Effects of Autistic Traits on Pain Empathy: The Mediating Role of Pain-Related Negative Emotion and Cognition (Postprint)

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Abstract

Previous research has suggested that pain empathy deficits in individuals with autism may originate from abnormalities in self-pain experience. Given the similarity between high autistic traits and autism, the present study evaluated state and trait empathy through experimental paradigms and questionnaire surveys, respectively, aiming to characterize the associations among autistic traits, self-pain, and empathy. In a pseudo-dyadic pain empathy paradigm, individuals with high autistic traits showed enhanced P2 responses and higher unpleasantness ratings when observing others receiving pain, which was partially attributable to elevated fear of pain. Questionnaire results revealed that higher autistic trait levels were associated with lower scores on the perspective-taking dimension of the empathy trait scale, but higher scores on the personal distress dimension, with pain fear and catastrophizing mediating the relationship between autistic traits and personal distress. Thus, individuals with high autistic traits exhibited heightened state and trait emotional empathy, partially stemming from more negative emotional and cognitive responses toward pain. This supports the perspective that empathy deficits and other social dysfunctions in individuals with high autistic traits and autism can be understood from the angle of abnormal self-pain experience, offering potential targets for clinical treatment and intervention.

Full Text

The Influence of Autistic Traits on Pain Empathy: The Mediating Role of Pain-Related Negative Emotion and Cognition

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Abstract

Previous research suggests that pain empathy deficits in individuals with autism spectrum disorder (ASD) may stem from abnormalities in first-hand pain experience. Given the similarities between high autistic traits and ASD, the present study assessed state and trait empathy through experimental paradigms and questionnaire surveys to characterize the associations among autistic traits, first-hand pain, and empathy.

In a pseudo-dyadic pain empathy paradigm, individuals with high autistic traits exhibited stronger P2 responses and higher unpleasantness ratings when witnessing others receiving painful stimulation, which were partially attributable to high levels of pain-related fear. Questionnaire survey results revealed that higher levels of autistic traits were associated with lower perspective-taking scores and higher personal distress scores on trait empathy scales, with pain fear and catastrophizing mediating the link between autistic traits and personal distress. Thus, individuals with high autistic traits demonstrate heightened state and trait emotional empathy, partially originating from more negative emotions and cognitions toward pain. This supports understanding empathy and other social functioning deficits in high autistic traits and ASD populations from the perspective of first-hand pain abnormalities, providing potential targets for clinical treatment and intervention.

Keywords: autistic traits, pain empathy, pain fear, pain catastrophizing, event-related potentials
Classification Number: R395

According to the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5), autism spectrum disorder (ASD) is defined as a neurodevelopmental disorder characterized by two core symptom domains: "deficits in social communication and social interaction" and "restricted, repetitive patterns of behavior and interests" (American Psychiatric Association, 2013). Autistic traits represent a constellation of behaviors, personality characteristics, and cognitive features associated with ASD (Sucksmith et al., 2011). These traits exist across both ASD and general populations, differing only in severity (Baron-Cohen,

Wheelwright, Skinner et al., 2001). The mental and physical health issues and social functioning problems of individuals with high autistic traits cannot be ignored: compared with those with low autistic traits, individuals with high autistic traits experience greater psychological distress and higher suicide risk (Dow et al., 2021; Pelton & Cassidy, 2017), exhibit relatively poorer social skills, show weaker motivation for interpersonal interaction, and demonstrate fewer prosocial behaviors.

Empathy refers to the capacity to feel and understand others' emotions and thoughts (Singer & Lamm, 2009) and plays a crucial role in interpersonal communication and prosocial behavior (Decety & Jackson, 2006; Jackson et al., 2005). Empathy primarily involves two components: emotional empathy, which entails perceiving and sharing others' emotional states, and cognitive empathy, which enables accurate understanding of others' thoughts, including perspective-taking and theory of mind abilities (Decety & Lamm, 2006; Decety & Svetlova, 2012). Baron-Cohen (2010) proposed that empathy deficits may constitute a primary contributor to social impairments in ASD. Previous research has consistently found cognitive empathy deficits in individuals with ASD (Song et al., 2019; Ziermans et al., 2019), whereas findings regarding emotional empathy remain controversial. Some studies have reported heightened emotional empathy in ASD individuals (Capps et al., 1993; Rogers et al., 2007). For instance, Gu et al. (2015) found that compared with control groups, the ASD group exhibited stronger skin conductance responses and greater anterior insula activation when viewing bodily pain images (e.g., a knife cutting a finger). Conversely, other studies have reported reduced emotional empathy in ASD individuals (Minio-Paluello et al., 2009), such as lower unpleasantness ratings after watching videos of others describing their painful experiences (Santiesteban et al., 2021). Still other research has found no significant differences in emotional empathy between ASD and typical individuals (Bird et al., 2010; Blair, 1999), such as comparable arousal ratings and sympathy scores for painful scenario images between ASD and control groups in multidimensional empathy tasks (Dziobek et al., 2008). Therefore, although the notion of empathy deficits in ASD is widely accepted, specific manifestations remain debated, and abnormalities in emotional empathy and their underlying mechanisms require further investigation.

Neuroimaging studies have demonstrated that experiencing pain firsthand and witnessing others in pain activate similar neural representations, including the anterior insula, anterior midcingulate cortex, and primary sensory cortex (Lamm et al., 2011). Beyond similar neural representations, when typical individuals' pain sensitivity is pharmacologically or non-pharmacologically manipulated, their pain ratings and brain responses to others' pain also change accordingly (Mischkowski et al., 2016; Rütgen, Seidel, Riečanský & Lamm, 2015; Rütgen, Seidel, Silani et al., 2015). For example, participants who took acetaminophen (an analgesic) not only showed reduced pain sensitivity themselves but also became less sensitive to others' suffering, suggesting that the analgesic suppressed perception of others' distress while inhibiting physical pain, thereby reducing empathic experience (Mischkowski et al., 2016). Patients

with congenital insensitivity to pain, due to physiological reasons (lack of pain receptors), have never experienced pain themselves and consequently find it difficult to empathize with others' pain (Danziger et al., 2009; Danziger et al., 2006). These findings support the Shared-representation Model of Empathy, which posits that individuals' empathy for others involves similar psychological and neural representations as self-perception (Decety & Jackson, 2004; Jackson et al., 2005). Given the connection between first-hand pain and pain empathy, pain empathy deficits in ASD individuals may originate from abnormalities in first-hand pain experience.

