The Effect 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 autistic individuals may originate from abnormalities in self-pain experience. Given the similarity between high autistic traits and autism, the present study assessed state and trait empathy through experimental paradigms and questionnaire surveys, aiming to characterize the relationship between autistic traits, self-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 pain, which was partially attributable to high levels of pain-related fear. Questionnaire survey results revealed that higher levels of autistic traits 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-related fear and catastrophizing mediating the relationship between autistic traits and personal distress. Therefore, individuals with high autistic traits demonstrated high levels of state and trait emotional empathy, which was partially derived from more negative emotional and cognitive responses toward pain. This supports understanding empathy and other social functional deficits in individuals with high autistic traits and autistic populations from the perspective of self-pain abnormalities, providing 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 both 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 elevated levels of pain-related fear. Questionnaire data revealed that higher autistic trait levels were associated with lower perspective-taking scores but higher personal distress scores on empathy trait scales, with pain-related fear and catastrophizing mediating the link between autistic traits and personal distress. Thus, individuals with high autistic traits display heightened state and trait emotional empathy, partially originating from more negative emotional and cognitive responses toward pain. These findings support understanding empathy and other social functional impairments in high autistic trait and ASD populations from the perspective of atypical pain experience, providing potential targets for clinical treatment and intervention.

Keywords: autistic traits; pain empathy; pain-related fear; pain catastrophizing; event-related potentials

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1 Introduction

According to the fifth edition of the Diagnostic and Statistical Statistical Manual of Mental Disorders (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, interests, or activities” (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 health and social functioning of individuals with high autistic traits warrant serious attention: compared to those with low autistic traits, individuals with high autistic traits experience greater psychological distress and elevated suicide risk (Dow et al., 2021; Pelton & Cassidy, 2017), exhibit relatively poorer social skills, show reduced motivation for interpersonal interaction, and demonstrate fewer prosocial behaviors (Austin, 2005; Cetinoglu & Aras, 2022; Kloosterman et al., 2011; Zhao et al., 2019). Twin studies indicate that genetic factors account for 36%–87% of the variance in autistic trait levels (Constantino & Todd, 2003; Ronald et al., 2008), and family members of individuals with ASD show significantly higher autistic trait levels than the general population (Pickles et al., 2000; Piven et al., 1997), suggesting shared genetic underpinnings between autistic traits and ASD.

Beyond genetic similarities, individuals with high autistic traits and those with ASD exhibit comparable patterns across emotional, cognitive, and behavioral domains (Gökçen et al., 2014; Kunihira et al., 2006; Takahashi et al., 2013), including heightened anxiety, reduced cognitive flexibility, and impoverished social skills. Given these substantial similarities, research on autistic traits not only advances understanding of the biological mechanisms of ASD and informs intervention development but also helps improve mental health and social functioning in high autistic trait populations.

Empathy refers to the capacity to perceive and understand others’ emotions and thoughts (Singer & Lamm, 2009) and plays a crucial role in interpersonal interactions and prosocial behavior (Decety & Jackson, 2006; Jackson et al., 2005). Empathy comprises two primary components: emotional empathy, which involves perceiving and sharing others’ emotional states, and cognitive empathy, which enables accurate understanding of others’ mental states, 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 the social impairments characteristic of ASD. Previous research has consistently identified cognitive empathy deficits in individuals with ASD (Song et al., 2019; Ziermans et al., 2019), yet findings regarding emotional empathy remain controversial. Some studies report heightened emotional empathy in ASD (Capps et al., 1993; Rogers et al., 2007); for instance, Gu et al. (2015) found that compared to controls, individuals with ASD showed stronger skin conductance responses and greater anterior insula activation when viewing images of bodily pain (e.g., a finger being cut). Conversely, other studies report reduced emotional empathy (Minio-Paluello et al., 2009); for example, Santiesteban et al. (2021) found that after watching videos of others describing painful experiences, individuals with ASD reported lower unpleasantness ratings. Still other studies find no significant differences in emotional empathy between ASD and typical populations (Bird et al., 2010; Blair, 1999); for instance, in multidimensional empathy tasks, individuals with ASD showed comparable arousal ratings for painful scenario images and sympathy ratings for protagonists relative to controls (Dziobek et al., 2008). Thus, although empathy deficits in ASD are widely acknowledged, specific manifestations remain debated, and the nature of emotional empathy abnormalities and their underlying mechanisms require further investigation.

Neuroimaging research demonstrates that both experiencing pain firsthand and observing others in pain activate similar neural representations, including the anterior insula, anterior midcingulate cortex, and primary somatosensory cortex (Lamm et al., 2011). Importantly, when typical individuals' pain sensitivity is pharmacologically or non-pharmacologically manipulated, their pain ratings and neural responses to others' pain stimuli correspondingly increase or decrease (Mischkowski et al., 2016; Rütgen, Seidel, Riečanský et al., 2015; Rütgen, Seidel, Silani et al., 2015). For example, participants who consumed acetaminophen (an analgesic) exhibited reduced pain sensitivity and decreased sensitivity to others' suffering, suggesting that the analgesic suppressed both somatic pain and the perception of others' pain, consequently reducing empathic experience (Mischkowski et al., 2016). Patients with congenital insensitivity to pain, who lack pain receptors and have never experienced pain, also struggle 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 empathizing with others and self-perception involve similar psychological and neural representations (Decety & Jackson, 2004; Jackson et al., 2005). Given this connection between first-hand pain and pain empathy, empathy deficits in ASD may originate from atypical pain experience. However, research on pain abnormalities in ASD has yielded heterogeneous results (Moore, 2015): some studies report pain hyperresponsiveness (Rattaz et al., 2013), while others find pain hyporesponsiveness or normal responses (Fründt et al., 2017; Li et al., 2015). A recent meta-analysis systematically examined pain sensitivity differences between individuals with ASD and typical controls (Zhang et al., 2021), finding that individuals with ASD showed heightened sensitivity to pressure pain and real-world medical pain.

