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Investigating emotion expression and empathy in ASD through abstract drawings and facial expressions

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Investigating emotion expression and empathy in ASD through abstract drawings and facial expressions

Structure:

Introduction

According to the World Health Organization (WHO, 2023) about 1% of the population has the neurodevelopmental condition autism. Since autism presents differently between individuals and developmental stages, study findings regarding Autism Spectrum Disorder (ASD) are not always consistent and several stereotypes may influence how we perceive and treat people who show traits of neurodivergence in our society. Since prior studies made inconsistent conclusions about social cognitive skills like emotion communication in ASD, the following master thesis is part of a PHD study on autistic adults and emotion communication through a new art paradigm while using a brain-imaging method (Pelowski, Kim & Silani, study proposal). Prior literature about autism often left out mentioning the Double Empathy Problem, a key concept describing underlying mechanisms in autistic- and non-autistic communication which served as a theoretical basis in the following study. My project focused on emotion expression and empathy comparing autistic individuals and neurotypicals in a facial expression- and a drawing task while measuring empathy-related brain activation, since research so far has concentrated on emotion recognition rather than emotion showing. The aim was to make the connection clearer between emotion expression, recognition and empathy and to give more insight into possible differences of social cognition in autistic people in comparison to neurotypical people. The overall goal was to reduce negative stereotypes towards autistic individuals by checking existing study results about atypical emotion communication and empathy on a behavioral as well as neurological level and also detecting some social cognitive similarities with non-autistic people. In addition, the project should bring scientific results that contribute to more mutual understanding between neurotypicals and autistic people.

This thesis consists of a theoretical part and a practical part. The theoretical part contains definitions of important terms and key research findings. It begins with a literature review on autism, including the development and neurobiological basis as well as common stereotypes, possible support for autistic people and study findings about social cognitive skills like reading facial expressions (the facial expression task is one of the main paradigms of the current study). This is followed by a literature review on empathy including the different types of empathy, the acquisition of empathy and its connection to the communication of emotions. After summarizing study findings about empathy and the recognition of emotions, it is described how

empathic processes play also a role in emotion expression, although there is not much research on the topic. To fill the research gap, a theoretical approach is made stating that parts of empathy such as perspective taking are required even in the expression of emotions (not only in emotion recognition). Finally, studies about ASD and possible differences in empathy are summarized. In the practical part, the current study investigating the activity of brain regions associated with empathy is described. To test the assumed connection between empathic brain activation and emotion expression, brain activity was measured during facial expressions and an art paradigm. Since there is evidence that autistic people benefit from other forms of communication (with pictures or symbols), empathy-related brain activation is compared between the classical facial expression task and the novel drawing task. The thesis ends with a discussion.

Autism

Definition and Diagnostics

According to ICD-11 ASD is characterized by three key symptoms:

> First, ‘persistent deficits in the ability to initiate and sustain reciprocal social interaction and social communication’ (World Health Organization, 2019a). Second, by ‘a range of restricted, repetitive, and inflexible patterns of behaviour, interests or activities that are clearly atypical or excessive for the individual’s age and sociocultural context’....Third, ‘symptoms should result in significant impairment in personal, family, social, educational, occupational or other important areas of functioning’. (Greaves-Lord et al., 2022, p.2)

The beginning of these symptoms generally develops early in childhood, however the degree to which and when they fully manifest can vary through all ages (Greaves-Lord et al., 2022). In summary, “ASD is a ‘lifelong condition, of which the manifestations and impact are likely to vary according to age [developmental stage], intellectual and language abilities, co-occurring conditions and environmental context’” (Greaves-Lord et al., 2022, p.2).

As the name suggests, ASD is meanwhile seen on a spectrum, implicating that all levels of intellectual and verbal skills can occur (Freitag, 2021). Previously, for example in the ICD-10 (version 2024), there were different types of autism with

certain criteria: Childhood autism, Asperger syndrome and Atypical autism (Freitag, 2021). According to the author, sub-diagnoses can still be made in the ICD-11 but both diagnoses, Childhood autism and Asperger syndrome, are summarized to ASD and the diagnosis of Atypical autism does not exist anymore. Taken from Freitag (2021), sub-diagnoses are made regarding the extent of cognitive development (intelligence, adaptive behavior) and language.