Only two studies have examined the relationship between first-hand pain and pain empathy in ASD individuals (Chen et al., 2017; Fan et al., 2014). Fan et al. (2014) found that ASD individuals showed heightened sensitivity to their own pain (lower pressure pain threshold); when viewing images of others in painful situations, ASD individuals exhibited stronger activation in primary/secondary somatosensory cortices but weaker activation in brain regions associated with emotional empathy such as the anterior midcingulate cortex and anterior insula, along with lower unpleasantness ratings for others' pain. First-hand pain sensitivity mediated the relationship between pain empathy-related primary/secondary somatosensory cortex responses and unpleasantness ratings. The researchers suggested that due to heightened first-hand pain sensitivity in ASD individuals, sensorimotor resonance responses in somatosensory cortices were enhanced during passive viewing of others' pain, leading ASD individuals to adopt attentional avoidance strategies to prevent excessive emotional distress, which manifested as weaker activation in emotional empathy brain regions and lower unpleasantness ratings. Chen et al. (2017) similarly found that the ASD group had lower pressure pain thresholds and lower unpleasantness ratings for others' pain compared with developmental disorder and control groups, with pressure pain threshold significantly positively correlated with unpleasantness ratings for others' pain, further supporting the view that pain empathy abnormalities in ASD may stem from first-hand pain perception abnormalities. These two studies assessed the sensory dimension of pain through pressure pain threshold and established connections between sensory dimension abnormalities and pain empathy abnormalities in ASD populations. However, pain is a multidimensional subjective experience that includes not only sensory aspects (e.g., discrimination of nociceptive stimulus properties, location, and intensity information) but also emotional (e.g., pain-related fear and anxiety) and cognitive (e.g., pain-related catastrophizing and anticipation) dimensions (Tracey, 2011; Wiech et al., 2008). The relationship between pain-related emotional and cognitive dimensions and pain empathy in ASD individuals remains to be investigated.

The cognitive and behavioral characteristics of ASD individuals limit methodological choices in research. For instance, prevalent verbal and cognitive impairments make related research work difficult, resulting in small sample sizes, low reproducibility, and high heterogeneity in ASD studies. Compared with ASD individuals, individuals with high autistic traits better tolerate structured testing

environments and can complete more complex experimental tasks. Therefore, the present study used individuals with high autistic traits as participants to examine the influence of autistic traits on empathy from both state and trait levels. State empathy refers to an immediate and temporary empathic state evoked by a current relevant situation, emphasizing the empathic state generated during temporary interactions between the empathizer and the target (Davis, 1980). Trait empathy is a relatively stable personality trait referring to an individual's general capacity to empathize with others' emotions and pain (Decety & Moriguchi, 2007). Experiment 1 used individuals with high and low autistic traits as participants, employing a pseudo-dyadic paradigm to characterize behavioral responses and event-related potential (ERP) responses during first-hand pain experience and witnessing others in pain, examining how autistic traits influence state empathic responses through first-hand pain. Experiment 2 randomly recruited healthy adult participants and used questionnaire surveys to examine the relationship among autistic traits, pain-related traits, and trait empathy in the general population, testing the mediating role of pain-related traits in the association between autistic traits and trait empathy.

Previous ERP studies on pain empathy have indicated that frontoparietal N1, P2, and N2 components primarily reflect early processing stages related to bottom-up emotional sharing, whereas parieto-occipital P3 and LPP components mainly reflect late processing stages related to top-down cognitive evaluation (Cheng et al., 2014; Decety, 2011; Fan & Han, 2008; Sessa et al., 2014). Fan et al. (2014) found that when participants were asked to judge whether picture materials depicted pain or non-pain, the ASD group showed significant differences in N2 responses to pain and non-pain picture stimuli, whereas control groups did not show this difference, possibly indicating enhanced neural responses in ASD individuals during early automatic processing stages of pain empathy (e.g., perception of others' pain, emotional sharing). Given the similarities between high autistic traits and ASD populations, we proposed Hypothesis 1: Compared with individuals with low autistic traits, individuals with high autistic traits would show stronger trait and state emotional empathy, manifested as higher scores on emotional empathy dimensions of trait empathy scales and stronger early ERP responses (e.g., N1 and P2 components) when witnessing others in pain; they would show weaker trait and state cognitive empathy, manifested as lower scores on cognitive empathy dimensions of trait empathy scales and weaker late ERP responses (e.g., P3 component) when witnessing others in pain. According to the shared representation theory of empathy, individuals' empathic responses to others' pain partially depend on their own pain-related psychological and neural representations. Therefore, we further proposed Hypothesis 2: The influence of autistic traits on pain empathy may originate from changes in first-hand pain, meaning that first-hand pain traits or states could explain pain empathy performance in individuals with high autistic traits.

Experiment 1: Differences in Empathy for Others' Pain Between Individuals with High and Low Autistic Traits

Participants

Based on previous research on autistic traits (Dunn et al., 2016; Li et al., 2020; Meng et al., 2017; Meng et al., 2019; Peled-Avron & Shamay-Tsoory, 2017), we first administered the Chinese version of the Autism-Spectrum Quotient (AQ) (Liu, 2008) to 1,131 university students to assess autistic traits. According to the distribution of AQ total scores ($M \pm SE = 20.08 \pm 0.17$), participants were divided into low autistic trait (LAQ; $AQ \leq 13$) and high autistic trait (HAQ; $AQ \geq 27$) groups based on the bottom and top 10% of scores, respectively. A subset of participants was then randomly recruited for subsequent experiments. Sample size was calculated using G*Power software (Faul et al., 2007). With a medium effect size ($f = 0.25$), desired power ($1 - \beta = 0.80$), and significance level ($\alpha = 0.05$), the required total sample size was no fewer than 24 participants. Ultimately, 30 participants (16 males; age: 20.77 ± 0.34 years) were included in the LAQ group and 30 participants (15 males; age: 21.30 ± 0.31 years) in the HAQ group. All participants were right-handed with normal or corrected-to-normal vision, had no acute or chronic pain conditions or current medication use, and no psychiatric history. All participants were informed about the experimental procedures and provided informed consent. The experimental protocol was approved by the Shenzhen University Ethics Committee (PN-2021-022).

