Absence of Embodied Empathy During Pain Observation in Asperger Syndrome

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Background: Asperger syndrome (AS) is a neurodevelopmental condition within the autism spectrum conditions (ASC) characterized by specific difficulties in communication, social interaction, and empathy that is essential for sharing and understanding others' feelings and emotions. Although reduced empathy is considered a core feature of ASC, neurophysiological evidence of empathic deficits before and below mentalizing and perspective taking is lacking. We explored whether people with AS differ from neurotypical control participants in their empathic corticospinal response to the observation of others' pain and the modulatory role played by phenomenal experience of observed pain and personality traits.

Methods: Sixteen right-handed men with AS (aged 28.0 ± 7.2 years) and 20 neurotypical controls (aged 25.3 ± 6.7 years) age, sex, and IQ matched, underwent single-pulse transcranial magnetic stimulation during observation of painful and nonpainful stimuli affecting another individual.

Results: When observing other's pain, participants with AS, in contrast to neurotypical control participants, did not show any amplitude reduction of motor-evoked potentials recorded from the muscle vicariously affected by pain, nor did their neurophysiological response correlate with imagined pain sensory qualities. Participants with AS represented others' pain in relation to the self-oriented arousal experienced while watching pain videos.

Conclusions: Finding no embodiment of others' pain provides neurophysiological evidence for reduced empathic resonance in people with AS and indicates that their empathic difficulties involve not only cognitive dimensions but also sensorimotor resonance with others. We suggest that absence of embodied empathy may be linked to changes at very basic levels of neural processing.

Key Words: Autism spectrum conditions, empathy, pain, embodiment, TMS, sensorimotor systems

Empathy, a defining feature of human interpersonal interaction, is crucial for sharing and comprehending another person's feelings and intentions and may ultimately shape our prosocial behavior (1). Broadly speaking, empathy is a complex construct ranging from low-level mechanisms such as contagion to higher-order processes such as perspective taking and mentalizing (2–4; see ref. 5 for a different view). Following Lipps's original concept that empathy allows one to access the inner state of others by "feeling into them," recent neuroscientific models postulate that watching or imagining another person's mental state automatically triggers the representation of the same state in the observer (2–4). Accordingly, observing facial expressions of disgust activates those sectors of the insula and the cingulate cortex that are involved in first-person experience of disgust (6). Similarly, observing another person being touched elicits the activation of somatosensory systems involved in first-person tactile perception (7–9). Thus, observing emotions or bodily sensations results in brain activations largely overlapping those occurring during the direct experience of the same feelings, which suggests that empathic brain responses may rely on resonant, mirror-like systems (2–4).

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Empathy is clearly called into play when we observe others suffering from either psychological (e.g., social rejection) or physical (e.g., cuts) pain. Pain has long been considered a private, subjective state; however, the ability to understand and indirectly experience others' pain is important in learning to minimize one's own exposure to it. Being able to share and understand others' pain is probably an important prerequisite to care for others, feel and show compassion, and ultimately act in a prosocial way (1,10,11).

Pain is an interesting model to test theories of empathy based on shared representations between self and others in that it has distinct sensory (e.g., intensity, localization) and emotional (e.g., unpleasantness) components that are respectively encoded in separate sensorimotor and affective nodes of a complex neural network called the "pain matrix" (12,13). The social dimension of pain has recently become the focus of neuroimaging and neurophysiological studies suggesting that vicarious experience of others' pain, just like direct experience of pain, activates both sensorimotor and affective nodes of the pain matrix (8,14–19).

Asperger syndrome (AS) is a neurodevelopmental condition within the autistic spectrum, characterized by impaired communication, difficulties in social interaction, repetitive behaviors, and narrow interests. Although AS, as well as other autism spectrum conditions, is often described in terms of reduced empathic abilities (20,21), evidence for reduced empathy in domains different from mentalizing and perspective taking is meager. It includes studies based on subjective measures on the phenomenology of empathy in AS (21–24) and an electromyographic study showing absence of automatic facial mimicry, an index of contagion, while observing others' emotional facial expressions (25). Resonant mirror-like systems may constitute the neural mechanism underlying embodied simulative processes grounding our social understanding and empathic response (26,27).

Interestingly, on the basis of anatomic (28) and neurophysiologic (29–31) findings, underactivation of mirror systems has

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recently been posited as a crucial feature of autism spectrum conditions (ASC) (32,33).