Only two studies have investigated the relationship between first-hand pain and pain empathy in ASD (Chen et al., 2017; Y. T. Fan et al., 2014). Y. T. Fan et al. (2014) found that individuals with ASD exhibited heightened sensitivity to their own pain (lower pressure pain threshold) and, when viewing images of others in painful situations, showed stronger activation in primary/secondary somatosensory cortex but weaker activation in anterior midcingulate cortex and anterior insula—regions associated with emotional empathy—and reported lower unpleasantness ratings for others' pain. First-hand pain sensitivity mediated the relationship between pain empathy-related somatosensory cortex responses and unpleasantness ratings. The researchers proposed that heightened first-hand pain sensitivity in ASD enhances sensorimotor resonance responses in somatosensory cortex during passive observation of others' pain, leading individuals with ASD to employ attentional avoidance to prevent excessive emotional distress, which manifests as weaker activation in emotional empathy brain regions and lower unpleasantness ratings. Chen et al. (2017) similarly found that, compared to developmental disorder and control groups, individuals with ASD had lower pressure pain thresholds and lower unpleasantness ratings for others' pain, with a significant positive correlation between pressure pain threshold and unpleasantness ratings for others' pain, further supporting the notion that pain empathy abnormalities in ASD may stem from atypical first-hand pain perception. These two studies assessed the sensory dimension of pain via pressure pain threshold and established links between sensory pain abnormalities and pain empathy deficits in ASD. However, pain is a multidimensional subjective experience encompassing not only sensory aspects (e.g., discrimination of nociceptive stimulus properties, location, and intensity) 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 emotional and cognitive dimensions of pain and pain empathy in ASD remains unexplored.

The cognitive and behavioral characteristics of individuals with ASD constrain methodological choices; pervasive verbal and cognitive impairments make research challenging, resulting in small sample sizes, low replicability, and high heterogeneity. In contrast, individuals with high autistic traits better tolerate structured testing environments and can complete more complex experimental tasks. Therefore, the present study investigated the influence of autistic traits on empathy at both state and trait levels in a high autistic trait population. State empathy refers to an immediate, transient empathic state evoked by a current situation, emphasizing the temporary interaction between the empathizer and the target (Davis, 1980). Trait empathy represents a relatively stable personality characteristic, reflecting a general capacity to empathize with others' emotions and pain (Decety & Moriguchi, 2007). Experiment 1 employed high and low autistic trait participants in a pseudo-dyadic paradigm to characterize behavioral and event-related potential (ERP) responses during first-hand pain experience and observation of others' pain, examining how autistic traits affect state empathic responses through their impact on first-hand pain. Experiment 2 randomly recruited healthy adult participants to investigate relationships among autistic traits, pain-related traits, and trait empathy in the general population, testing whether pain-related traits mediate the association between autistic traits and empathy traits.

Previous ERP research on pain empathy indicates that frontoparietal N1, P2, and N2 components reflect early processing associated with bottom-up emotional sharing, whereas parieto-occipital P3 and LPP components reflect later processing associated with top-down cognitive evaluation (Cheng et al., 2014; Decety, 2011; Y. Fan & Han, 2008; Sessa et al., 2014). Fan et al. (2014) found that when required to judge whether pictorial materials depicted pain or non-pain, individuals with ASD showed significant differences in N2 responses between pain and non-pain stimuli, whereas controls did not, potentially indicating enhanced neural responses during early automatic processing stages of pain empathy (e.g., perception and emotional sharing of others' pain) in ASD. Given the similarities between high autistic trait and ASD populations, we propose Hypothesis 1: Compared to individuals with low autistic traits, those with high autistic traits will exhibit stronger trait and state emotional empathy, reflected in higher scores on emotional empathy dimensions of trait empathy scales and stronger early ERP responses (e.g., N1 and P2 components) when witnessing others' pain; they will also show weaker trait and state cognitive empathy, reflected in lower scores on cognitive empathy dimensions of trait empathy scales and weaker late ERP responses (e.g., P3 component) when witnessing others' pain. According to the shared-representation theory of empathy, individuals' empathic responses to others' pain partially depend on psychological and neural representations of their own pain. Therefore, we further propose Hypothesis 2: The influence of autistic traits on pain empathy may originate from altered first-hand pain experience, such that trait or state aspects of first-hand pain can account for pain empathy performance in high autistic trait individuals.

2.1.1 Participants

Following previous autistic trait research (Dunn et al., 2016; X. Li et al., 2020; Meng et al., 2017; Meng et al., 2019; Peled Avron & Shamay Tsoory, 2017), we first assessed autistic traits in 1131 university students using the Mandarin version of the Autism-Spectrum Quotient (AQ) (Liu, 2008). Based on the distribution of AQ total scores (M ± SE = 20.08 ± 0.17), we classified participants into low autistic trait (LAQ; AQ $\leq$ 13) and high autistic trait (HAQ; AQ $\geq$ 27) groups using the bottom and top 10% cutoffs, respectively, and randomly recruited subsets for subsequent experiments. Using G*Power software (Faul et al., 2007), we calculated that a minimum total sample size of 24 participants was required for a medium effect size ($p^2 = 0.25$), desired power ($1-\beta = 0.80$), and significance level ($\alpha = 0.05$). Ultimately, 30 participants were enrolled in the LAQ group (16 males; age: 20.77 ± 0.34 years) and 30 in the HAQ group (15 males; age: 21.30 ± 0.31 years). All participants were right-handed, had normal or corrected-to-normal vision, reported no acute or chronic pain conditions or current medication use, and had no psychiatric history. Participants were informed of the experimental procedures and provided written informed consent. The experimental protocol was approved by the Shenzhen University Ethics Committee (PN-2021-022).

