHER condition \( (\text{both FDR-corrected } p = 0.16) \). Paired t-tests detected a significant difference between SELF and OTHER conditions within the group with an ASD diagnosis \( (M_{\text{SELF}} = 33.35, M_{\text{OTHER}} = 20.75; \text{FDR-corrected } p = 0.01, \text{Cohen’s } d = 0.89) \), but not in the control group \( (M_{\text{SELF}} = 27.13, M_{\text{OTHER}} = 26.38; \text{FDR-corrected } p = 0.76) \). Levene’s tests for homogeneity of variances were non-significant \( (\text{both } p \geq 0.12) \). To test for associations with alexithymia, we computed Pearson correlations of alexithymia scores with mean pain intensity ratings. Alexithymia was not associated with rated pain intensity in either condition; not overall and not within groups \( (\text{all FDR-corrected } p \geq 0.79) \). To test whether intensity of own pain differed between groups after keeping intelligence constant we ran an ANOVA using IQ scores as a covariate. We did not detect any significant differences in mean pain intensity ratings \( (p = 0.25) \). Rated intensity of own pain was not associated with pain catastrophizing \( (p = 0.70) \). Estimated intensity of others’ pain was not associated
with self-reported empathy, nor with the subscale of perspective taking (FDR-corrected \( p \geq 0.46 \)).

**Rated pain intensity in others at different intensity levels**

To test for deviations of perceived pain intensity in others from the actual intensity levels, we performed four one sample t-tests separate for each diagnostic group (Control vs. ASD) with levels 0, 20, 40, and 60 as respective true values of the mean. Mean intensity ratings for others’ pain at different intensity levels are depicted in Figure 1(b). In the ASD group, mean rated pain intensity at each level was significantly different from the actual intensity (mean rated pain intensity at level 0 = \( M_0 = 7.04 \), FDR-corrected \( p_0 < 0.01 \), Cohen’s \( d_0 = 1.44 \); \( M_{20} = 11.07 \), FDR-corrected \( p_{20} < 0.01 \), Cohen’s \( d_{20} = 1.22 \); \( M_{40} = 28.07 \), FDR-corrected \( p_{40} = 0.01 \), Cohen’s \( d_{40} = 0.85 \); \( M_{60} = 36.80 \), FDR-corrected \( p_{60} < 0.01 \), Cohen’s \( d_{60} = 1.59 \)). In the control group, mean rated pain intensity was significantly different from actual intensities at level 0 (\( M_0 = 9.29 \), FDR-corrected \( p_0 < 0.01 \), Cohen’s \( d_0 = 1.09 \)) and level 60 (\( M_{60} = 42.83 \), FDR-corrected \( p_{60} < 0.01 \), Cohen’s \( d_{60} = 0.90 \)). This means that both groups overestimated pain intensity at intensity level 0. For the videos showing painful expressions, the ASD group underestimated pain intensity at all levels, whereas the control group only underestimated pain at the highest intensity level.
Associations of rated pain intensity in self and others

To test for differences in associations of perceived pain intensity in self and other, we computed Pearson correlations for each group (Control vs. ASD). Associations of pain intensity ratings for different groups are depicted in Figure 1(c). In the control group, there was a significant association of pain intensity ratings between the SELF condition and the OTHER condition ($r = 0.62$, FDR-corrected $p = 0.02$). This association was not found in the group with an ASD diagnosis (FDR-corrected $p = 0.35$). An independent t-test detected no significant difference between these two correlations ($p = 0.23$).

Figure 1. Self- and other-evaluations of pain intensity in participants with ASD and control participants

Rated pain unpleasantness in self and others

To test for differences in experienced pain unpleasantness we entered unpleasantness ratings in a mixed ANOVA, using stimulus condition (SELF vs. OTHER) as within-subjects factor and diagnosis (Control vs. ASD) as between-subjects factor.