Here we used single-pulse transcranial magnetic stimulation (TMS) to explore a rudimentary form of empathy, called "sensorimotor contagion," elicited in neurotypical participants when they observe painful stimuli applied to the body of another person (16,17). Sensorimotor contagion is here indexed by a reduction of corticospinal excitability recorded from the specific body part that is vicariously affected by the observed painful stimulation. Importantly, this observational pain-related inhibition is reminiscent of the corticospinal inhibition found during actual noxious stimulation (34–36). We asked whether individuals with AS do embody others' pain as if they were vicariously feeling it and whether their proposed empathic difficulties may extend from higher-order to more basic levels of neural processing. We expected to find neurophysiological evidence of reduced empathic abilities in people with AS in terms of reduced or absent sensorimotor contagion during the observation of pain affecting another person, and at the same time to replicate our previous findings on neurotypical participants (16,17). Further, for each group, we expected to find a different relationship between participants' neurophysiological response and their subjective ratings and dispositional measures.

Methods and Materials

Participants

Sixteen right-handed (37) men with AS (aged 28.0 ± 7.2 years) and 20 neurotypical controls (C) (aged 25.3 ± 6.7 years) free from any contraindication to TMS (38) agreed to take part in the study by giving written informed consent. Participants with AS received their diagnosis from an Autism Research Centre expert and qualified professional clinicians according to DSM-IV criteria (*Diagnostic and Statistical Manual of Mental Disorders,* 4th ed., 1994) and had no history of neurological or other psychiatric disorders. Control participants had no neurological, psychiatric, or medical problems. All participants completed the Autism Spectrum Quotient (AQ) (39), a self-administered questionnaire for measuring the number of autistic traits. Although the AQ is not diagnostic, it is a useful check on diagnosis because it has been validated in a clinical sample (40). Among participants with AS, 14 of 16 (87.5%) scored above the cutoff for AS (= 32), compared with only one individual in the control group (5%). The two groups were matched for sex (all men), age (AS = 28.0 ± 7.2 years, C = 25.3 ± 6.7 years, $t = -1.16$, $p = .25$), full-scale IQ (AS = 118.9 ± 15.6, C = 122.9 ± 6.9, $t = 1.01$, $p = .32$), Verbal IQ (AS = 118.7 ± 9.7, C = 121.3 ± 8.3, $t = .74$, $p = .39$), and Performance IQ (AS = 119.5 ± 13.1, C = 119.9 ± 10.1, $t = 0{,}11$, $p = .32$) effectively assessed using the WASI (41,42). The study was approved by Addenbrookes Hospital Local Research Ethics Committee and was carried out in accordance with the ethical standards of the 1964 Declaration of Helsinki.

Electromyogram and TMS Recordings

We assessed functional modulation of corticospinal excitability during the observation of video clips showing painful and nonpainful stimuli. Motor-evoked potentials (MEPs) induced by focal single-pulse TMS of the left primary motor cortex (M1) were simultaneously recorded from two right-hand muscles, the first dorsal interosseous (FDI), and the abductor digiti minimi (ADM). TMS pulses were delivered by setting the intensity at 120% of the resting motor threshold (Supplement 1).

Visual Stimuli

Four types of video clips (lasting 1.8 sec each) were presented on a 19-inch screen. Video clips were 1) "static": static right hand; 2) "Pain": needle deeply penetrating the FDI muscle; 3) "Touch": cotton swab gently touching the FDI region; and 4) "Tomato": needle deeply penetrating a tomato. Thus, whereas participants' FDI muscle was vicariously involved by the painful stimulation, the ADM muscle served as a somatotopic control because it was not shown to be penetrated. All video clips were the same as used in our previous study (17). Studies of TMS report that watching moving body parts or a hand using tools increases corticospinal excitability (43,44). To avoid such an effect, the model's hand did not move, and the syringe holder was not visible. Videos did not include the model's face to avoid potential confounding effects due to possible emotion recognition deficits among individuals with ASC (45).

Observational Paradigm and Task Instruction

The experiment was programmed using Cogent 2000 (Cogent 2000 team, Functional Imaging Laboratory, Institute of Cognitive Neuroscience) and Matlab 7 (http://www.mathworks.com). Each type of video clip was presented in a separate block of 18 trials. Block order was counterbalanced across participants. A magnetic pulse was randomly delivered during the last .6 sec before the end of the clip, once the needle had completely penetrated or the cotton swab had touched the hand. This procedure ruled out the possibility that any changes in corticospinal excitability were due to tool use observation per se (43). Intertrial interval lasted 10 ± .3 sec based on the evidence that TMS per se delivered for 1 hour at .1 Hz does not change corticospinal excitability (46).