2.1.2 Measures

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

The AQ scale comprises 50 items covering core 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" responses to positively worded items scored as 1 and "slightly disagree" or "definitely disagree" scored as 0 (reverse-scored items are reversed). Total scores range from 0 to 50, with higher scores indicating higher autistic traits. In the present study, Cronbach' s α was 0.91.

The Pain Sensitivity Questionnaire (PSQ; Quan et al., 2018; Ruscheweyh et al., 2009) includes 17 items covering common pain experiences 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 scores indicate greater pain sensitivity. Cronbach' s α was 0.90 in this study.

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 scores indicate greater pain-related fear. Cronbach' s α was 0.90 in this study.

The Pain Catastrophizing Scale (PCS; Sullivan et al., 1995; Yap et al., 2008) comprises 13 items evaluating individuals' affective 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 scores indicate greater pain catastrophizing. Cronbach' s α was 0.92 in this study.

The Interpersonal Reactivity Index (IRI; Davis, 1983; Siu & Shek, 2005) includes 22 items assessing trait empathy from both 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 PT and FS dimensions are considered measures of cognitive empathy, whereas PD and EC are considered measures of emotional empathy. Cronbach' s α was 0.71 in this study.

2.1.3 Pain Stimulation 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 experimental pain stimulation intensity. 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 rapidly successive constant-current square wave pulses. The initial current intensity was 0.5 mA, with increments of 0.5 mA, an inter-stimulus interval of 4000–6000 ms, and a pulse width of 0.5 ms. After each stimulus, participants rated the experienced pain intensity on a 0–10 scale, where 0 represented "no pain" and 10 represented "extremely painful." The two measurement rounds were separated by approximately 3 minutes. Based on the average stimulus-response curve, we identified the stimulation intensity that evoked a pain rating of 6 (Yao et al., 2021; Zhou et al., 2019) for use in the formal experiment.

2.1.4 Experimental Design

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

2.1.5 Experimental Task

Upon arrival, participants were informed that they would complete 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 et al., 2015). As shown in Figure 1 [Figure 1: see original paper]A, after pain calibration, participants sat at adjacent tables separated by a screen and began the formal experimental task.

The experiment comprised four 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 they and their partner would receive electrical stimulation on 50% of trials. The trial procedure is illustrated in Figure 1B: a central fixation cross "+" was followed by an arrow cue indicating the upcoming pain target (left arrow: self-pain; right arrow: other-pain; up arrow: random target; proportions: 25%, 25%, 50%). After a 4 ± 1 s interval following cue offset, the painful stimulus was delivered. Participants then rated their experienced unpleasantness on a 0–10 scale (0 = "no unpleasantness," 10 = "extremely unpleasant"). After each block, participants rated their fear level for the three cue types (0 = "no fear," 10 = "extreme fear"). Rest intervals of 3–5 minutes separated blocks. Behavioral data were recorded using E-Prime 3.0, and EEG was recorded continuously throughout the experiment.

Figure 1. Flowchart of the pseudo-dyadic pain empathy paradigm

2.1.6 EEG Data Collection and Analysis

EEG data were recorded using a Brain Products ERP system with a 64-channel electrode cap arranged according to the extended international 10–20 system. The reference electrode was located at FCz, the ground electrode at AFz, the bandpass filter at 0.01-100 Hz, and the sampling rate at 1000 Hz. Impedance between electrodes and scalp was maintained below 10 kΩ.

EEG preprocessing and analysis were conducted using MATLAB R2016b and the EEGLAB 14.1.2 toolbox (Delorme & Makeig, 2004). Offline analysis involved re-referencing to the average of bilateral mastoids and applying a 0.2–30 Hz bandpass filter. Epochs were extracted from 500 ms pre-stimulus to 1000 ms post-stimulus, with the 500 ms pre-stimulus interval serving as baseline. Trials with severe artifacts were manually rejected, and ocular artifacts were corrected using independent component analysis.

For self-pain trials, pain-evoked N1 and P2 components were analyzed; for other-pain trials, pain empathy-evoked P2 and P3 components were analyzed. Based on waveform topographies and relevant literature (Liao et al., 2018; Peng et al., 2019), electrode sites and time windows were selected for calculating mean amplitudes. For self-pain conditions, N1 was measured at electrodes C1, Cz, C2, and FCz within 110–140 ms; P2 was measured at C1, Cz, C2, and CPz within 270–300 ms. For other-pain conditions, P2 was measured at C1, Cz, C2, and FCz within 220–250 ms; P3 was measured at CP1, CPz, CP2, and Pz within 400–550 ms.

2.1.7 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 compared LAQ and HAQ groups on gender, age, stimulation intensity, and questionnaire scores, with false discovery rate (FDR) correction for multiple comparisons. For pain empathy task behavioral data, we conducted a 2 (Group: LAQ, HAQ) × 3 (Cue Type: self, other, uncertain) repeated-measures ANOVA on anticipatory pain fear ratings, and a 2 (Group) × 2 (Pain Target) × 2 (Certainty) repeated-measures ANOVA on unpleasantness ratings. For ERP component mean amplitudes evoked by self-pain and other-pain, separate 2 (Group) × 2 (Certainty) repeated-measures ANOVAs were performed. Greenhouse-Geisser correction was applied when sphericity assumptions were violated. Post-hoc comparisons used Bonferroni correction. Mediation analysis employed bias-corrected bootstrap methods with 5000 iterations; parameters were considered significant if the 95% confidence interval did not contain zero (Preacher & Hayes, 2008).