Non-clinical models of ASD

Since in the current project we saw autism as a part of the participant's personality, and not as a disorder from which they suffer, the following part looks at non-clinical perspectives of autism. The aforementioned definition and diagnostic criteria are set in a very clinical and rather deficit-oriented context. However, there are other perspectives on ASD as well, starting with how one defines an autistic individual: Cascio (2014) cited Estroff (1993) stating that ASD is one of the "'I am' illnesses" (Cascio, 2014, p.1). This was explained, by the author, as a case where autism affects the individual so broadly that autistic people often call themselves "autistics" or "people on the spectrum"; put in other words, ASD is an integral part of identity and the way life is experienced. Additional to just the disorder aspects, other aspects are essential as well: "...A holistic view that considers the place of autism in the larger sociocultural context…[,] Attention to the local and historical particularity of the concept of autism…[and] Attention to the lived experience of people with autism and those close to them" (Cascio, 2014, p.2).

In a study by Kirchner et al. (2016), autistic adults without intellectual deficits were compared to neurotypical individuals concerning subjective character strengths and satisfaction with life. Even though the ASD group generally scored lower in the character strengths rating, except for intellectual strengths and strengths of restraint (no group differences), the most frequent strengths in autistics were open mindedness, authenticity and love of learning (Kirchner et al., 2016). The researchers found a significantly higher frequency for creativity in the ASD group compared to the control group and the most frequent social skill mentioned by autistic participants was fairness. In terms of life satisfaction, they found that emotional and interpersonal strengths had the highest positive correlations with it in the ASD group. This emphasizes that support in social and emotional domains is important for autistic people (Kirchner et al. 2016).

In an interview that our research group held with autistic individuals about what they would improve in our study plan, we also received interesting insights on this topic. When we additionally asked them about their strengths or what they "liked" about their ASD, they mentioned their way of thinking, a good spatial orientation, a strong sense for justice and that they prefer in-depth conversations over small-talk. As mentioned earlier, the interview demonstrated that these participants fully integrated the diagnosis as part of their personality and do not see it as a separate condition from which they suffer. To summarize, both, clear diagnostic criteria and seeing the person behind it with their strengths, are crucial for understanding and supporting autistic individuals. As later described in more detail, this helps with finding methods that make communication and showing empathy easier for autistic people (e.g. with the visualization of emotions).

ASD is genetically determined; however, the interaction and fit between an autistic person and their environment can have a major impact on their quality of life (Greaves-Lord et al., 2022).

Development of ASD

To understand how autistic structures are part of a person's being from the beginning, it is important to understand the development of ASD. In a paper by Courchesne et al. (2020, p.1) it is stated that "ASD is a highly heritable, multistage, multiprocess, progressive brain-wide disorder of prenatal and early postnatal development, with prenatal subtypes leading to clinical outcome heterogeneity". According to the publication, atypical changes in different stages of neural development such as cell migration and synaptogenesis are contributing factors to ASD. Recapping study findings about ASD risk genes, it is evident that they "are involved in excess cell proliferation, disrupted neurogenesis and maturation, mis-migration, disrupted synaptic development and function, and deviant neurofunctional activity and connectivity" (Courchesne et al., 2020, p.4). Taken from the paper, since atypically multiplying neurons are excitatory, one possible issue in autistic brains can be the imbalance between excitatory and inhibitory cells. Gene expression varies in quantity in two different prenatal phases and in the categories *regulatory* or *brain specific*; furthermore, the *hcASD* genes have an impact on certain processes (neurogenesis, migration, neurite outgrowth, wiring of networks, etc.) depending on the prenatal phase (Courchesne et al., 2020).

Neural correlates

Since in this project I was especially interested in the neural features (of empathy) in autistic individuals compared to non-autistic individuals, neural correlates of ASD are summarized in the following. Magnetic Resonance Image (MRI) studies have shown the anatomical differences described above concerning brain volume and gray and white matter between young autistic- and non-autistic children (Sungji et al., 2015). The researchers referred to Zielinski et al. (2014) who found that as autistic individuals get older, the cortex becomes thinner in various brain areas, so that cortical overgrowth in early developmental stages is followed by a drastic thinning. According to the review, ASD is in general defined by atypical connections in networks of the brain. The findings on regions associated with clinical characteristics of ASD are summed up below:

> abnormalities in (1) the inferior frontal gyrus (IFG, Broca's area), superior temporal sulcus (STS), and Wernicke's area might be related to defects in social language processing and social attention…, (2) the frontal lobe, superior temporal cortex, parietal cortex, and amygdala might mediate impairments of social behaviors…and (3) the orbitofrontal cortex (OFC) and caudate nucleus have been associated with RRBs [=restricted repetitive behaviors] of ASD… (Sungji et al., 2015, p.2)

Rahko et al. (2015) did a functional MRI (fMRI) study on working memory and attention in so-called ‘high-functioning’ adolescents diagnosed with and without ASD, since attention deficit often goes along as co-morbidity. On the behavioral level the researchers did not find significant differences between both groups when doing the attentional task. Looking at the brain regions, they found less activation in the right superior, middle and inferior gyri as well as in the right fusiform gyrus and the cerebellum in the ASD group during attentional processing. Rahko et al. (2015) found that the degree of deactivation in the default mode network during attention was similar between both groups, however the deactivation was more widespread in the ASD group in the regions described above. These results showing underlying differences in brain regions and even in whole brain networks between autistic and non-autistic individuals regarding attention could explain struggles with (emotion)

communication since this process requires a high level of attention for the counterpart.

Support instead of treatment - person-environment fit

Since this study tried contributing to more acceptance and well-being of autistic people by revealing misperceptions about emotion communication and empathic reactions and interviewing participants on their experiences, the next part emphasizes the importance of the person-environment fit. In a paper by Lai et al. (2020) possibilities for support and ways to improve the person-environment fit were discussed. The authors differentiated between two perspectives, the medical and the neurodiversity model: while the first is solely about disabilities and interventions, the second focuses on individual strengths, weaknesses and experiences of autistic people. Bringing these together into a dual perspective, allows researchers to address specific needs and to create an autism-friendly environment in an interdisciplinary way (Lai et al., 2020).

The researchers brought attention to deficient evidence and a lack of studies when it comes to treatments and call for an improvement in the quality of system-level groundwork. According to the paper, traditional early interventions like applied behavior analysis (ABA) show some effects on children's adaptive behavior (e.g. language development) but not on the main autistic features. A newer approach is less structured than ABA, called naturalistic developmental behavioral intervention (NDBI) but further testing for its effect is needed as well; in general, the strong involvement of a caregiver in these interventions is beneficial (Lai et al., 2020). Citing Lai et al. (2020), targeted interventions during childhood and youth like social skills training can improve social competence when dealing with peers.

As already mentioned before, there are methods to make communication with other people easier for autistic individuals: it is suggested that using specific pictures or sign language can help non- or minimally verbal children to improve language and express their needs. The method using these means of communication is called Augmentative and Alternative Communication (AAC) (National Autistic Society, 2023). In the current project this was considered by including a new drawing task in addition to the facial expression task. Several arguments spoke for an art-based task: concerning the social motivation hypothesis, stating that social stimuli are less rewarding and hence less processed in autistic people, it could be easier to show

emotions through other ways than facial expressions. For autistic individuals who have experienced communicating with pictures and abstract symbols this way of communicating emotions could be beneficial. Pelowski et al. (2020) did a study on emotion communication through visual art and found matching emotions between artist and viewer as well as from the artist intended emotions in the viewer. Additionally, they found a relationship between a component of trait cognitive empathy and emotional arousal when viewing the artworks. It may also be argued that drawing emotions in an abstract way requires even higher perspective taking processes than expressing emotions directly when thinking about how to paint the emotion so somebody else understands it.

One approach that targets the person-environment fit is the Treatment and Education of Autistic and related Communication-Handicapped Children (TEACCH), where the environment is structured considering the autistic person's strengths and challenges in close cooperation with the family (Lai et al., 2020). The authors stated that spreading awareness and knowledge among neurotypical individuals adds to the wellbeing of autistic individuals.

Draaisma (2009) explained in an article how stereotypes about autism are supported and consolidated through media like movies and books. Instead of showing how an autistic character is diagnosed and giving information about ASD, often solely "typically autistic" behavior and features of them are shown to the consumer (Draaisma, 2009). Looking at certain traits like restricted interests, according to the article, those are often represented as savant skills in media: autistic individuals are either shown cognitively handicapped or as super-intelligent in specific areas. The author states that in reality, savant skills are rare and even experts advising the producers/writers may contribute to stereotypes by leaving out such facts about ASD. Another stereotype is that autistic brains work like machines without being capable of producing real emotions (Draaisma, 2009). However, the author emphasized that apart from the differences, some media also show similarities with neurotypical individuals: for example, autistic traits in a non-autistic person like some obsessive behavior or routines during organizing. To improve the person-environment fit, it is important to decrease stereotypes and openly get to know an autistic person with the neurodiversity paradigm in mind.