Unpleasantness ratings for own pain and for others’ pain are depicted in Figure 2. There was a significant effect of stimulus condition ($M_{\text{SELF}} = 22.30$, $M_{\text{OTHER}} = 4.87$, $p < 0.01$, $\eta^2_G = 0.35$), but no effect of diagnosis ($p = 0.82$), and no interaction effect of
stimulus condition and diagnosis ($p = 0.14$). Levene’s tests for homogeneity of variances were non-significant (both $p \geq 0.11$). Alexithymia scores were not associated with unpleasantness ratings for others’ pain (FDR-corrected $p = 0.64$), but there was an unexpected positive correlation with rated unpleasantness of own pain. Further exploration showed that this result was driven by a single outlier with a TAS-20 score more than two standard deviations above the mean. After excluding this participant, the correlation test was no longer significant (FDR-corrected $p = 0.64$). To test whether unpleasantness of own pain differed between groups after keeping intelligence constant we ran an ANOVA using IQ scores as a covariate. We did not detect any significant differences in mean pain intensity ratings ($p = 0.39$). Experienced unpleasantness of own pain was not associated with pain catastrophizing ($p = 0.14$). Experienced unpleasantness for others’ pain was not associated with self-reported empathy, or with the subscale of empathic concern (both FDR-corrected $p = 0.88$).

[Insert Figure 2]

**Figure 2.** Experience of pain unpleasantness for self and others in participants with ASD and control participants

**Discussion**

It has been suggested that people with ASD experience difficulties with recognizing
others’ emotional states due to general problems of relating to emotional experience, be it own experience or that of others. Conversely, findings of atypical processing of social information in ASD point to the account that difficulties in emotion processing may be specific to the perception of others. This study aimed to compare these two explanations by linking emotional and sensory perception, looking at pain evaluation in self and others. Further, it focused on two aspects of pain perception and experience – pain intensity and pain unpleasantness.

Participants with and without ASD showed no difference in evaluating the intensity of their own pain. This finding was further corroborated by the fact that their individually calibrated pain intensity levels were comparable on average. When relating these results to previous studies it is important to consider the varying types of pain these have focused on. To our knowledge, no previous study has compared evaluations of pain intensity in individuals with and without ASD using suprathreshold stimulation. One study used a suprathreshold stimulus, but focused on comparing pain unpleasantness (Bird et al., 2010). However, in line with the present study, it did not detect a group difference in calibrated pain intensity levels, providing some indication for normal pain reactivity. This study also resembled the present study in two other aspects: it tested samples with comparable levels of alexithymia, and used electrical pain stimulation. Another previous study found normal pain detection thresholds in ASD using thermal pain stimulation (Duerden et al., 2015). However, our results
contradict the findings of other experimental studies on thermal pain (Cascio et al., 2008), and on pressure pain (Fan et al., 2014; Riquelme et al., 2016), as well as case reports describing the experience of various types of pain (reviewed in Allely, 2013). One important difference to previous research is that this study covered pain perception on a broader range. Aside from mere pain detection, we also investigated how people evaluate stimulation at different suprathreshold intensity levels. It should be mentioned, however, that this experimental setup specifically instructed participants to pay attention to their own bodily experience. This is probably quite different from case studies reporting on everyday life experiences, in which other factors could distract individuals with ASD from paying attention to bodily pains.

Our hypothesis that participants with ASD have more difficulties estimating others’ pain intensity found some support. Both groups underestimated high intensity pain expressions, which is in line with previous research demonstrating that people tend to show an underestimation bias when judging others’ pain intensity from their expressions or reports (Prkachin et al., 2006). However, only the group with ASD showed a more generalized form of pain underestimation bias, which was also present at lower pain levels. Both groups overestimated pain intensity of others at the non-painful level 0, i.e. in those videos in which the depicted patients reported a pain intensity of 0. While this was not explicitly discussed in the instructions, the inflation of ratings might be due to implicit expectations of the participants that every video
presented would depict a patient in pain. Importantly, participants with ASD generally seemed to be able to distinguish dynamic facial expressions in terms of intensities, but they underestimated pain intensity at all levels (except level 0), whereas control participants only did so at the highest level. We are aware of one previous study that investigated how individuals with ASD estimate pain intensity of others (Krach et al., 2015). In that study, people assessed intensity based on still images of hands and feet in painful situations, such as an accidental cutting of a finger with scissors. No differences between groups with and without ASD were found in estimated pain intensity and associated pupil dilation and brain activation. These previous results suggest that people with ASD do not have any difficulties inferring emotions from a painful context. However, as the current study implies, this emotional inference becomes more challenging for them when it can merely be based on facial expressions of pain.