2.2.1 Demographic Variables and Questionnaire Results

As shown in Table 1 , LAQ and HAQ groups did not differ significantly in gender or age. The electrical stimulation intensities used to evoke pain were comparable between groups. No significant group differences emerged on pain sensitivity or pain fear scales. However, the HAQ group scored significantly higher on the pain catastrophizing scale than the LAQ group ($t_{58} = -3.39$, p_{fdr} < 0.001), indicating more negative emotional and cognitive responses to pain in the HAQ group. On the Interpersonal Reactivity Index, the HAQ group showed marginally lower perspective-taking scores ($t_{58} = 2.35$, p_{fdr} = 0.060), suggesting weaker cognitive empathy, and significantly higher personal distress scores ($t_{58} = -5.33$, p_{fdr} < 0.001), indicating stronger emotional empathy. No significant group differences were observed on empathic concern or fantasy dimensions.

Table 1. Demographic variables and questionnaire results for LAQ and HAQ groups (M ± SE)

Variable	LAQ Group (n = 30)	HAQ Group (n = 30)	t_{58} / ^{2}	p
Gender (Male/Female)	16/14	15/15	-	-
Age	20.77 ± 0.34	21.30 ± 0.31	-1.16	-
AQ Total Score	10.73 ± 0.35	30.53 ± 0.54	-30.81	<0.001
Stimulation Intensity (mA)	4.34 ± 0.37	4.18 ± 0.30	-	-
Pain Sensitivity	5.45 ± 0.23	5.73 ± 0.23	-0.86	-
Pain Fear	80.13 ± 2.46	82.53 ± 2.82	-0.64	-
Pain Catastrophizing	24.83 ± 1.48	32.87 ± 1.85	-3.39	<0.001
IRI-Perspective Taking	13.47 ± 0.48	11.87 ± 0.49	2.35	0.060
IRI-Personal Distress	8.57 ± 0.62	13.67 ± 0.73	-5.33	<0.001
IRI-Empathic Concern	12.77 ± 0.41	13.57 ± 0.40	-1.41	-
IRI-Fantasy	12.57 ± 0.41	12.93 ± 0.58	-0.52	-

Note: LAQ = low autistic traits; HAQ = high autistic traits; n = sample size. Significant effects (p_{fdr} < 0.05) are bolded.

2.2.2 Behavioral Data

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

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

Figure 2. Behavioral results from the pseudo-dyadic pain empathy task

Note: LAQ = low autistic traits; HAQ = high autistic traits; $p < 0.05$, **$p < 0.01$,** *$p < 0.001$.*

The HAQ group reported significantly higher fear of pain cues and significantly higher unpleasantness ratings when witnessing others'pain compared to the LAQ group. Mediation analysis was conducted to explore the relationships among autistic traits, anticipatory pain fear, and unpleasantness evoked by witnessing others' pain. As shown in Figure 3 [Figure 3: see original paper], anticipatory pain fear mediated the difference in unpleasantness ratings between LAQ and HAQ groups when witnessing others' pain. In this model, autistic traits (LAQ vs. HAQ group) served as the predictor, cue-evoked pain fear (averaged across self, other, and uncertain conditions) as the mediator, and unpleasantness ratings for others' pain (averaged across certain and uncertain conditions) as the outcome. Bootstrap mediation analysis revealed a significant total effect ($c = 0.40$, $SE = 0.12$, $CI = [0.16, 0.64]$), significant direct effect ($c' = 0.29$, $SE = 0.12$, $CI = [0.05, 0.53]$), and significant indirect effect ($a*b = 0.11$, $SE = 0.06$, $CI = [0.01, 0.25]$), indicating that anticipatory pain fear partially mediated the relationship between autistic traits and emotional empathy for pain.

Figure 3. The mediating role of pain fear in the influence of autistic traits on emotional empathy

Note: LAQ = low autistic traits; HAQ = high autistic traits. Path coefficients are standardized regression coefficients. $p < 0.05$, **$p < 0.01$.*

2.2.3 Electrophysiological Data

ERP waveforms and topographies for self-pain and other-pain conditions are shown in Figure 4 [Figure 4: see original paper]. For self-pain trials, two-way ANOVAs were conducted on N1 and P2 amplitudes. For N1, neither main effects nor interactions were significant (all $p > 0.05$). For P2, the main effect of certainty was significant ($F(1, 58) = 5.19$, $p = 0.026$, $p^2 = 0.08$), with larger P2 amplitudes in uncertain versus certain contexts (20.37 ± 1.22 V vs. 19.27 ± 1.16 V, Figure 4B). Neither the main effect of group nor the group × certainty interaction was significant (all $p > 0.05$).

For other-pain trials, two-way ANOVAs were conducted on pain empathy-evoked P2 and P3 amplitudes. For P2, the main effect of group was significant ($F(1, 58) = 4.39$, $p = 0.041$, $p^2 = 0.07$), with the HAQ group showing significantly larger P2 amplitudes than the LAQ group when witnessing others' pain (5.68 ± 0.74 V vs. 3.50 ± 0.74 V, Figure 4D). The main effect of certainty was marginally significant ($F(1, 58) = 3.85$, $p = 0.054$, $p^2 = 0.06$), with larger P2 amplitudes in uncertain versus certain contexts (4.92 ± 0.58 V vs. 4.25 ± 0.54 V). The group × certainty interaction was not significant ($p > 0.05$).