Participants were given written instructions to stay relaxed, watch carefully, and "try to identify with the model and think how is he/she feeling." Previous TMS research suggests that in control participants, corticospinal inhibition during pain observation is largely automatic and independent from whether the observer is instructed to identify with the model (17). However, because of AS's known difficulties in empathizing and mentalizing (21,47), we decided to ask participants explicitly to identify with the model. Direction of eye gaze toward the screen was monitored using a rearview mirror. At the end of each block, to verify further and encourage attention, participants had to answer two questions concerning the videos (e.g., "How old do you think the model is?" and "Were different syringes used to pierce the tomato?").

Subjective Ratings of the Observed Stimuli and Dispositional Measures

Before the TMS session, participants were shown all the videos and rated (along a 10-cm-long visual analog scale [VAS]) the level of arousal ("how much does the video grab your attention?") and aversion ("how much does the video upset you?") experienced while watching each video. After the TMS session, participants rated their ability to identify with the model ("how much during the experiment were you able to identify with the model by simulating internally his experiences and sensations?"), the intensity ("how much do you consider the pain sensation represented in the video to be intense?"), and unpleasantness ("how much do you consider the pain sensation shown in the video to be unpleasant?") of the pain shown in the videos.

Participants were also asked to imagine how the pain would feel, if applied to them. The qualities of the imagined pain were measured using the McGill Pain Questionnaire (MPQ) (48), which is made up of Sensory (items 1–10, 17–19) and Affective