Comparing self- and other-evaluations, we found that participants with ASD rated their own pain intensity to be considerably higher than the perceived pain intensity of others. The opposite pattern was observed for participants without ASD, whose pain intensity ratings for self and others were not only comparable on average, but also positively associated. One might argue that a lack of difference between rated intensity of own pain versus others’ pain does not necessarily imply that people were successful at identifying others’ pain. Considering that patients in the videos experienced a different type of pain, it is possible that they used pain scales in different ways than participants
in the present study. We tried to account for this by selecting videos of patients who rated their pain at the same four intensity levels that were used in pain stimulation. Second, we used a pain stimulation protocol aimed at minimizing habituation to pain stimulation, to ensure that pain intensity ratings over time would reflect the four intensity levels calibrated to individuals’ sensitivity. Third, we also compared estimated intensity of others’ pain to the actual pain intensity that patients reported, revealing a similar picture of differences between participants with and without ASD.

Participants with and without ASD did not differ in how unpleasant they experienced their own pain. This matches the findings of Bird et al. (2010), who observed comparable unpleasantness ratings in a sample matched for alexithymia levels. This points to a typical experience of own pain in ASD, with regard to both extent (i.e. intensity) and negative emotional valence (i.e. unpleasantness). There was also no difference between participants with and without ASD in how unpleasant they experienced observing others’ pain. This means that participants with ASD showed typical emotional responses to others’ pain, despite underestimating others’ pain intensity. Here it is important to note that ratings of pain intensity and ratings of pain unpleasantness differed in the form of self-other distinction they involved. Unpleasantness ratings assessed the emotional response to observing others in pain, thus asking participants to infer their own emotions in response to others’ pain. Pain intensity ratings assessed the interpretation of others’ pain, thus asking participants to
infer others’ emotions. Results indicate that inferring others’ emotions seems to be more challenging for individuals with ASD. Intriguingly, most previous research focusing on emotional responses to others in ASD has yielded similar results. One study examined neural activation during the viewing of dynamic facial expressions of pain, finding no difference between individuals with and without ASD (Hadjikhani et al., 2014). While the authors did not directly assess emotional responses to these pain expressions, brain activation was associated with emotional empathy scores. Results were thus interpreted as evidence for intact emotional contagion in ASD. This interpretation is in line with the lack of difference in unpleasantness responses observed in the present study. Yet, our findings on pain intensity indicate that differences in neural activation would be expected if participants completed the more complex task of interpreting the emotional intensity of these facial expressions. In fact, a similar dissociation in neural processing has been observed in a previous study which used pain context rather than bodily pain expressions (Fan et al., 2014). In that study, individuals with ASD showed typical emotional responses, but reduced neural activation in response to stimuli that had a higher social complexity. Previous studies have also investigated empathic concern for others’ facial emotion expressions in ASD using the Multifaceted Emotion Task. Our findings are in line with one of these studies, which observed no differences in empathic concern (Dziobek et al., 2008). Another study showed contradicting results, observing lower empathic concern for negative
emotions in ASD (Mazza et al., 2014). None of these previous studies assessed levels of alexithymia, which could be one explanation for diverging results. In a sample matched for alexithymia levels, Bird et al. (2010) observed no differences in reported unpleasantness related to others experiencing high intensity pain. When others’ pain was of lower intensity, individuals with ASD felt even more unpleasant for them than those without ASD. There were also no overall differences in neural activation after accounting for alexithymia. This study differed from the current study in several aspects: pain in others was potentially more salient, involving live pain stimulation delivered to people that participants had a significant relationship with. Participants did not see the others’ faces, and thus empathic concern was independent of individuals’ ability to interpret emotional expressions. Yet, findings of the current study are in line with the overall conclusion that individuals with ASD show emotional responses to others’ pain that are at least as strong as those of people with matched alexithymia levels. For the current study, mean unpleasantness experienced during video watching was rather low in both groups, and there were no associations with self-reported empathy. It is possible that the facial expressions presented in this study were not sufficient to provoke strong emotional responses, and thus not sensitive enough to reveal differences. However, in light of previous findings it appears plausible that individuals with ASD show typical emotional responses to others’ pain.
Importantly, the present study found no difference in alexithymic traits between participants with and without ASD, and average TAS-20 scores in both groups were below the threshold of what would indicate possible alexithymia. Had we tested a sample with ASD showing a more representative average level of alexithymic traits, we might have seen differences in pain evaluation caused by alexithymia. TAS-20 scores were not associated with pain reactivity, nor were they related to the experience and evaluation of others’ pain. One limitation of this study is the relatively small sample size, which might explain why we did not detect some associations. However, our findings match those of previous studies with individuals with ASD, observing no associations of alexithymia with pain or empathy for others’ pain (Bird et al., 2010; Silani et al., 2008).