For P3, the main effect of certainty was significant ($F(1, 58) = 103.45$, $p < 0.001$, $p^2 = 0.64$), with significantly larger P3 amplitudes in uncertain versus certain contexts (10.71 ± 0.80 V vs. 5.62 ± 0.61 V, Figure 4D). Neither the main effect of group nor the group × certainty interaction was significant.

Figure 4. ERP waveforms, topographies, and mean amplitudes for self-pain and other-pain conditions

Note: In waveform plots, red dashed lines represent the HAQ group in certain contexts (HAQ-Cer), red solid lines represent HAQ in uncertain contexts (HAQ-Uncer), blue dashed lines represent LAQ in certain contexts (LAQ-Cer), and blue solid lines represent LAQ in uncertain contexts (LAQ-Uncer). White-highlighted electrodes indicate those included in the analysis. Bar graphs show mean amplitude differences (M ± SE). Cer = certain; Uncer = uncertain; $p < 0.05$; ***$p < 0.001$.*

2.2.4 Correlation Analysis

The HAQ group exhibited significantly larger P2 amplitudes and higher unpleasantness ratings than the LAQ group when witnessing others'pain. Spearman correlation analysis further examined the relationship between other-pain-evoked P2 responses and unpleasantness. As shown in Figure 5 [Figure 5: see original paper], P2 amplitudes when witnessing others’ pain were significantly positively correlated with unpleasantness ratings in the HAQ group ($r_{28} = 0.43$, $p = 0.017$). However, this correlation was not significant in the LAQ group ($r_{28} = -0.23$, $p = 0.229$).

Figure 5. Scatter plot of the correlation between other-pain-evoked P2 amplitude and unpleasantness ratings

Note: LAQ = low autistic traits; HAQ = high autistic traits; $p < 0.05$.*

3.1.1 Participants

Using G*Power software and referencing previous research on autistic traits and empathy (Zhang et al., 2022; Zhao et al., 2019), we calculated that a minimum sample size of 193 participants was required to detect an absolute correlation coefficient of 0.20 with desired power ($1\text{-}\beta = 0.80$) and significance level ($\alpha = 0.05$). Experiment 2 ultimately recruited 381 healthy university students (202 males; age: 20.95 ± 0.12 years) to complete questionnaires. Participants reported no acute or chronic pain conditions, current medication use, or psychiatric history.

3.1.2 Data Collection and Analysis

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

Data were analyzed using SPSS 22.0 and AMOS 24.0. Pearson correlation analysis examined relationships among autistic traits, pain traits, and empathy traits, with p-values FDR-corrected for multiple comparisons. Structural equation modeling assessed model fit using ^{2}/df, GFI, AGFI, CFI, NFI, and RMSEA indices: (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, 2015). Mediation effects were tested using bias-corrected bootstrap methods with 5000 iterations; parameters were significant if the 95% confidence interval did not contain zero (Preacher & Hayes, 2008).

3.2.1 Common Method Bias Test

All variables in Experiment 2 were collected via self-report, raising potential common method bias concerns. Harman’ s single-factor test was conducted by performing an unrotated principal component factor analysis on all items. The first factor explained 11.03% of the variance, below the 40% critical threshold, indicating no severe common method bias.

3.2.2 Descriptive Statistics and Correlation Analysis

Table 2 presents means, standard errors, and intercorrelations among variables. Pearson correlation analysis revealed that autistic traits were not significantly correlated with pain sensitivity or pain fear but were significantly positively correlated with pain catastrophizing ($r_{379} = 0.19$, p_{fdr} < 0.001), indicating that higher autistic trait levels were associated with more negative emotional and cognitive responses to pain. Regarding relationships between autistic traits and empathy traits, autistic traits were significantly negatively correlated with perspective taking ($r_{379} = -0.19$, p_{fdr} < 0.001) and significantly positively correlated with personal distress ($r_{379} = 0.27$, p_{fdr} < 0.001). No significant correlations emerged with empathic concern or fantasy dimensions. These results suggest that individuals with high autistic traits exhibit weaker perspective-taking ability and stronger personal distress during empathy. Additionally, pain-related cognition was significantly positively correlated with trait emotional empathy: higher pain catastrophizing was associated with higher personal distress scores ($r_{379} = 0.47$, p_{fdr} < 0.001). Pain-related emotion was also significantly positively correlated with trait emotional empathy: higher pain fear was associated with higher personal distress scores ($r_{379} = 0.25$, p_{fdr} < 0.001). Thus, individuals with higher pain catastrophizing and pain fear are more prone to personal distress during empathy.

Table 2. Descriptive statistics and correlation matrix of study variables

Variable	M	SE	1	2	3	4	5	6	7	8
1. AQ	20.08	0.17	-	0.02	0.19***	0.19***	−0.19***	0.27***	−0.10	−0.07
2. PSQ	5.59	0.16	-	-	0.53***	0.39***	−0.02	0.16**	−0.01	−0.08
3. PCS	28.85	1.21	-	-	-	0.47***	0.19***	0.25***	−0.01	0.15**
4. FPQ	81.33	1.87	-	-	-	-	0.19***	0.29***	0.28***	0.36***
5. IRI-PT	12.67	0.34	-	-	-	-	-	−0.01	0.15**	0.19***
6. IRI-PD	11.12	0.45	-	-	-	-	-	-	0.28***	0.29***
7. IRI-EC	13.17	0.28	-	-	-	-	-	-	-	0.36***
8. IRI-FS	12.75	0.35	-	-	-	-	-	-	-	-