Social cognition in ASD

Since this project focused especially on emotion communication, the following part is addressing emotion expression and recognition, as well as general social cognition in autistic people. Despite deeper brain structures could not be investigated in the current study, these are additionally discussed for background information about crucial social and emotional regions which are connected through networks to the cortical brain regions that were investigated. “Social cognition refers to processing that is elicited by, about, and directed towards other people” (Kennedy & Adolphs, 2012, p.1). Kennedy and Adolphs (2012) preferred a network view of brain regions involved in social cognitive tasks, introducing the Amygdala-, Mentalizing-, Empathy- and Mirror/Action-Perception network. They claimed that these networks constitute the social brain. As mentioned before, in ASD connections in the brain are altered, hence also conjunctions in the social brain, for example an atypical functional connection between amygdala and temporal cortex could be found (Kennedy & Adolphs, 2012).

Emotion communication in ASD

In a review by Fossati (2012) the focus laid on the connection between the social and emotional brain and their neural correlates. The author cited Phillips et al. (2003) summarizing the regions of the emotional brain: the medial and lateral prefrontal cortex, the anterior cingulate, the basal ganglia, the amygdala and the hippocampus. When processing faces, among other areas the amygdala is active, especially when it comes to fearful facial expressions (Fossati, 2012). Although often associated with negative emotions, the review offered evidence that the amygdala is also activated during positive emotions, so it was suggested that not the valence of emotion itself, but the amount of arousal triggers the amygdala (more arousal leads to more activation). Fossati (2012) stated that these cortico-limbic regions mentioned before are also involved in the processing of social stimuli, showing the overlap between social and emotional brain. In the paper, Adolphs (2010) was cited as suggesting that the amygdala reacts to social signals like faces and gaze direction; therefore, amygdala dysfunction can lead to impairments in social cognition. For example, differences in the perception of appropriate interpersonal distance in autistic individuals may be connected to the over-activation of amygdala. The medial prefrontal cortex (mPFC) is strongly connected with the amygdala and the dorsal

part of the mPFC is activated in highly social processes like mentally putting oneself in someone else's position and social interaction (Fossati, 2012). This shows how strongly cognitive and emotional processes are connected in the brain and how the impairment of one can lead to an impairment of the other.

Kleinhans et al. (2011) did a fMRI study on face processing in autistic adults. The authors suggested that atypical gaze-behavior is associated with differences in neural activation in the fusiform gyrus and the amygdala between autistic and non-autistic people when it comes to facial processing. Referring to what was mentioned before about the amygdala, the authors cited Dawson et al. (2005) who claimed that atypical activation of the amygdala could lead to atypical attention towards socially significant stimuli such as faces, as well as decreased social motivation in general. Kleinhans et al. (2011) investigated subcortical face processing networks in their study and found, as they predicted, less activation in the ASD group compared to the control group in the amygdala and other regions responsible for rapid face processing like the bilateral fusiform gyrus and the superior colliculi. This finding could be one mechanism explaining struggles in reading facial expressions in autistic individuals.

Another fMRI study by Richey et al. (2022) looked at the role of the anterior cingulate and the dorsolateral prefrontal cortex in facial emotion recognition. In their fMRI task, autistic male adolescents and adults participated with a matched control group. They used basic emotions for recognition and a machine learning approach to make predictions about brain activation. No significant difference at the behavioral level was found between groups (Richey et al., 2022). The results showed activation patterns predicting a correct decision in the facial emotion recognition task that included the anterior cingulate, the bilateral insula and the right dorsolateral prefrontal cortex. Activation in the anterior cingulate predicted correct decisions only in the control group while activation in the dorsolateral prefrontal cortex immediately before reflecting on a correct judgment predicted an incorrect response in the ASD group (Richey et al., 2022). They argued that the anterior cingulate cortex is crucial "for the integration of affect (…)" (Richey et al. 2022, p.14). Therefore, it can be reasoned that altered function of this region, as well as in the dorsolateral prefrontal cortex, is contributing to different emotion recognition processes in autistic individuals and therefore influencing social interactions and own emotional reactions.