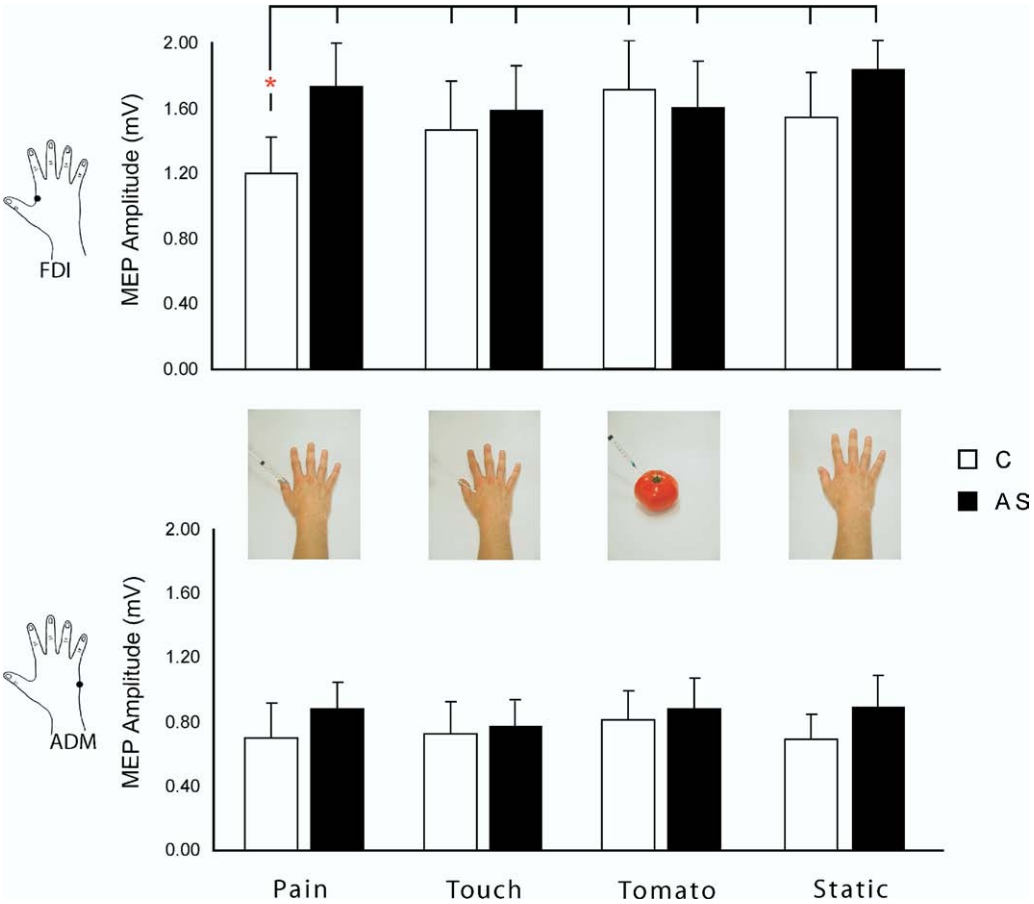

Figure 1. Neurophysiological results. Raw motor-evoked potentials (MEP) amplitudes recorded from first dorsal interosseous (FDI; target) and abductor digiti minimi (ADM; somatotopic control) muscles for each observation condition. The Asperger syndrome group (black bars), unlike control participants (white bars), do not show pain-specific corticospinal modulation of the activity recorded from the FDI, the muscle vicariously involved by the painful stimulation. Neither group showed any modulation of the activity recorded from ADM, the control muscle not involved by the painful stimulation. Asterisk indicates significant comparisons ($p < .05$). Bars denote SEM. C, controls; AS, Asperger syndrome.

(items 11–15, 20) subscales, and through the Hurts value, a rating between 0 and 10 indicating how much the participants thought the injection would hurt them.

Before the experimental session, participants filled in three self-report questionnaires: the AQ (39), the Systemizing Quotient-Revised (SQ-R) (49), and the Empathy Quotient (EQ) (22). After taking part in the experiment, participants filled in the Interpersonal Reactivity Index (IRI) (50), which is composed of two cognitive subscales (perspective taking [PT] and fantasy scale [FS]) and two affective subscales (empathic concern [EC] and personal distress [PD]), and the 20-item Toronto Alexithymia Scale (TAS 20) (51). All participants completed all questionnaires except for the SQ-R, which was not filled in by two AS participants, and the TAS 20, which was not filled in by four AS and six C.

Results

Neurophysiological Results

MEP amplitudes were entered in two (one for each muscle) mixed-model, two-way analysis of variance with Group (AS, C) as between-subjects and Condition ("Static", "Pain," "Touch," "Tomato") as within-subject factors. Analysis of MEPs recorded from the FDI muscle revealed a significant Group × Condition interaction [$F(3,102) = 3.67$, $p = .01$], which was accounted for by the lower FDI MEP amplitude in controls during the Pain condition (all $ps < .05$; Figure 1; Supplement 1). No modulation of MEPs recorded from the ADM muscle was found (all $ps > .45$).

In contrast to neurotypical control subjects, participants with AS did not show a muscle-specific modulation to the observation of other's pain. Therefore, when observing another person's pain, participants with AS did not respond as if they were themselves affected by the noxious stimulation (16,17,34–36).

Importantly, the absence of modulation in response to others' pain cannot be ascribed to nonspecific across-groups differences in overall corticospinal reactivity. The two groups had in fact comparable motor thresholds (AS: 49% ± 11, C: 47% ± 8, $t = -.79$, $p = .43$) and MEP amplitudes (as evidenced by the lack of main effect of Group for both muscles, $ps > .6$) in keeping with a previous TMS study on people with AS (30).

Subjective Ratings

Table 1 shows subjective ratings. All experimental video clips were similarly rated by the two groups ($ps > .10$) except for the Static condition, which was significantly less arousing for AS ($p < .02$); this is not surprising in the context of ASC reduced interest in social stimuli (52,53). Participants with AS, compared with control participants, perceived themselves less able to identify with the model being touched ($t = 2.07$, $p = .050$, see Table 1) and tended to judge the touch as more painful ($t = -1.82$, $p = .08$), which is in line with subjective reports (54,55) and recent

Table 1. Subjective Ratings of the Video Stimuli and of the Imagined Pain

	AS	C	*t*	*p*
Arousal Static	2.06	3.48	2.34	.02[a]
Arousal Pain	5.91	6.22	.50	.62
Arousal Touch	3.53	4.63	1.65	.11
Arousal Tomato	4.66	4.86	.27	.79
Aversion Static	.65	.68	.16	.87
Aversion Pain	5.15	4.84	–.37	.71
Aversion Touch	1.71	1.19	–1.22	.23
Aversion Tomato	2.09	2.17	.13	.90
Intensity Pain	7.42	7.25	–.27	.79
Intensity Touch	2.05	1.11	–1.82	.08
Unpleasantness Pain	7.75	7.42	–.56	.58
Unpleasantness Touch	2.08	1.37	–1.20	.24
Empathy Static	4.83	4.69	–.14	.89
Empathy Pain	5.21	6.59	1.55	.13
Empathy Touch	4.56	6.15	2.07	.05[a]
Hurt	5.75	6.50	.98	.33
MPQ Sensory	23.62	24.10	.17	.87
MPQ Affective	4.75	4.10	–.49	.63