Note: M = mean; SE = standard error; AQ = Autism-Spectrum Quotient; PSQ = Pain Sensitivity Questionnaire; PCS = Pain Catastrophizing Scale; FPQ = Fear of Pain Questionnaire; IRI-PT = Interpersonal Reactivity Index Perspective Taking; IRI-PD = Interpersonal Reactivity Index Personal Distress; IRI-EC = Interpersonal Reactivity Index Empathic Concern; IRI-FS = Interpersonal Reactivity Index Fantasy. p_{fdr} < 0.05; **p_{fdr} < 0.01;** *p_{fdr} < 0.001. Bolded values indicate significant correlations.*

3.2.3 Mediation Analysis

Building on correlation analyses, we tested whether pain-related negative emotion and cognition mediated the association between autistic traits and personal distress. Autistic traits served as the predictor, personal distress as the outcome, and pain-related negative emotion and cognition as mediators (indexed by pain fear and pain catastrophizing). The structural equation model is shown in Figure 6 [Figure 6: see original paper]; all factor loadings exceeded 0.4. Model fit indices were: ^{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 revealed a significant total effect (c = 0.27, SE = 0.05, CI = [0.17, 0.36], $p < 0.001$), significant direct effect (c' = 0.16, SE = 0.05, CI = [0.07, 0.25], $p = 0.002$), and significant indirect effect (a*b = 0.11, SE = 0.04, CI = [0.05, 0.19], $p < 0.001$). These findings indicate that pain-related negative emotion and cognition partially mediate the influence of autistic traits on emotional empathy.

Figure 6. The mediating role of pain-related negative emotion and cognition in the influence of autistic traits on emotional empathy

Note: Path coefficients are standardized regression coefficients. ***$p < 0.01$;*** $p < 0.001$.*

4 Discussion

The present study combined a pseudo-dyadic pain empathy paradigm with questionnaire surveys to examine associations among autistic traits, first-hand pain, and empathy. In the pain empathy paradigm, individuals with high autistic traits exhibited stronger P2 responses and higher unpleasantness ratings when witnessing others'pain, partially attributable to elevated pain-related fear. Questionnaire data revealed that higher autistic trait levels were associated with lower perspective-taking scores but higher personal distress scores, with pain-related fear and catastrophizing mediating the link between autistic traits and personal distress. Thus, findings from both state and trait empathy studies support that high autistic traits are associated with heightened emotional empathy, partially stemming from more negative emotional and cognitive responses to pain.

Experiment 1 adopted a "quasi-autism" approach (Robinson et al., 2011; Guan & Zhao, 2015) by selecting individuals with high and low autistic traits from the general population to investigate state empathy differences using behavioral and ERP measures. The paradigm presented cue stimuli indicating whether participants would experience pain themselves or witness a partner (confederate) receiving painful stimulation, requiring ratings of emotional responses during pain anticipation and experience. This approach allowed characterization of pain empathy processing in a relatively naturalistic context. When rating anticipated pain stimuli, the HAQ group reported greater pain fear regardless of pain target, indicating more negative emotional responses to pain. During first-hand pain experience, HAQ and LAQ groups showed comparable pain-evoked responses, including N1 (sensory discrimination) and P2 (emotional-motivational) components, as well as comparable unpleasantness ratings. This lack of group difference is attributable to the individualized pain stimulation calibration, ensuring that all participants received moderately painful stimulation (rating of 6/10). When witnessing others' pain, the HAQ group exhibited significantly stronger pain empathy-evoked P2 responses and higher unpleasantness ratings than the LAQ group across both certain and uncertain contexts. No group differences emerged in empathy-evoked P3 responses, suggesting that autistic traits do not significantly affect late cognitive-evaluative processes of pain empathy. Similarly, Fan et al. (2014) using bodily pain images found no group differences in late ERP components (e.g., LPP) between ASD and control groups when participants judged whether images depicted pain or non-pain, suggesting comparable late cognitive-evaluative processing of pain empathy in ASD. However, meta-analytic findings indicate that individuals with ASD show significantly weaker cognitive empathy than typical individuals (Song et al., 2019), as assessed by 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 reflect differences in empathy assessment paradigms. Pain empathy-evoked P2 responses are associated with early, bottom-up emotional arousal and sharing processes, whereas P3 components reflect later, top-down cognitive evaluation (Cheng et al., 2014; Decety, 2011; Y. 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 region that encodes the affective dimension of pain (Baumgärtner et al., 2006). Thus, individuals with high autistic traits show stronger emotional arousal and experience more personal distress when facing others' pain, reflecting heightened emotional empathy. Correlation analysis revealed that in the HAQ group, larger empathy-evoked P2 responses were associated with higher personal unpleasantness ratings, supporting the notion that P2 responses reflect empathy-related emotional arousal. Given that the HAQ group reported stronger fear of impending pain and greater personal distress when witnessing others' pain, mediation analysis further revealed that autistic traits enhanced personal distress during pain empathy by increasing pain fear levels. This provides evidence linking autistic traits, pain-related negative emotion, and emotional empathy, highlighting the role of pain-related negative emotion in mediating the influence of autistic traits on emotional empathy.

Previous research has found that individuals with ASD exhibit low intolerance of uncertainty (Chamberlain et al., 2013; Neil et al., 2016). For example, Vasa et al. (2018) compared 57 children with ASD (ages 7–16) to 32 typically developing children and found that children with ASD reported significantly lower intolerance of uncertainty on both parent-report and self-report measures. Therefore, the present study included certainty as a variable. Results showed that certainty influenced unpleasantness ratings, P2, and P3 responses when witnessing others' pain (significant main effects of certainty), with uncertain contexts evoking higher unpleasantness ratings and stronger P2 and P3 responses. However, autistic traits' effects on empathy were not moderated by certainty (non-significant certainty × group interactions). No previous studies have examined whether pain and empathy abnormalities in ASD populations are related to certainty.