Wieckowski and White (2017) did a study about difficulties with emotion recognition and expression in autistic people and specifically analyzed eye-gaze. The researchers cited several studies that autistic individuals struggle to recognize both basic and more subtle emotions from early childhood on (e.g. Lindner & Rosén, 2006; Humphreys, Minshew, Leonard, & Behrmann, 2007). When it comes to emotion expression, they listed different findings: autistic individuals show less nonverbal emotional expressions and those are often perceived as inappropriate or flat; in addition, they have difficulties to imitate and show instructed emotional expressions (e.g. Yirmiya, Kasari, Sigman, & Mundy, 1989; Loveland, Tunali-Kotoski, Pearson, Brelsford, Ortegon & Chen, 1994). Therefore, impairments in emotion recognition as well as in expression, have severe influence on social relationships. Referring to eye-tracking studies, Wieckowski and White (2017) summarized their results: autistic people tend to show less attention to the eyes in social interaction and rather focus on the mouth or non-social objects around, however study findings are not consistent (e.g. Dalton et al., 2005). In their study they used eye-tracking to investigate emotion communication in autistic adolescents in comparison to a neurotypical control group. With the help of videos showing and instructing facial expressions, the authors found out that, in contrast to their hypothesis, there was no difference in the amount of eye fixation between the groups and no effect of focusing the eye region for emotion recognition accuracy. In general, emotion recognition ability did not differ significantly but only for the emotions disgust and sadness in favor of the control group (Wieckowski & White, 2017). The study could find that the ASD group imitated emotions less and less accurately than the control group in the expression task, but only in the free-choice response task, not when they were instructed. According to the paper, this result is a possible explanation for challenges of autistic people in natural social situations when it comes to mirroring emotions of the counterpart.

Eack et al. (2014) conducted a study which included basic emotions and neutral expressions to find out more about patterns of emotional misinterpretation in autistic adults. Their results showed that the ASD group was significantly worse in identifying emotional expressions than the control group, especially when it came to happy, neutral and sad faces. The authors found that autistic participants more frequently interpreted happy faces as neutral and confused sad ones with fearful ones. Also, the ASD group had more difficulties recognizing neutral expressions,

they were often confused with angry ones and actual sad faces were frequently labeled as neutral ones (Eack et al., 2014).

In a review by Keating and Cook (2020) findings about emotion expression in autistic individuals were summarized: although there is no difference in the intensity of the shown emotion compared to neurotypicals, their emotions are visually different and lower in quality (or less accurate) during mimicry. In addition, they show emotional expressions generally less and for a shorter time (Keating & Cook, 2020). Concerning emotion recognition, the authors emphasized the various study findings, several of them giving strong evidence for impairments in emotion recognition in ASD and others that did not detect significant differences to neurotypical individuals. They argued that those contradicting findings can be caused through differences in task stimuli, for example regarding intensity and static vs. dynamic stimuli. Furthermore, they added that success in emotion recognition depends partly on the participant's characteristics (age, intelligence, comorbidities). Lastly, Keating and Cook (2020) mentioned the difficulty for neurotypical people to read facial expressions of autistic people and how this mutual misunderstanding consolidates struggles in social interactions.

Empathy

Definition and related concepts

There are several definitions for empathy and many additional concepts, such as emotional contagion, are related to it (Singer & Lamm, 2009). Referring to Batson and Ahmad (2009), empathy consists of different psychological states: imagine-self perspective, imagine-other perspective, emotion matching and empathic concern. Imagine-self perspective refers to the ability to imagine oneself in another's situation, imagine-other perspective refers to the ability to comprehend how another person feels in that situation, emotion matching is the ability to feel what the other person feels, and empathic concern is the ability to feel for that person.

In the literature, cognitive and affective empathy are often considered as distinct concepts. Kerr-Gaffney et al. (2020, p.2) cited Blair (2005) that "cognitive empathy refers to the ability to recognize and understand the mental states of others (overlapping with the concept of ToM [Theory of Mind]); affective empathy is the ability to share the feelings of others, without any direct emotional stimulation to oneself". In a meta-analysis of Fatima and Babu (2023) it was stated that cognitive

empathy comprises perspective taking, emotion recognition and empathic accuracy in addition to ToM.

ToM or mentalizing describes the “ability to explain and predict the behavior of ourselves and others by attributing to them independent mental states, such as beliefs, desires, emotions or intentions” (Gallagher & Frith, 2003, p.1).