Overall, the two groups judged the stimuli and the imagined pain similarly. The Asperger syndrome group (AS) judged the static hand to be less arousing and tended to consider the touch more painful than the control group (C). Furthermore, compared with control subjects, participants with AS reported to be less able to identify with the model during the Touch condition.

MPQ, McGill Pain Questionnaire.

[a]Statistically significant *p* levels ($p < .05$).

empirical results (56,57) of tactile hypersensitivity. Crucially, when asked to imagine how they would feel if receiving the painful stimulation shown in the videos and to rate the sensory and affective qualities of imagined pain, control participants and individuals with AS gave similar ratings (all $ps > .33$). Thus, the lack of sensorimotor contagion in AS cannot be attributed to group differences in the imagined "painfulness" of the observed events.

Dispositional Measures

Participants filled in five self-report questionnaires that measure autistic traits (AQ), drive to systemize (SQ-R), empathy (EQ and IRI) and alexithymia (20-item TAS-20). The AS group scored significantly higher on AQ, SQ-R ($ps < .008$), and significantly lower on both empathy questionnaires (EQ and IRI, $ps < .001$) than control participants. The group with AS also scored higher than control participants on TAS-20 ($p < .001$) and met the clinical cutoff for high degree of alexithymia (51) (Table 2, Supplement 1).

Importantly, no difference between AS and control participants was found on the IRI Personal Distress (PD) subscale ($t = .20$, $p = .84$; Cohen's $d = .07$), a measure of the tendency to respond in a self-oriented manner despite the situation would require a more adaptive other-oriented behavior (49). By contrast, AS scored much lower than controls in other-oriented dimensions of empathy (all $ps < .03$) and in particular on the IRI Fantasy scale ($t = 3.2$, $p = .003$; Cohen's $d = 1.08$), a measure of the tendency to identify with others (50) (Table 2).

Relation Between Subjective and Neurophysiological Measures

To explore the relation between corticospinal responses to others' pain and participants' subjective measures, correlation analyses were performed (Supplement 1).

Within the control group, we found a significant negative correlation between pain-related corticospinal modulation and sensory qualities of pain ($r = –.50$, $p = .03$; Figure 2A); thus, corticospinal inhibition was stronger in those who judged the observed-pain to be more intense and with more pronounced sensory qualities; by contrast, no relation with pain affective qualities was found ($r = –.34$, $p = .14$; Figure 2B). This relation with sensory but not affective qualities of pain is in keeping with previous research (8,16,17) and suggests that the corticospinal inhibitory response may reflect the simulation of sensory (locus, intensity) but not emotional qualities of the pain attributed to the model.

Importantly, corticospinal responses to others' pain in AS correlated only with self-oriented arousal experienced while watching Pain ($r = .67$, $p = .005$; Figure 2C). This result suggests that AS embodiment of others' pain may be affected by the level of evoked self-oriented arousal.

To check for a continuum between ASC and neurotypical development, correlations across groups were performed. Corticospinal inhibition was maximal in participants with fewer autistic traits (AQ: $r = .49$, $p = .003$; Figure 2D) and in those with higher empathic scores (EQ: $r = –.33$, $p = .04$, Figure 2E; cognitive IRI: $r = –.36$, $p = .03$, Figure 2F). This indicates that a lack of sensorimotor contagion is associated, along the neurotypical–AS continuum, with reduced empathy and presence of autistic traits. Moreover, control participants with high scores on the SQ-R (on which AS individuals typically score high) showed less inhibition ($r = .49$, $p = .03$; Figure 2G).

Discussion

We found that when observing pain affecting another person, participants with AS, in contrast to neurotypical control participants, did not show any neurophysiological modulation of their corticospinal system. Because inhibition of MEP amplitude contingent on observation of others' pain is considered an index of sensorimotor contagion (16,17), our findings indicate that embodied empathic pain resonance effects are absent in AS participants.