This study discussed two alternative accounts of emotion perception difficulties in ASD. Findings do not support the idea that people with ASD have general deficits in relating to emotions. However, if this study had revealed different pain experience in ASD, there are several conditions specific to noxious stimulation that might have explained this difference, such as a lower number of pain receptors or altered synaptic transmission of pain signals. A recent study on embarrassment found that emotional responses in those with ASD prevailed more over time, which might point to challenges in emotion regulation (Adler et al., 2015). Thus, to clarify to what extent people with ASD experience problems relating to emotions, it will be important to
focus on different types of emotions. With regard to the second account we discussed, findings of the current study do provide some support. The idea that inferring emotions is difficult for individuals with ASD when emotional information is conveyed in facial expressions is in line with the pain underestimation bias we observed. However, pain underestimation bias is a common phenomenon in the general population (Prkachin et al., 2007), raising the question of whether the increased difficulties observed in ASD are specific to this emotion. While there is increasing evidence that individuals with ASD have no difficulties inferring emotion intensity from contexts of pain (Krach et al., 2015) or embarrassment (Adler et al., 2015), there seems to be a profound lack of research that focuses on inferring such information from facial or bodily expressions. Importantly, it is also not clear from this second account whether difficulties in interpreting facial expressions concern the processing of facial cues per se. Inferring emotions from faces is a complex task that is influenced by context and individual expectations (e.g. Barrett et al., 2011). In the present study, participants with ASD might have expected that individuals without ASD express emotions in a more intense fashion than themselves, which could have caused them to downgrade their estimations of others’ pain intensity. Taken together, a definite answer to overarching accounts of emotion perception difficulties in ASD requires additional research focusing on facial and bodily expressions, involving different types of emotions, and addressing the role of individual expectations.
There seemed to be a discrepancy between the evaluation of own pain states and others’ pain states in ASD, as opposed to comparable and positively associated self- and other evaluations in the control group. One possible explanation for these findings could be that individuals without ASD calibrated their estimates of others’ pain more to their own experience. Embodied accounts of social cognition have suggested that interoceptive processing contributes to the recognition of emotions in others (e.g. Fukushima et al., 2011; Kragel and LaBar, 2016; Ondobaka et al., 2015). A pain stimulation task might momentarily increase interoceptive responsiveness, making individuals more sensitive to the pain experience of others in the subsequent task. However, the extent to which individuals with ASD use their interoceptive experience to evaluate others’ states might be different. Indeed, a recent study demonstrated that individuals with ASD relied less on emotional and interoceptive information when making decisions, and were less manipulated by emotional interference, compared to a control group matched on alexithymia levels (Shah et al., 2016). Following this line of thought, the discrepancy we see in participants with ASD could mean that they evaluate their own pain in a typical manner, but integrate these sensory and emotional experiences less when evaluating others’ pain experiences. This interpretation puts forward a possible third explanation for difficulties in emotion perception in ASD, in the form of a weaker link between interoceptive experience and emotion perception despite intact perception of own emotions. Future research could investigate this
explanation by systematically manipulating the order in which own experience and evaluation of others are assessed.

**Conclusion**

This study found no evidence for atypical pain reactivity in ASD. Participants with ASD perceived suprathreshold pain stimulation to be as intense and unpleasant as those without ASD. Further, groups showed comparable unpleasantness responses to observing others’ pain expressions. However, participants with ASD estimated the pain intensity of others to be lower than their own, displaying an underestimation bias at both lower and higher intensity pain expressions. We found a diverging pattern for the control group, with a significant association between the intensities participants ascribed to their own pain and to others’ pain. To put these results into perspective we related them to two alternative accounts of emotion perception difficulties. Our findings favor the account that individuals with ASD evaluate their own emotional states like individuals without ASD, but specifically have difficulties with perceiving the emotion states in the facial expressions of others. The observed discrepancy between self- and other evaluations might indicate a weaker link between interoceptive experience and emotion perception in ASD. This study contributes to the picture of pain reactivity in ASD, helps constrain the theoretical mechanisms of emotion perception difficulties in ASD and relates sensory, emotional and social aspects of
autistic experience.

References


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