Another process strongly connected with empathy is mimicry (Prochazkova & Kret, 2017). “Automatic mimicry is defined as the unconscious or automatic imitation of speech and movements, gestures, facial expressions and eye gaze” (Prochazkova & Kret, 2017, p.1). The researchers explained that if this leads to the same affective state as in the opposite person, it is called emotional contagion. They referred to Preston and de Waal (2002) claiming that emotion contagion is the most basic form of empathy. The authors stated that there are two kinds of mimicry: motor mimicry like facial expressions or laughing and autonomic mimicry, meaning synchrony between physiological parameters like heart rate or hormone release. In their neurocognitive model of emotional contagion (NMEC), coupling of the mirror neuron system is crucial for motor mimicry and emotion system coupling is crucial for autonomic mimicry between sender and receiver.

Development of empathy

In a review by Heyes (2018) it was mentioned that empathy is widely seen as automatic and innate as it is believed to have evolutionary benefits. However, the Learned Matching hypothesis claims that empathy can be developed also through associative learning (Heyes, 2018). The author explained this by matching emotional associations, happening during social interaction, meaning the connection between an observed motoric or somatic response and the affective state. These matching emotional associations again are produced by showing the same emotion simultaneously and when caregivers mirror the emotion of infants (e.g. matching facial expressions) (Heyes, 2018). In the review it was stated that learning is important for the genesis of mirror neurons which - as mentioned before - play a role in empathy. However, Heyes (2018) acknowledged the combination of native social cognitive processes and nurtured ones leading to empathy and related concepts.

State- and trait empathy

Empathy can be assessed as a personality trait or as reaction to a specific situation: “Trait empathy is the general ability to show empathy, whereas state empathy is the transient affective reaction elicited in concrete situations” (Van der Graaff et al., 2016, p.2). It would be logical that a positive correlation exists between these two, however, study results are inconsistent. Van der Graaf et al. (2016) investigated the relationships between motor (mimicry)-, affective- and cognitive empathy and trait and state empathy with 379 adolescents. They also looked at the emotions happiness and sadness separately. The authors found significant positive correlations between motor- and affective (state) empathy and motor- and cognitive (state) empathy mediated by affective (state) empathy for both emotions. Additionally, they found more robust associations between trait and state empathy regarding the emotion sadness. Their calculated relations were all modest and suggest a relationship between trait and state empathy at least to some extent.

Neural correlates

The mirror neuron system in humans is suggested to be one of the fundamental neural mechanisms for empathic reactions; it consists of the rostral inferior parietal lobule, the caudal sector of the inferior frontal gyrus and parts of the premotor cortex (Decety & Lamm, 2006). According to the paper by Decety and Lamm (2006) this network is active when observing actions of others that trigger motor representations in the self and is relevant for empathic processes like mimicry. The authors summarized evidence that this mechanism applies not only for motoric perceptions but for emotional expressions as well: viewing an emotion on somebody’s face can activate the same brain regions as experiencing this emotion. Decety and Meyer (2008) referred to Preston and de Waal (2002) who summarized these mechanisms into the perception-action-model claiming shared neural representations functioning as bottom-up basis for empathic reactions.

Shamay-Tsoory (2010) stated that affective empathy requires the ability to recognize emotions and includes emotional contagion and shared pain. Referring to the paper, again, the mirror neuron system is responsible for emotional contagion, especially the inferior frontal gyrus which also plays an important role in emotion recognition. To conclude, specifically the right inferior frontal gyrus is an important region for affective empathy since it shows increased activation during compassion

(Simon-Thomas et al., 2011), is involved in facial mimicry (Hsu et al., 2018) as well as in pain-related empathy (Li et al., 2021).

When it comes to ToM, parts of the frontal cortex are involved during perspective taking (Decety & Lamm, 2006). In the review, a functional neuroimaging study was cited that found that those parts are the frontopolar cortex, the ventromedial prefrontal cortex, the medial prefrontal cortex, and the right inferior parietal lobule, independent of the emotional content. When including the emotional level, areas for emotion processing were activated additionally (e.g. amygdala and temporal poles) (Decety & Lamm, 2006). The right dorsolateral prefrontal cortex is another crucial region in addition to the mPFC since it is activated during facial emotion recognition and is also involved in mentalizing (Hétu et al., 2012).