Further, whereas control participants' response is linked to the sensory relevance of the pain attributed to the model (the more somatomotor contagion the stronger the imagined pain) (16,17), the neurophysiological response of participants with AS

Table 2. Personality Trait Measures Compared in the Two Groups

	AS	C	*t*	*p*	*d*
AQ	37 (5)	18 (6)	–9.7	<.001[a]	3.27
EQ	19 (8)	30 (11)	6.1	<.001[a]	2.10
SQ-R	84 (24) ($n = 14$)	62 (19)	–2.9	.007[a]	.98
IRI PT	14 (4)	17 (3)	2.4	.020[a]	.80
IRI FS	11 (5)	17 (5)	3.2	.003[a]	1.08
IRI EC	15 (5)	19 (4)	2.6	.013[a]	.86
IRI PD	12 (5)	13 (5)	.2	.844	.07
IRI	52 (10)	65 (10)	.2	<.001[a]	1.22
TAS 20	63 (10) ($n = 14$)	41 (8) ($n = 14$)	–6.1	<.001[a]	2.33

Mean (SD) scores on personality trait measures. The two groups differed in all but IRI PD.

AQ, Autism Quotient; AS, Asperger syndrome; C, control groups; EC, Empathic Concern; EQ, Empathy Quotient; FS, Fantasy Scale; IRI PD, Interpersonal Reactivity Index Personal Distress subscale; PT, Perspective Taking; SQ-R, Systemizing Quotient; TAS 20, Twenty-item Toronto Alexithymia Scale.

[a]Statistically significant *p* levels ($p < .05$).