Measures

Upon arrival at the laboratory, participants completed several questionnaires. The AQ scale assessed autistic traits, pain trait scales (Pain Sensitivity Questionnaire, Pain Fear Questionnaire, and Pain Catastrophizing Scale) evaluated pain-related perception, emotion, and cognition, and the Interpersonal Reactivity Index assessed trait empathy.

The AQ scale contains 50 items covering major autistic symptoms and behavioral patterns across five dimensions: attention switching, social skills, communication, attention to detail, and imagination. Items are scored on a 0–1 scale, with "definitely agree" or "slightly agree" scored as 1 for positively worded items and "slightly disagree" or "definitely disagree" scored as 1 for reverse-scored items. Total AQ scores range from 0 to 50, with higher scores indicating higher autistic traits. In this study, the scale's Cronbach's α coefficient was 0.91.

The Pain Sensitivity Questionnaire (PSQ; Quan et al., 2018; Ruscheweyh et al., 2009) contains 17 items covering common pain types in daily life, with two dimensions: mild pain and moderate pain. Items are rated on a 0–10 scale, where 0 represents "no pain" and 10 represents "extremely painful." Higher total PSQ scores indicate higher pain sensitivity. In this study, the scale's Cronbach's α coefficient was 0.90.

The Fear of Pain Questionnaire (FPQ; McNeil & Rainwater, 1998; Yang et al.,

2013) contains 30 items assessing individual levels of pain-related fear across three dimensions: minor pain, severe pain, and medical pain. Items are rated on a 0–4 scale, where 0 represents "not at all fearful" and 4 represents "extremely fearful." Higher total FPQ scores indicate higher pain fear levels. In this study, the scale's Cronbach's α coefficient was 0.90.

The Pain Catastrophizing Scale (PCS; Sullivan et al., 1995; Yap et al., 2008) contains 13 items assessing individuals' emotional and cognitive attitudes toward pain across three dimensions: rumination, magnification, and helplessness. Items are rated on a 0–4 scale, where 0 represents "never" and 4 represents "always." Higher total PCS scores indicate higher pain catastrophizing levels. In this study, the scale's Cronbach's α coefficient was 0.92.

The Interpersonal Reactivity Index (IRI; Davis, 1983; Siu & Shek, 2005) contains 22 items assessing individual trait empathy capacity from cognitive and emotional perspectives across four dimensions: Perspective Taking (PT), Personal Distress (PD), Empathic Concern (EC), and Fantasy (FS). Items are rated on a 0–4 scale, where 0 represents "does not describe me well" and 4 represents "describes me very well." The Perspective Taking and Fantasy dimensions are considered measures of cognitive empathy, while Personal Distress and Empathic Concern are considered measures of emotional empathy. In this study, the scale's Cronbach's α coefficient was 0.71.

Pain Stimulus Intensity Calibration

Painful stimuli were delivered using a constant voltage electrical stimulator (DS7A, Hertfordshire, UK). Before the formal experiment, participants underwent two rounds of stimulus-response curve measurements to determine the pain stimulus intensity used in the experiment. A pair of ring electrodes was placed on the participant's left ring finger, through which a series of electrical current stimuli were delivered. Each stimulus consisted of several rapid successive constant-current square wave pulses. The initial current intensity was 0.5 mA, with step increments of 0.5 mA, inter-stimulus intervals of 4000–6000 ms, and pulse width of 0.5 ms. After each stimulus, participants rated the experienced pain intensity on a scale of 0–10, where 0 represented "no pain" and 10 represented "extremely painful." The interval between the two measurement rounds was approximately 3 minutes. Based on the average stimulus-response curve, the stimulus intensity that could evoke a pain rating of 6 was determined (Yao et al., 2021; Zhou et al., 2019) and used for painful stimulation in the formal experiment.

Experimental Design

The experiment employed a 2 (Group: LAQ, HAQ) $\times$ 2 (Pain Target: self, other) $\times$ 2 (Certainty: certain, uncertain) mixed factorial design, with group as a between-subjects factor and pain target and certainty as within-subjects factors.

Experimental Task

Upon arrival at the laboratory, participants were informed that they would participate in a dyadic experiment with a partner (actually a confederate played by a fixed female experimenter) (Cui et al., 2015; Peng et al., 2019; Rütgen, Seidel, Riečanský & Lamm, 2015). As shown in [Figure 1: see original paper]A, after completing pain calibration, participants sat at two adjacent tables separated by a screen in the same room as the confederate partner and began the formal experimental task.

The experiment consisted of 4 blocks, each containing 40 trials. Five practice trials preceded the formal experiment to familiarize participants with the procedure. Throughout the experiment, participants wore a pair of ring electrodes on their left ring finger. During the instruction phase, participants were informed that both parties would receive electrical stimulation 50% of the time during the entire experiment. The specific experimental procedure is illustrated in [Figure 1: see original paper]B: First, a fixation cross "+" appeared at the center of the screen, followed by an arrow-shaped cue. The arrow direction indicated the upcoming pain target (left arrow: self receives pain; right arrow: other receives pain; up arrow: random person receives pain). The proportions of the three arrow types were 25%, 25%, and 50%, respectively. The painful stimulus was delivered 4 ± 1 s after the cue disappeared. Following the stimulus, participants were required to rate the unpleasantness they experienced using a key press (range: 0–10, where 0 represented "not at all unpleasant" and 10 represented "extremely unpleasant"). After each block, participants rated their fear level when seeing the three different types of arrows (range: 0–10, where 0 represented "no fear at all" and 10 represented "extremely fearful"). E-prime 3.0 recorded participants' behavioral data, and participants' responses were recorded throughout the experiment.