Emotion communication and empathy

The following section describes how empathy is involved in emotion communication, i.e. emotion recognition and expression. Holland et al. (2020) did a meta-analysis investigating the correlations between mimicry, empathy and emotion recognition. They included studies where trait and state empathy were assessed with questionnaires, and found a significant moderate positive effect between facial mimicry and overall empathy. However, the authors could not find a significant effect between mimicry and facial emotion recognition accuracy. Moderator analyses showed that the correlation between mimicry and empathy was only significant for the emotions anger, fear, happiness and sadness, but not for pain or disgust (Holland et al., 2020). When they looked at affective and cognitive empathy separately, they were both significantly influencing the correlation between mimicry and overall empathy. The researchers could report the same for separately examining the influence of trait and state empathy on this relationship.

Besel and Yuille (2010) investigated how individual levels of cognitive empathy are related with facial emotion recognition. In their study, 135 participants tried to recognize the emotions anger, sadness, happiness, disgust, surprise, fear and neutral, as well as fill out The Empathy Quotient (EQ) (Besel & Yuille, 2010). In addition, the researchers used the subscale *Empathic Concern* of the Interpersonal Reactivity Scale to measure affective empathy. For the shorter exposure time of the facial expressions they found that empathic concern was stronger associated with emotion recognition accuracy than the EQ. However, for the longer exposure time,

the EQ was significantly correlated with emotion recognition accuracy and empathic concern was not (Besel & Yuille, 2010). The researchers had their focus on the expression fear; contrary to their hypothesis, the EQ was connected to difficulties with recognizing fear, but empathic concern was not. They explained this due to individual emotional personality traits when it comes to emotional reactivity to fearful expressions.

In a study by Svetieva and Frank (2015) the relationship between trait empathy and recognition of micro expressions was examined. They used natural micro expression stimuli through videos and the Micro expression test (METT) including the emotions sadness, anger, fear, happiness and disgust. They assessed empathy with the Basic Empathy Scale (BES) measuring both cognitive and affective empathy. The authors found that in general expressions were easier to recognize in the standardized test than from natural stimuli (videos). Overall, they were not able to find a significant correlation between empathy and micro expression recognition accuracy in both conditions. Only affective empathy was positively associated with recognizing the emotion anger in the videos (Svetieva & Frank, 2015).

Gary et al. (2016) looked at the individual factors underlying emotion recognition abilities. They used static face expression stimuli, again with the emotions anger, disgust, fear, happiness and sadness. Since one of the factors was (trait) empathy, this was measured with the Interpersonal Reactivity Index (Gary et al., 2016). The authors found a significant positive correlation between empathy and emotion recognition ability, but still a small effect.

Zaki et al. (2009) did a fMRI study on empathic accuracy (EA) testing which brain regions were activated when trying to understand another person's emotions. Specifically, they looked at brain areas related to *shared representations* and *mental state attributions* which are described in the following. The researchers investigated if participants rated the emotional state (positive to negative) of a target in a video correctly, comparing it with the own rating of the target person in the video. EA was predicted through activation in the dorsal and rostral subregions of the mPFC and in the superior temporal sulcus (STS), which are regions involved in mental state attributions (Zaki et al.,2009). At the same time, they found activation in the mirror neuron system (involved in shared representations) predicting EA: in the right inferior parietal lobule (IPL) and the bilateral dorsal premotor cortex (PMC).

Taken together, the collected evidence suggests that empathic processes play a crucial role in facial expression recognition, however, study results for the association between individual levels of empathy and emotion recognition accuracy are mixed or show only small effects. Because my project focused on the connection between emotion expression and empathy, building a bridge between those is the content of the next section.

As already mentioned, mimicry and emotion contagion can be seen as subcomponents of empathy. Sonnby-Borgström (2002) did an electromyography (EMG) study measuring facial reactions as well as assessing affective empathy. She divided participants into a low-empathy group and a high-empathy group and examined differences in mimicry to various emotional expressions. The high-empathy group showed more automatic mimicry than the low-empathy group, while the latter showed more smiling tendencies when reacting to an angry face (Sonnby-Borgström, 2002).