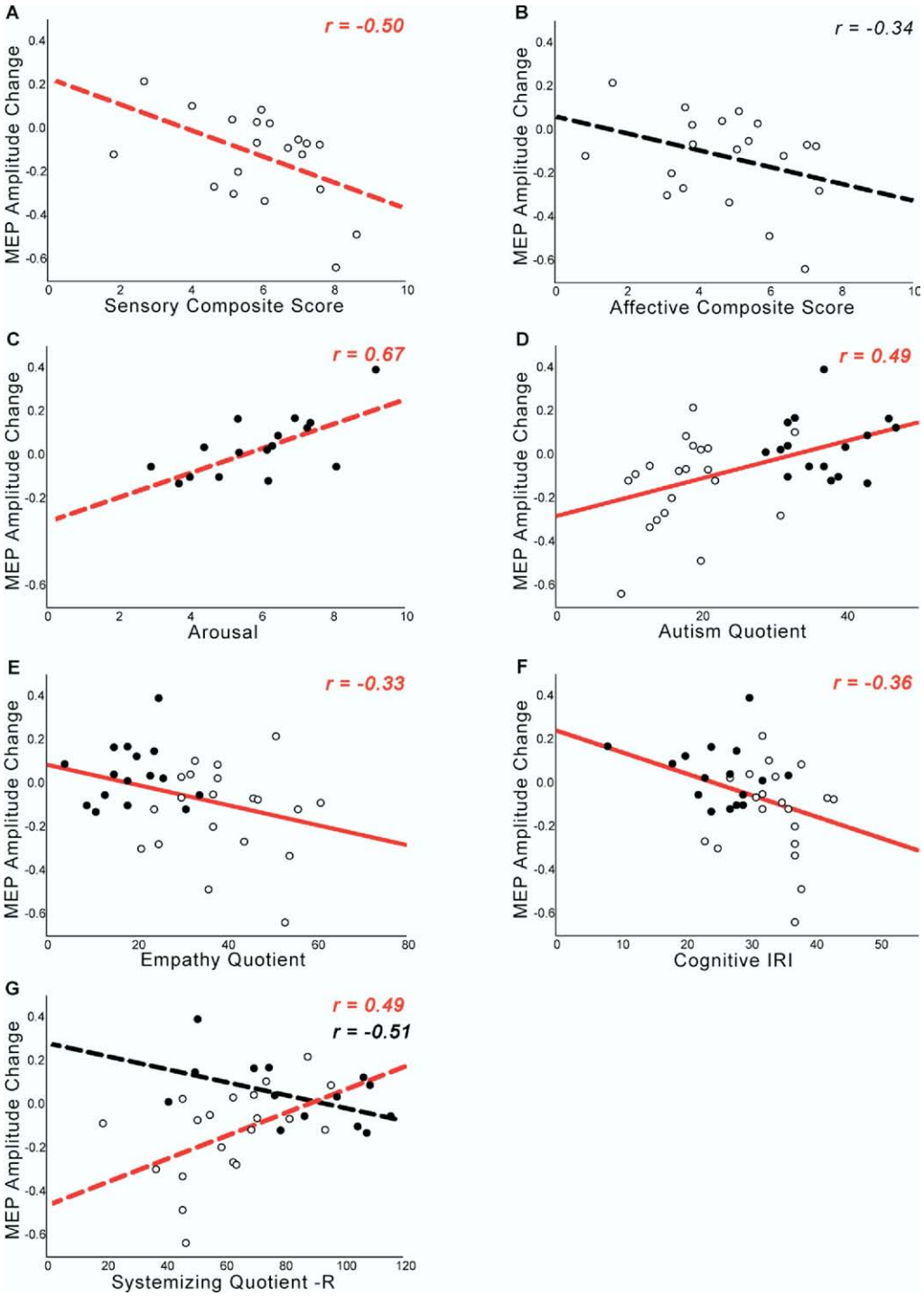

Figure 2. Correlations between brain responses to others' pain and subjective measures. Control participants (unfilled circles): correlations between motor-evoked potentials (MEP) amplitude change index and **(A)** sensory qualities of the pain shown in the videos ($p = .03$), **(B)** affective qualities of the pain shown in the videos ($p = .14$), **(G)** Systemizing Quotient—Revised ($p = .03$). Participants with Asperger syndrome (filled circles): correlation between MEP amplitude change and **(C)** self-oriented arousal felt during the observation of Pain videos ($p = .005$), **(G)** Systemizing Quotient—Revised ($p = .06$). Across groups correlations between MEP amplitude change and **(D)** Autism Quotient ($p = .003$), **(E)** Empathizing Quotient ($p = .04$), **(F)** Cognitive Interpersonal Reactivity Index ($p = .03$). Lighter color characters and lines represent statistically significant correlations ($p < .05$). Dashed regression lines represent correlations within one group of participants, whereas solid regression lines represent across groups correlations. IRI, Interpersonal Reactivity Index.

is instead linked to the level of self-oriented arousal experienced while observing Pain videos (the more somatomotor contagion, the weaker the arousal). Participants with AS seemed to code others' pain in a self-oriented manner, likely by taking an egocentric stance. We investigated a particularly salient and socially relevant situation—namely, the observation of another person's pain—and, to the best of our knowledge, found for the first time in people with ASC neurophysiological evidence of

absent embodied empathic reactivity to the sensations experienced by another person. Evidence of reduced embodiment in ASC comes from recent behavioral studies investigating motor contagion. In contrast to neurotypical control participants, people with ASC showed reduced automatic mimicry of somebody else's emotional facial expression (25,31) and absence of contagious yawning (58). Moreover, their grasping was not affected by observation of another person performing a similar action or gazing at the object being grasped (59). In a similar vein, neuroimaging studies showed that unlike neurotypical controls, participants with ASC fail to activate somatosensory and premotor cortices during observation of neutral faces (60).

In this study, although participants with AS did not embody others' pain, the observation of painful stimuli inflicted to the hand muscle of another person inhibited control participants' corticospinal representation of the same muscle (i.e., the FDI muscle). That MEPs recorded from the ADM muscle are not modulated cannot be explained in the terms of reduced reactivity of this muscle. Indeed, when videos depict the ADM being penetrated by a needle, similar corticospinal inhibition of this muscle has been observed (17).

The corticospinal inhibition found in neurotypical observers resembles that found during actual experience of pain (34–36) and may be linked to a vicarious attempt to decrease pain by relaxing the muscle that is penetrated by the needle.

This inhibitory effect is likely due to the activation of sensory mirror-like resonance mechanisms extracting basic sensory qualities of another person's painful experience (e.g., location and intensity of the noxious stimulus) and mapping them onto the observers' sensorimotor system according to fine-grained somatotopic rules (16,17). This view is supported by the inhibitory sign of the effect, by the muscle specificity and by the correlation of MEP inhibition with observed pain sensory qualities. The involvement of the pain matrix sensory node in the empathic mapping of others' pain is also supported by specific modulation of somatosensory (8) and laser-evoked potentials (14) and by neuroimaging evidence of parietal somatic and multisensory activations during pain observation (18,61).