EEG Data Acquisition and Analysis

EEG data were recorded using an ERP recording system from Brain Products GmbH, with a 64-channel electrode cap based on the extended international 10-20 system. The reference electrode was located at FCz, and the ground electrode at AFz. The bandpass filter was 0.01–100 Hz, sampling frequency was 1000 Hz, and impedance between electrodes and scalp was maintained below 10 kΩ.

EEG data preprocessing and analysis were conducted using MATLAB R2016b and the EEGLAB 14.1.2 toolbox (Delorme & Makeig, 2004). For offline analysis, the reference was converted to the average of bilateral mastoids, and the bandpass filter was set to 0.2–30 Hz. The analysis epoch was segmented from 500 ms before to 1000 ms after stimulus onset, with the 500 ms pre-stimulus interval serving as baseline. Trials with severe artifact interference were manually excluded, and ocular artifacts were corrected using independent component analysis.

When participants received painful stimuli themselves, the pain-evoked N1 and

P2 components were analyzed. When witnessing others receiving painful stimuli, the pain empathy-evoked P2 and P3 components were analyzed. Based on waveform plots, topographic maps, and relevant literature, electrode sites and time windows were selected for calculating mean amplitudes of ERP components (Liao et al., 2018; Peng et al., 2019). For self-pain conditions, the N1 component was measured at electrodes C1, Cz, C2, and FCz within the 110–140 ms time window; the P2 component was measured at electrodes C1, Cz, C2, and CPz within the 270–300 ms time window. For other-pain conditions, the P2 component was measured at electrodes C1, Cz, C2, and FCz within the 220–250 ms time window; the P3 component was measured at electrodes CP1, CPz, CP2, and Pz within the 400–550 ms time window.

Statistical Analysis

Statistical analyses were performed using SPSS 22.0 and the PROCESS 3.2 plugin (Hayes, 2012). Independent samples t-tests or chi-square tests were used to compare differences between LAQ and HAQ groups in gender, age, stimulus intensity used, and questionnaire scores, with False Discovery Rate (FDR) correction applied for multiple comparisons. Behavioral data from the pain empathy task included pain fear level ratings during the anticipation phase and unpleasantness ratings during the pain phase. Pain fear level ratings were analyzed using a 2 (Group: LAQ, HAQ) × 3 (Cue Type: self, other, uncertain) repeated measures ANOVA. Unpleasantness ratings were analyzed using a 2 (Group: LAQ, HAQ) × 2 (Pain Target: self, other) × 2 (Certainty: certain, uncertain) repeated measures ANOVA. Mean amplitudes of ERP components evoked by self-pain and other-pain were analyzed using separate 2 (Group: LAQ, HAQ) × 2 (Certainty: certain, uncertain) repeated measures ANOVAs. When the sphericity assumption was violated in repeated measures ANOVAs, the Greenhouse-Geisser correction was applied. Post-hoc pairwise comparisons were Bonferroni-corrected. Mediation analyses used bias-corrected bootstrap methods with 5000 bootstrap samples. If the 95% confidence interval did not contain 0, the parameter estimate was considered significant; otherwise, it was not significant (Preacher & Hayes, 2008).

Results

Demographic Variables and Questionnaire Scores As shown in , the LAQ and HAQ groups did not differ significantly in gender or age. The electrical stimulus intensity used to evoke pain did not differ between groups. No significant group differences were found on the Pain Sensitivity Questionnaire or Fear of Pain Questionnaire. However, the HAQ group scored significantly higher on the Pain Catastrophizing Scale than the LAQ group ($t(58) = -3.39$, $p < 0.001$), indicating that the HAQ group had more negative emotions and cognitions toward pain. Regarding Interpersonal Reactivity Index scores, the HAQ group scored marginally significantly lower on the Perspective Taking subscale than the LAQ group ($t(58) = 2.35$, $p = 0.060$), suggesting weaker cognitive

empathy in the HAQ group. The HAQ group scored significantly higher on the Personal Distress subscale than the LAQ group ($t(58) = -5.33$, $p < 0.001$), indicating stronger emotional empathy in the HAQ group. No significant group differences were found on the Empathic Concern or Fantasy subscales.

Behavioral Data Pain fear ratings during the anticipation phase are shown in [Figure 2: see original paper]A. The main effect of cue type was significant, $F(2, 57) = 30.43$, $p < 0.001$, $^2p = 0.52$. Post-hoc tests revealed that fear levels for uncertain pain cues were significantly greater than for self-pain cues (3.09 ± 0.31 vs. 2.70 ± 0.29, $p < 0.001$), and fear levels for self-pain cues were significantly higher than for other-pain cues (2.70 ± 0.29 vs. 1.34 ± 0.18, $p < 0.001$). The main effect of group was significant ($F(1, 58) = 63.90$, $p = 0.014$, $^2p = 0.10$), with the HAQ group showing significantly higher fear levels for pain cues than the LAQ group (2.97 ± 0.33 vs. 1.78 ± 0.33). The interaction between cue type and group was not significant.

Unpleasantness ratings during the pain experience phase are shown in [Figure 2: see original paper]B. The main effect of pain target was significant ($F(1, 58) = 41.68$, $p < 0.001$, $^2p = 0.42$), with self-pain unpleasantness ratings significantly greater than other-pain ratings (3.89 ± 0.27 vs. 2.08 ± 0.22). The main effect of group was significant ($F(1, 58) = 7.28$, $p = 0.009$, $^2p = 0.11$), with the HAQ group showing significantly higher unpleasantness ratings than the LAQ group (3.53 ± 0.29 vs. 2.44 ± 0.29). The interaction between pain target and certainty was significant, $F(1, 58) = 15.18$, $p < 0.001$, $^2p = 0.21$. Simple effects analysis revealed that when witnessing others' pain, unpleasantness ratings in uncertain contexts were significantly higher than in certain contexts (2.16 ± 0.25 vs. 1.99 ± 0.23, $p = 0.001$); when receiving pain themselves, there was no significant difference in unpleasantness ratings between uncertain and certain contexts (3.86 ± 0.28 vs. 3.92 ± 0.27, $p = 0.157$). The interaction between pain target and group was marginally significant, $F(1, 58) = 3.06$, $p = 0.086$, $^2p = 0.05$. Simple effects analysis showed that when facing others' pain, the HAQ group's unpleasantness ratings were significantly higher than the LAQ group's (2.81 ± 0.35 vs. 1.34 ± 0.27, $p = 0.002$), whereas when facing self-pain, there was no significant difference between groups (4.25 ± 0.43 vs. 3.53 ± 0.33, $p = 0.184$). No other main effects or interactions were significant.