Future research should incorporate certainty as a variable in studies of pain and empathy in ASD populations to validate the present findings.

Experiment 2 used questionnaire surveys to examine relationships among autistic traits, pain, and empathy traits in a randomly recruited general population sample. The perspective-taking dimension of the empathy trait scale assesses individuals' tendency to understand and adopt others' psychological perspectives in real-life situations and is considered a measure of cognitive empathy. The personal distress dimension assesses self-centered reactions when encountering others in distress or stressful situations and is considered a measure of emotional empathy (Davis, 1983). Results showed that high autistic traits were associated with lower perspective-taking ability 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 observed in Experiment 1 (i.e., heightened personal distress and emotional arousal in HAQ individuals when witnessing others' pain). Regarding pain trait scales, pain fear and pain catastrophizing were significantly positively correlated with the personal distress dimension of trait empathy, and autistic traits were positively correlated with pain catastrophizing. Given these associations, structural equation modeling revealed that pain-related negative emotion and cognition mediated the relationship between autistic traits and heightened trait emotional empathy, further validating Experiment 1' s findings. For cognitive empathy, results showed divergent patterns across trait and state measures: trait cognitive empathy was negatively correlated with autistic traits (weaker cognitive empathy in high autistic trait individuals), whereas state cognitive empathy (P3 component when witnessing others' pain) did not differ significantly between high and low autistic trait groups, suggesting that autistic traits do not significantly affect state cognitive empathy. Similar dissociations between trait and state empathy have been reported, such as ERP responses to observed pain being unrelated to trait empathy scale scores (Galang et al., 2020; W. Li & Han, 2010) and bilateral insula activation during pain observation showing no significant association with trait empathy scores (Jackson et al., 2005). Future research should employ alternative empathy assessment paradigms, such as the Reading the Mind in the Eyes Test and Multifaceted Empathy Test, to further validate these findings.

Combined results from Experiments 1 and 2 demonstrate that high autistic traits are associated with heightened emotional empathy (i.e., more intense self-centered distress when others are in painful situations) and weaker cognitive empathy (i.e., difficulty adopting and understanding others' perspectives). Given the similarities between high autistic trait individuals and those with ASD across sensory, emotional, and cognitive domains, the present findings supplement evidence regarding empathy deficits in ASD populations from a subclinical perspective. Previous research indicates that individuals with ASD consistently show cognitive empathy deficits (Song et al., 2019; Ziermans et al., 2019), yet findings regarding emotional empathy are mixed (Bird et al., 2010;

Gu et al., 2015; Santiesteban et al., 2021). For example, some studies report that individuals with ASD can automatically imitate others' facial emotional expressions without emotional empathy impairment (Oberman et al., 2009), while others find reduced skin conductance responses when viewing facial emotion pictures, inferring weaker emotional empathy (Clark et al., 2008). Still others report stronger electromyographic responses to happy and fearful expressions, suggesting excessive emotional empathy (Magnée et al., 2007). The present results suggest that high autistic trait and ASD populations may not exhibit systematic empathy impairment but rather show a pattern of weaker cognitive empathy and excessive emotional empathy. Y. T. Fan et al. (2014) proposed that excessive emotional empathy may partially explain external social behaviors such as apparent coldness in ASD: when confronted with others' pain, individuals with ASD experience excessive emotional empathy and consequently employ attentional avoidance to prevent overwhelming distress, manifesting as apparent coldness and reduced prosocial behavior. Therefore, empathy interventions for these populations should emphasize "leveraging strengths while compensating for weaknesses" (Huo et al., 2021), attending to differential patterns across empathy dimensions to guide precise intervention development and enhance intervention efficacy.

Regarding first-hand pain, individuals with high autistic traits scored higher on pain catastrophizing and reported greater fear of pain cues during the task, indicating more negative emotional and cognitive responses. 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 individuals with ASD do not differ significantly from controls in pain thresholds overall, though modality-specific effects show lower pressure pain thresholds (greater sensitivity) in ASD groups. Regarding pain-evoked physiological responses, individuals with ASD show stronger responses to real-world medical pain than controls (Zhang et al., 2021). Pain is a multidimensional subjective experience comprising sensory, affective, and cognitive dimensions (Wiech et al., 2008). Pain thresholds reflect the sensory dimension, whereas pain-evoked physiological responses often reflect emotional and cognitive dimensions. Thus, meta-analytic results support that individuals with ASD do not differ significantly from typical individuals in pain sensory dimensions but show heightened sensitivity in emotional and cognitive dimensions. For example, individuals with ASD do not differ from controls in heat pain thresholds but report significantly higher pain intensity ratings for suprathreshold heat stimuli and score higher on pain anxiety and pain fear scales (Failla et al., 2020), supporting heightened affective and cognitive pain sensitivity in ASD populations. Gu et al. (2018) compared neural responses during pain anticipation and processing between ASD and control groups, finding that during pain anticipation, individuals with ASD showed significantly greater activation in dorsal and rostral anterior cingulate cortex than controls, whereas no group differences emerged in pain-related brain regions (e.g., anterior cingulate cortex and insula) during pain processing. This suggests that individuals with ASD may experience greater anxiety and fear while anticipating painful stimuli. Thus, findings from high autistic trait and ASD populations show similarities: both exhibit more negative emotional and cognitive responses to pain. Specifically, when facing actual or potential pain, high autistic trait and ASD individuals may appraise pain more negatively and show greater anxiety and fear. Given these response tendencies, medical professionals should be particularly attentive and patient with these populations in clinical settings (e.g., during injections or blood draws), perhaps by providing prior explanation to alleviate their anxiety and fear.