One may alternatively wonder whether the neurophysiologic effect found in neurotypical control participants but not in participants with AS could be explained by the simulation of a defensive motor reaction to pain. However, real pain may cause an upper limb withdrawal reflex that implies suppression of all hand muscles' activity (34–36). Thus, the high selectivity of the pain-related resonant effect speaks against the simulation of a massive retraction reflex (16).

As previously mentioned, the strength of the modulatory effect found in neurotypical control participants was related to their subjective ratings of imagined pain sensory qualities. Stronger corticospinal inhibition was found in those control participants who rated the observed pain as more intense and vice versa. In principle, our finding of absent sensorimotor contagion in people with AS could have been due to participants with AS being insensitive to pain. However, this is not the case because the two groups did not differ in their ratings of pain sensory and affective qualities. In keeping with this, anecdotal reports indicating that individuals with ASC have reduced pain sensitivity failed to be empirically supported. Behavioral and facial reactions to venipuncture are in fact comparable in AS/high-functioning autism (HFA) and neurotypical individuals (62) and adults with HFA even have increased sensitivity to thermal pain (57).

The only empathic measure on which the group with AS did not score lower than control subjects was the IRI Personal Distress (PD) subscale, which taps the tendency to experience self-oriented distress and discomfort in response to somebody else's distress or misfortune (50). PD scores may in fact be even higher in AS than control participants (22,24). It is worth noting that throughout typical development, the level of personal distress decreases while appropriateness of helping behaviors increases (63). Human infants initially respond to the distress of others with their own distressed cries, and only later on they do engage in more appropriate other-oriented helping behaviors (64,65). Furthermore, if an adult observer of somebody in need of help is overwhelmed by his or her own emotional experience, this reduces comforting and helping behaviors toward those suffering (1,66). Individuals with ASC also often show reduced or absent comforting responses toward a distressed other (67).

The propensity of participants with AS to respond in a self-oriented manner may be linked to their tendency not to incorporate the model's hand into their own sensorimotor system. Individuals with AS tend to adopt an egocentric stance by which the others' states are represented primarily in relation to the self and find it difficult to identify with others (68). This view is supported by the absence of sensorimotor contagion as well as by the self-referred coding of others' pain. In keeping with this, our evidence of reduced empathic abilities in AS is consistent with Piaget's account of empathy as "decentering," or responding nonegocentrically (69).

AS scored much lower than neurotypical control participants in mature dimensions of empathy and in particular on the IRI Fantasy scale, which taps respondent's tendencies to transpose himself or herself imaginatively into the feelings and actions of fictitious characters in books, movies, and plays (50). According to Davis's definition and to the items included (e.g., "When I watch a good movie, I can very easily put myself in the place of a leading character"; "After seeing a play or movie, I have felt as though I were one of the characters"), the Fantasy subscale is the IRI subscale tapping more into the tendency to identify with others, taking their place as if getting into their body, which is particularly relevant to our experiment.

It is important to point out that reduced empathic abilities and egocentric stance in people with AS do not imply that they lack moral sense or behave egoistically. Clinical accounts describe people with AS as having a strong sense of opposing injustice and caring about social issues and equality (70,71). Moreover investigations of moral sense in children with autism failed to find any differences with matched typically developing control participants (72,73).

Before this work, three studies (24,74,75) have addressed the relationship between AS and alexithymia a multifaceted construct encompassing 1) difficulty in identifying, describing, and communicating subjective feelings; 2) difficulties in differentiating feelings from bodily sensations of emotional arousal; 3) diminished fantasy; and 4) stimulus-driven, externally oriented cognitive style (76). Our results converge to indicate that individuals with AS have a high degree of alexithymia.

Moreover, it has been suggested (74) that individuals with AS present with a type of alexithymia in which conscious awareness of emotional arousal is intact or even increased, whereas its cognitive expression is reduced. This, too, is in accord with our finding of a positive correlation between the absence of neurophysiologic modulation and high levels of self-perceived arousal in individuals with AS. Awareness of emotional states in the self can be considered as the basis for identification and sharing the feelings and thoughts of others (4,77). The complex relation

between empathy, alexithymia, and ASC hence provides an interesting topic for further investigations.

In our everyday life, we often encounter people suffering. Observing another person's physical pain may immediately make us wince and "feel" her or his pain in our own body. Finding no embodiment of others' pain provides neurophysiologic evidence for reduced empathic resonance in people with Asperger syndrome and further indicates that their empathic difficulties involve not only cognitive dimensions but also a reduction in the basic sensorimotor resonance with others.

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