The HAQ group showed significantly higher pain fear than the LAQ group, and significantly higher unpleasantness ratings when witnessing others' pain. Mediation analysis was used to explore the relationship among autistic traits, pain fear evoked by cues during the anticipation phase, and unpleasantness when witnessing others' pain. As shown in [Figure 3: see original paper], pain fear during the anticipation phase mediated the difference in unpleasantness ratings when witnessing others' pain between the LAQ and HAQ groups. In this mediation model, autistic traits (LAQ vs. HAQ groups) served as the predictor, cue-evoked pain fear as the mediator (averaged across self-pain, other-pain, and uncertain conditions), and unpleasantness ratings for others' pain as the out-

come (averaged across certain and uncertain conditions). Bootstrap mediation effect testing revealed a significant total effect (c = 0.40, SE = 0.12, 95%CI = [0.16, 0.64]), a significant direct effect (c = 0.29, SE = 0.12, 95%CI = [0.05, 0.53]), and a significant indirect effect (a×b = 0.11, SE = 0.06, 95%CI = [0.01, 0.25]). This indicates that cue-evoked pain fear partially mediated the relationship between autistic traits and emotional empathy for pain.

Electrophysiological Data ERP waveforms and topographic maps evoked by self-pain and witnessing others' pain are shown in [Figure 4: see original paper]. For self-pain conditions, two-way ANOVAs were conducted on mean amplitudes of pain-evoked N1 and P2 components. For the N1 component, neither main effects nor interactions were significant (all $p > 0.05$). For the P2 component, the main effect of certainty was significant ($F(1, 58) = 5.19$, $p = 0.026$, $^2p = 0.08$), with P2 amplitudes in uncertain contexts significantly greater than in certain contexts (20.37 ± 1.22 V vs. 19.27 ± 1.16 V, [Figure 4: see original paper]B). Neither the main effect of group nor the Group × Certainty interaction was significant (all $p > 0.05$).

For witnessing others' pain conditions, two-way ANOVAs were conducted on mean amplitudes of pain empathy-evoked P2 and P3 components. For the P2 component, the main effect of group was significant ($F(1, 58) = 4.39$, $p = 0.041$, $^2p = 0.07$), with the HAQ group showing significantly greater P2 amplitudes than the LAQ group when witnessing others' pain (5.68 ± 0.74 V vs. 3.50 ± 0.74 V, [Figure 4: see original paper]D). The main effect of certainty was marginally significant ($F(1, 58) = 3.85$, $p = 0.054$, $^2p = 0.06$), with P2 amplitudes in uncertain contexts greater than in certain contexts (4.92 ± 0.58 V vs. 4.25 ± 0.58 V). For the P3 component, the main effect of certainty was significant ($F(1, 58) = 103.45$, $p < 0.001$, $^2p = 0.64$), with P3 amplitudes in uncertain contexts significantly greater than in certain contexts (10.71 ± 0.80 V vs. 5.62 ± 0.61 V, [Figure 4: see original paper]D). Neither the main effect of group nor the Group × Certainty interaction was significant.

Correlation Analysis When witnessing others' pain, the HAQ group showed significantly greater P2 amplitudes and higher unpleasantness ratings than the LAQ group. Spearman correlation analysis was further used to explore the relationship between P2 responses and unpleasantness ratings when witnessing others' pain. As shown in [Figure 5: see original paper], for the HAQ group, P2 amplitudes when witnessing others' pain were significantly positively correlated with unpleasantness ratings, $r(28) = 0.43$, $p = 0.017$. However, for the LAQ group, the correlation between P2 amplitudes and unpleasantness ratings when witnessing others' pain was not significant, $r(28) = -0.23$, $p = 0.229$.

Experiment 2: Associations Among Autistic Traits, Pain, and Trait Empathy

Participants

Sample size was calculated using G*Power software. Referencing previous studies examining correlations between autistic traits and empathy (Zhang et al., 2022; Zhao et al., 2019), Experiment 2 set the absolute correlation coefficient at 0.20, with desired power ($1 - \beta = 0.80$) and significance level ($\alpha = 0.05$), yielding a required sample size of no fewer than 193 participants. Ultimately, 381 healthy university students (202 males; age: 20.95 ± 0.12 years) were recruited to complete questionnaires. All participants had no acute or chronic pain conditions or current medication use, and no psychiatric history.

Data Collection and Analysis

Participants completed a series of questionnaires at the laboratory, including the AQ scale, pain trait scales (Pain Sensitivity Questionnaire, Fear of Pain Questionnaire, and Pain Catastrophizing Scale), and the Interpersonal Reactivity Index.

SPSS 22.0 and AMOS 24.0 were used for descriptive statistics, correlation analysis, and structural equation modeling. Pearson correlation analysis examined relationships among autistic traits, pain traits, and trait empathy, with p-values FDR-corrected for multiple comparisons. In structural equation modeling, model fit indices included ^{2}/df, GFI, AGFI, CFI, NFI, and RMSEA: (1) ^{2}/df should be less than 2; (2) GFI and AGFI should exceed 0.90; (3) CFI and NFI should exceed 0.95; (4) RMSEA should be less than 0.06 (Hu & Bentler, 1999; Kline, 2016). Bias-corrected bootstrap methods with 5000 bootstrap samples were used to test mediation effects. If the 95% confidence interval did not contain 0, the parameter estimate was considered significant; otherwise, it was not significant (Preacher & Hayes, 2008).

Results

Common Method Bias Test All variables in this experiment were collected using self-report methods, which may introduce common method bias. Harman's single-factor test was used for assessment, conducting an unrotated principal component factor analysis on all items. Results showed that the first factor explained 11.03% of variance, below the 40% critical value, indicating no serious common method bias in this experiment.