Results from both state and trait empathy studies support the mediating role of pain-related negative emotion and cognition in the association between autistic traits and emotional empathy. The heightened personal distress reported by high autistic trait individuals when witnessing others' pain is attributable to excessive negative emotion and cognition regarding pain. Cognitive bias modification for pain-related information can effectively alleviate pain-related negative emotion and cognition (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 (to allocate attentional resources to neutral information) showed lower anxiety sensitivity and functional disability at 6-month follow-up compared to controls (Sharpe et al., 2012). Chronic pain patients who received interpretation bias modification training (to interpret pain-related information as neutral) showed reduced pain-related negative emotion (An et al., 2020). Cognitive-behavioral therapy has also been found effective in reducing fear of movement, pain anxiety, and pain vigilance, thereby decreasing pain interference in daily life (Elomaa et al., 2009). Future research should investigate whether cognitive bias modification and cognitive-behavioral therapy can improve social skills in high autistic trait and ASD populations by reducing pain-related negative emotion and cognition.

The high autistic trait population is substantial, and their mental health and social functioning issues warrant attention. The present findings contribute to understanding pain and empathy problems in this population and inform development of targeted interventions to promote their mental health and social functioning. Furthermore, given the similarities between high autistic trait and ASD populations, this study supplements evidence regarding empathy deficits in ASD from a subclinical perspective, suggesting that empathy and social impairments may be understood from the perspective of atypical pain processing, thereby offering novel insights for improving social functioning in ASD populations.

However, research in ASD populations has primarily supported the role of heightened pain sensory sensitivity in explaining emotional empathy deficits (Chen et al., 2017; Y. T. Fan et al., 2014). Y. T. Fan et al. (2014) and Chen et al. (2017) combined psychophysical pain measurement and picture-evoked empathy paradigms to examine relationships between first-hand pain and pain empathy in ASD. They found that individuals with ASD had lower pressure pain thresholds than typical individuals, which were significantly positively correlated with unpleasantness ratings for others' pain in picture-evoked paradigms, suggesting that heightened sensory pain sensitivity in ASD leads to heightened emotional arousal during pain empathy. Research in typical populations has found that pain sensory sensitivity influences emotional empathy through its effects on pain-related emotion and cognition (Ren et al., 2020). Individuals high in pain catastrophizing tend to exaggerate their own pain and overestimate others' pain (Sullivan et al., 2006), and observers' pain fear levels positively predict pain empathy when viewing bodily pain images (Serbic et al., 2020). 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 populations exhibit heightened emotional arousal and personal distress when witnessing others' pain due to heightened sensitivity to their own pain across sensory, emotional, and cognitive dimensions. Future research should systematically characterize pain processing patterns in high autistic trait and ASD populations across multiple levels—including sensory, affective, and cognitive dimensions and their neural mechanisms—using subjective reports, physiological responses, electrophysiology, and neuroimaging to establish specific linkages between atypical pain processing patterns and core symptoms such as empathy deficits.

This study has several limitations. First, participants were healthy university students from a single institution with normal intelligence levels; however, we did not assess intelligence or other basic psychological traits, precluding complete control for potential confounding effects. Future research should match high and low autistic trait groups on intelligence and other factors to further validate the effects of autistic traits on pain and empathy. Second, the confederate (empathy target) was a single female experimenter to minimize variability across confederates. However, given the gender differences in ASD prevalence, the effects of autistic traits on empathy may be moderated by the confederate' s gender. Future studies should include confederates of different genders to examine whether gender congruence between empathizer and target influences results. Third, the present study used only moderately painful stimulation, precluding examination of stimulation intensity effects. Future research should employ multiple pain intensities and include non-painful control conditions to test whether the effects of autistic traits on empathy are pain-specific. Finally, the present study focused on a high autistic trait population; whether findings generalize to ASD populations remains to be tested. Despite similarities between high autistic trait and ASD populations in genetics, cognition, and behavior, which may facilitate understanding of ASD mechanisms and interventions, high autistic traits are not equivalent to ASD (Sasson & Bottema-Beutel, 2022). Autistic traits are considered an independent sixth dimension beyond the Big Five personality traits (Wakabayashi et al., 2006) and are not specific to ASD, as non-ASD populations such as those with anxiety disorders and schizophrenia also exhibit high autistic traits (Barlati et al., 2019; Lau et al., 2014; Tonge et al., 2016). Future research should validate the relationships among ASD symptoms, first-hand pain, and pain empathy in ASD populations.

5 Conclusion

The present study combined a pseudo-dyadic pain empathy paradigm with questionnaire surveys to investigate relationships among autistic traits, pain, and empathy. High autistic traits are associated with heightened emotional empathy, manifesting as more intense personal distress when others are in painful situations, partially attributable to more negative emotional and cognitive responses toward pain. However, high autistic traits are also associated with weaker cognitive empathy, manifesting as difficulty adopting and understanding others' perspectives. Given the similarities between high autistic trait and ASD populations, these findings may advance understanding of the biological mechanisms of ASD and inform intervention development, while also improving mental health and social functioning in high autistic trait populations. The results suggest that empathy and social impairments in these populations may be understood from the perspective of atypical pain processing. Future research should combine multiple pain modalities and multidimensional pain assessment systems to characterize pain processing features in high autistic trait and ASD individuals, establishing specific linkages between pain response patterns and core symptoms such as empathy deficits to provide targets for clinical intervention.

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