Descriptive Statistics and Correlation Analysis presents means, standard errors, and correlation coefficients for all variables. Pearson correlation analysis revealed that regarding the relationship between autistic traits and pain traits, autistic traits were not correlated with pain sensitivity or pain fear; however, autistic traits were significantly positively correlated with pain catastro-

phizing ($r(379) = 0.19$, $p < 0.001$), indicating that individuals with higher autistic traits had stronger negative emotions and cognitions toward pain. Regarding the relationship between autistic traits and trait empathy, autistic traits were significantly negatively correlated with Perspective Taking ($r(379) = -0.19$, $p < 0.001$) and significantly positively correlated with Personal Distress ($r(379) = 0.27$, $p < 0.001$); autistic traits were not correlated with Empathic Concern or Fantasy dimensions. This suggests that individuals with high autistic traits show weaker perspective-taking abilities and stronger personal distress when empathizing. Furthermore, regarding the relationship between pain traits and trait empathy, pain-related cognition was significantly positively correlated with trait emotional empathy, such that higher pain catastrophizing levels were associated with higher Personal Distress scores ($r(379) = 0.47$, $p < 0.001$); pain-related emotion was also significantly positively correlated with trait emotional empathy, such that higher pain fear levels were associated with higher Personal Distress scores ($r(379) = 0.25$, $p < 0.001$).

Mediation Analysis Based on correlation analysis, we further tested whether pain-related negative emotion and cognition mediated the link between autistic traits and personal distress. With autistic traits as the predictor, personal distress as the outcome, and pain-related negative emotion and cognition as mediators (with pain fear and pain catastrophizing as indicator factors), the structural equation model is shown in [Figure 6: see original paper]. All factor loadings exceeded 0.4. Model fit indices were as follows: $^2/df = 1.604$ ($p = 0.205$), GFI = 0.998, AGFI = 0.979, CFI = 0.997, NFI = 0.992, RMSEA = 0.040, indicating good model fit. Results showed a significant total effect ($c = 0.27$, SE = 0.05, 95%CI = [0.17, 0.36], $p < 0.001$), a significant direct effect ($c = 0.16$, SE = 0.05, 95%CI = [0.07, 0.25], $p = 0.002$), and a significant indirect effect ($a \times b = 0.11$, SE = 0.04, 95%CI = [0.05, 0.19], $p < 0.001$). This indicates that pain-related negative emotion and cognition partially mediated the effect of autistic traits on emotional empathy.

General Discussion

The present study combined a pseudo-dyadic pain empathy paradigm and questionnaire survey to examine the associations among autistic traits, first-hand pain, and empathy. In the pseudo-dyadic pain empathy paradigm, individuals with high autistic traits showed stronger P2 responses and higher unpleasantness ratings when witnessing others receiving painful stimulation, which were partially attributable to high levels of pain-related fear. Questionnaire survey results revealed that higher levels of autistic traits were associated with lower perspective-taking scores and higher personal distress scores on trait empathy scales, with pain fear and catastrophizing mediating the link between autistic traits and personal distress. Therefore, findings from both state and trait empathy studies support that high autistic traits are associated with heightened emotional empathy, partially originating from more negative emotions and cognitions toward pain. This supports understanding empathy and other social

functioning deficits in high autistic traits and ASD populations from the perspective of first-hand pain abnormalities, providing potential targets for clinical treatment and intervention.

Experiment 1 adopted an "autism-simulating" approach (Robinson et al., 2011; Guan & Zhao, 2015), screening individuals with high and low autistic traits from the general population and combining behavioral and ERP techniques to explore differences in state empathy between groups. In terms of experimental paradigm, this study presented cue stimuli to inform participants whether they would experience pain themselves or witness their partner (a confederate) receiving painful stimulation, and required participants to rate their emotional responses during pain anticipation and experience phases. This allowed characterization of pain empathy processing features in a relatively natural context. When required to rate upcoming painful stimuli, the HAQ group reported higher pain fear regardless of pain target, indicating more negative emotional responses toward pain in the HAQ group. When experiencing pain themselves, the HAQ and LAQ groups showed no significant differences in pain-evoked responses, including the N1 component related to sensory discrimination and the P2 component related to emotional motivation, nor in behavioral unpleasantness ratings. This was primarily because the pain stimulus intensity delivered to both groups was individually calibrated, meaning that the experimental pain stimuli could evoke moderate pain experiences (rating of 6/10) for all participants. When witnessing others' pain, the HAQ group showed significantly stronger pain empathy-evoked P2 responses than the LAQ group in both certain and uncertain contexts, along with higher unpleasantness ratings. The pain empathy-evoked P3 response did not differ significantly between HAQ and LAQ groups, suggesting that autistic traits do not significantly affect late-stage cognitive evaluation processes of pain empathy. Similarly, Fan et al. (2014) used bodily pain images to evoke empathic responses and found that when participants were required to judge whether images depicted pain or non-pain, no significant differences were observed between ASD and control groups in late ERP components (e.g., LPP), suggesting that ASD individuals show similar neural responses to typical individuals during late cognitive evaluation stages of pain empathy. However, meta-analytic results indicate that cognitive empathy in ASD individuals is significantly weaker than in typical individuals (Song et al., 2019), with cognitive empathy primarily assessed through tasks such as the Reading the Mind in the Eyes Test (Baron-Cohen et al., 2015; Baron-Cohen, Wheelwright, Hill et al., 2001; Murray et al., 2017; Ponnet et al., 2004), the Multifaceted Empathy Test (Dziobek et al., 2008; Mul et al., 2018), and emotion discrimination tasks (Eyuboglu et al., 2018; Zuluaga Valencia et al., 2018). These discrepant findings may result from different empathy assessment paradigms used across studies. The P2 component evoked by witnessing others' pain is associated with early, bottom-up emotional arousal and sharing processes, whereas the P3 component is more related to late, top-down cognitive evaluation processes (Cheng et al., 2014; Decety, 2011; Fan & Han, 2008; Sessa et al., 2014). The P2 component is thought to originate primarily from the midcingulate cortex

(Mobascher et al., 2009; Perchet et al., 2008), a brain region that mainly encodes the affective dimension of pain (Baumgärtner et al., 2006). Therefore, when facing others' pain, participants with high autistic traits showed stronger emotional arousal and experienced more personal distress, indicating stronger emotional empathy responses. Correlation analysis revealed that for the HAQ group, larger pain empathy-evoked P2 responses were associated with higher personal unpleasantness ratings, supporting the notion that P2 responses can reflect emotion-related empathic arousal. Given that the HAQ group reported stronger fear of impending pain and higher personal unpleasantness when witnessing others' pain than the LAQ group, further mediation analysis revealed that autistic traits enhanced pain fear levels, which in turn increased personal distress in pain empathy. This provides evidence for the link among autistic traits, pain-related negative emotion, and emotional empathy, highlighting the role of pain-related negative emotion in how autistic traits influence emotional empathy.

Previous research has found that ASD individuals have low tolerance for uncertainty (Chamberlain et al., 2013; Neil et al., 2016). For example, Vasa et al. (2018) compared 57 ASD children aged 7–16 years with 32 typically developing children and found that ASD children showed significantly lower tolerance for uncertainty in both parent-report and self-report measures. Therefore, this study introduced certainty as a variable in the experimental design. Results showed that certainty affected unpleasantness ratings, P2, and P3 responses when witnessing others' pain (significant main effect of certainty), such that uncertain contexts evoked higher unpleasantness ratings and stronger P2 and P3 responses. However, the effect of autistic traits on empathy was not moderated by certainty (no significant Certainty × Group interaction). No previous studies have examined whether pain and empathy abnormalities in ASD populations are related to certainty. Future research should introduce certainty as a variable in studies of pain and empathy in ASD populations to further validate the present findings.

Experiment 2 used questionnaire surveys to examine the relationship among autistic traits, pain, and trait empathy in a randomly recruited general population sample. In the trait empathy scale, the Perspective Taking dimension assesses individuals' tendency to understand and simultaneously adopt others' psychological perspectives in real-life situations and is considered to reflect cognitive empathy; the Personal Distress dimension assesses individuals' self-centered reactions when facing others' distress or stressful situations and is considered to reflect emotional empathy (Davis, 1983). Results showed that high autistic traits were associated with lower perspective-taking abilities and stronger personal distress when facing others' suffering. Thus, in the general population, high autistic traits are associated with weaker cognitive empathy and stronger emotional empathy, further supporting the link between autistic traits and state emotional empathy found in Experiment 1 (i.e., HAQ individuals showed stronger personal distress and emotional arousal when witnessing others' pain). Regarding pain trait scales, pain-related fear and pain catastrophizing were sig-

nificantly positively correlated with the Personal Distress dimension of trait empathy, and autistic traits were positively correlated with pain catastrophizing. Given the connections among autistic traits, pain-related negative emotion and cognition, and trait empathy, structural equation modeling revealed the mediating role of pain-related negative emotion and cognition in the relationship between autistic traits and trait emotional empathy, further validating findings from Experiment 1. Regarding cognitive empathy, the present study showed different patterns for trait and state cognitive empathy in individuals with high autistic traits: at the trait level, autistic traits were negatively correlated with cognitive empathy, indicating weaker cognitive empathy abilities; at the state level, no significant differences were found between high and low autistic trait individuals in P3 components evoked when witnessing others' pain, suggesting that autistic traits do not significantly affect state cognitive empathy. Similarly, some studies have reported dissociations between trait and state empathy, such as ERP responses evoked by observing others' pain being unrelated to trait empathy scale scores (Galang et al., 2020; Li & Han, 2010), and bilateral insula activation when witnessing others' pain being unrelated to trait empathy scores (Jackson et al., 2005). Future research should employ other empathy assessment paradigms, such as the Reading the Mind in the Eyes Test and the Multifaceted Empathy Test, to further validate the present findings.

Regarding first-hand pain, individuals with high autistic traits scored higher on the Pain Catastrophizing Scale and reported higher fear levels for pain cues during the task, indicating more negative emotions and cognitions toward pain. However, autistic traits were not significantly associated with the sensory dimension of pain. This suggests that the relationship between autistic traits and pain may be dimension-specific, with high autistic trait individuals showing heightened sensitivity only in affective and cognitive dimensions. Similarly, meta-analytic findings indicate that ASD individuals do not differ significantly from typical individuals in pain threshold overall, but show heightened sensitivity to pressure pain thresholds (more sensitive) and stronger physiological responses to real medical pain (Zhang et al., 2021). Pain is a multidimensional subjective experience comprising sensory, affective, and cognitive dimensions (Wiech et al., 2008). Pain threshold reflects the sensory dimension, whereas pain-evoked physiological responses often reflect emotional and cognitive dimensions. Therefore, meta-analytic results support that ASD individuals generally do not differ from typical individuals in the sensory dimension of pain but show heightened sensitivity in emotional and cognitive dimensions. For example, ASD and control groups did not differ significantly in heat pain thresholds, but the ASD group gave significantly higher intensity ratings for suprathreshold heat stimuli and scored significantly higher on pain anxiety and pain fear scales than the control group (Failla et al., 2020), supporting heightened sensitivity in affective and cognitive dimensions of pain in ASD populations. Gu et al. (2018) compared neural responses between ASD and control groups during pain anticipation and processing and found that during pain anticipation, the ASD group showed significantly higher activation in dorsal and rostral anterior cingulate

cortex than the control group, whereas during pain processing, no significant group differences were found in pain-related brain regions (such as anterior cingulate cortex and insula). This suggests that ASD individuals may experience greater anxiety and fear while anticipating painful stimuli. Therefore, regarding first-hand pain, findings from high autistic trait individuals show similarities with those from ASD individuals, with both groups exhibiting more negative emotional and cognitive patterns toward pain. Specifically, when facing actual or potential pain, high autistic trait and ASD individuals may have more negative cognitions about pain and show greater tendencies for anxiety and fear responses. Given the response tendencies of high autistic trait and ASD groups to pain, in real medical settings (e.g., injections or blood draws), medical staff need to be more considerate and patient with these groups, such as by explaining procedures beforehand to alleviate their anxiety and fear.

Findings from both state and trait empathy studies support that pain-related negative emotion and cognition play a mediating role in the association between autistic traits and emotional empathy. Individuals with high autistic traits report stronger personal distress when witnessing others in pain due to excessive negative emotions and cognitions toward pain. Cognitive bias modification for pain-related information can effectively alleviate pain-related negative emotions and cognitions (An et al., 2020; Elomaa et al., 2009; Sharpe et al., 2012; Yang et al., 2016). For example, acute and chronic pain patients who received attention bias modification training (trained to allocate attentional resources to neutral information) showed lower anxiety sensitivity and functional disability than control groups in 6-month follow-up studies (Sharpe et al., 2012); chronic pain patients who received interpretation bias modification training (trained to interpret pain-related information as neutral) showed lower levels of pain-related negative emotion (An et al., 2020). Additionally, cognitive behavioral therapy has been found to effectively reduce fear of movement, pain anxiety, and pain vigilance in chronic pain patients, decreasing pain interference in daily life (Elomaa et al., 2009). Therefore, future research could examine whether cognitive bias modification and cognitive behavioral therapy could effectively improve social skills in individuals with high autistic traits and ASD by reducing negative emotions and cognitions toward pain. The population with high autistic traits is substantial in number, and their mental and physical health and social functioning problems cannot be ignored. Therefore, the present findings can help understand pain and empathy issues in high autistic trait individuals in the general population and facilitate the development of intervention strategies to promote their mental health and social functioning. Additionally, given the similarities between high autistic trait and ASD populations, this study supplements evidence on empathy deficits in ASD populations from a subclinical perspective, suggesting that empathy and social functioning deficits in these groups could be explained from the perspective of first-hand pain abnormalities, providing new insights for improving social functioning in ASD populations.

However, research on ASD populations has primarily supported the view that heightened sensitivity in the sensory dimension of pain can explain emotional

empathy deficits in ASD (Chen et al., 2017; Fan et al., 2014). Fan et al. (2014) and Chen et al. (2017) combined psychophysical pain measurement and picture-evoked empathy paradigms to explore the relationship between first-hand pain and pain empathy in ASD individuals, finding that ASD individuals had lower pressure pain thresholds than typical individuals, which were significantly positively correlated with unpleasantness ratings for others' pain in picture-evoked paradigms. This suggests that heightened sensitivity in the sensory dimension of pain in ASD individuals leads to heightened emotional arousal during pain empathy. Findings from typical individuals show that sensory dimension sensitivity of pain influences emotional empathy through its impact on pain-related emotion and cognition (Ren et al., 2020); individuals with high pain catastrophizing tend to exaggerate their own pain and overestimate others' suffering (Sullivan et al., 2006); Serbic et al. (2020) also found that observers' pain fear levels could positively predict pain empathy when viewing bodily pain images. These findings support the influence of observers' pain-related emotion and cognition on pain empathy. One possible explanation is that high autistic trait and ASD groups show heightened sensitivity to first-hand pain, including sensory, emotional, and cognitive dimensions, leading to more intense emotional arousal and personal distress when witnessing others' pain. Future research should systematically examine pain processing patterns in high autistic trait and ASD populations across multiple levels, including subjective reports, physiological responses, electrophysiology, and neuroimaging, covering sensory, affective, and cognitive dimensions and their neural mechanisms, to establish connections between atypical pain processing patterns and empathy deficits.

The present study has several limitations. First, participants were healthy university students from the same institution with normal intelligence levels, but we did not assess participants' intelligence levels or other basic psychological traits, making it impossible to completely rule out the influence of intelligence level and other factors on the results. Future research should match intelligence levels and other factors between high and low autistic trait groups to further validate the effects of autistic traits on pain and empathy. Second, the confederate (empathy target) was the same female experimenter, which was done to reduce the impact of different confederates on results. However, considering the gender difference in ASD prevalence, the effect of autistic traits on empathy may be moderated by the target's gender. Future research could introduce confederates of different genders to verify whether gender congruence between empathizer and target influences the present findings. Third, in terms of pain stimulus intensity selection, this study only used stimuli that evoked moderate pain intensity, which cannot reveal the impact of stimulus intensity on results. Future research could employ painful stimuli of different intensities and introduce non-painful stimulation conditions as control conditions to examine whether the effect of autistic traits on empathy is pain-specific. Finally, the present study's participants were individuals with high autistic traits, and whether the findings can be generalized to ASD populations remains to be investigated. Given the similarities between autistic traits and ASD in genetics, cognition, and behavioral patterns, this

study's findings can promote understanding of biological mechanisms and intervention strategies for ASD. However, high autistic traits are not equivalent to ASD (Sasson & Bottema-Beutel, 2022). Autistic traits are considered an independent sixth trait beyond the Big Five personality traits (Wakabayashi et al., 2006) and are not specifically related to ASD, as non-ASD populations such as those with anxiety disorders and schizophrenia also often show high autistic traits (Barlati et al., 2019; Lau et al., 2014; Tonge et al., 2016). Future research should further verify the relationships among ASD symptoms, first-hand pain, and pain empathy in ASD populations.

This study combined a pseudo-dyadic pain empathy paradigm and questionnaire survey to explore the connections among autistic traits, pain, and empathy. High autistic traits are associated with more intense emotional empathy, showing stronger personal distress when others are in painful situations, partially due to more negative emotions and cognitions toward pain. However, high autistic traits are associated with weaker cognitive empathy, making it difficult to adopt and understand others' perspectives and thoughts. Given the similarities between high autistic trait and ASD populations, these findings not only promote understanding of biological mechanisms and development of intervention strategies for ASD but also help improve mental health and social functioning in high autistic trait populations. For instance, empathy and social functioning deficits in high autistic trait and ASD populations could be explained from the perspective of first-hand pain abnormalities. Future research should combine multiple pain stimulation modalities and multidimensional pain assessment systems to characterize pain processing features in high autistic trait and ASD individuals and establish specific relationships between pain response patterns and core symptoms such as empathy deficits.

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Note: Figure translations are in progress. See original paper for figures.