Nummenmaa et al. (2008) did a fMRI experiment testing if there is a difference between affective and cognitive empathy when it comes to emotional contagion. The female participants looked at emotional (somebody gets attacked) or unemotional (somebody having a conversation) pictures and were instructed to empathize with certain people or just watch them (Nummenmaa et al., 2008). The researchers assessed the emotional experience of the participants with self-ratings (fear, anger, disgust, sadness, surprise, pleasure, neutral). When they compared neural reactions to stimuli triggering affective (emotional content) or cognitive (unemotional content) empathy, they saw that affective empathy activated more and different brain regions than cognitive empathy, mainly the regions of the mirror neuron system, which are areas responsible for emotion processing, perspective taking, and understanding body language.

In summary, literature has shown that there is a link between expressing emotions and empathy, at least when it comes to mimicry and emotional contagion. According to a review by Mitchell and Phillips (2015), emotion perception and ToM share, to some extent, the same neural activation patterns, namely the amygdala, the medial PFC and parts of the temporal lobe. In the studies above, findings were always about the perceiver understanding emotional states in another person (e.g. pictures of facial expressions, actors in videos presenting emotions). It is logical to reason that processes of empathy like ToM are not solely happening during

observing but as well as during showing emotions to somebody else. Especially in study settings, in a task where someone has to think how to depict a certain emotion so that somebody else is able to recognize it, it makes sense that similar processes like ToM can be found. The person needs to adapt the perspective of the viewer and has to consider how to express an emotion clearly to recognize it from their perspective.

Autism and Empathy

Because this project focused on autistic people and empathy, the following part summarizes study findings about autism and empathy in general and in the connection with emotion recognition and expression.

The double empathy problem

One important concept is the double empathy problem introduced by Milton (2012). Basically, this posits a miscommunication between two people with different dispositions, like autistic and non-autistic, due to struggles to understand the other person's intentions and meaning of what they communicated. This goes both ways, not only does the autistic person have problems understanding the non-autistic person leading to the described impairments in social interaction, but also the non-autistic person cannot figure out what the autistic person tries to communicate (Milton, 2012). Summarizing this, there is not one way of 'normal' communication, but autistic and neurotypical people just communicate differently with diverse underlying processes. In a follow-up paper by Milton et al. (2022) it was explained that one reason for miscommunication and problems to empathize is the discrimination of experiences that autistic and non-autistic people have made in their lives, e.g. altered sensory perceptions. A lack in ToM in both communication partners can be seen as the cognitive basis for the double empathy problem (Bolis et al., 2017; Mitchell et al., 2021). According to Fletcher-Watson and Bird (2019) the link between decreased empathy and autism in research exists because ToM is often seen as cognitive empathy instead of only one part of cognitive empathy.

Impairments of Theory of Mind in ASD

In a review by Senju (2012), studies were summarized showing evidence that the development of ToM is indeed atypical in autistic children, as tested with the false belief test. However, since the test is cognitively demanding, autistic children

with better verbal skills do pass the test (Senju, 2012). On the other hand, the author stated that ToM consists of more components than just passing the false belief test, such as identifying mental states from photos of eyes, with which even 'high - functioning' autistics struggle (note: the term high-functioning will be discussed in the part *Discussion*). According to the researcher, when comparing adults with Asperger (one 'high-functioning' form of ASD) to neurotypical adults, they all passed the standard false belief test, but the Asperger group was significantly worse in the spontaneous false belief test. The standard false belief test is passed when the participant guesses for the original position of an object that has been moved in the absence of a protagonist who, after that, looks for the object (Senju, 2012). In a spontaneous false belief test, no specific instructions are given, and it can be more interactive; in the current study the researcher used video stimuli depicting the object-change-position situation and eyetracking to examine spontaneous gaze movements. The finding above is congruent with real-life problems in social interactions of autistic individuals (Senju, 2012). Not only verbal skills influence the ability for ToM, but also the level of executive functioning, which is atypical in autistic people (Kimhi, 2014). Kimhi (2014) cited Mathersul et al. (2013) who found that advanced ToM abilities such as understanding sarcasm and deception were shown less by 'high-functioning' autistics compared to non-clinical controls. However, the author stated that study results testing ToM in ASD were mixed depending on the task design and that especially 'high-functioning' autistics can compensate reduced ToM through structured social interactions and learning with experience.

In a recent research paper from 2024, Cheang et al. did a study on the double empathy problem testing the empathic accuracy of non-autistic people. Participants who were non-autistic according to the Autism-Spectrum-Quotient (AQ), had to watch several videos where either autistic or non-autistic individuals tell autobiographical situations presenting the emotions anger, fear, happiness, sadness or neutral (Cheang et al., 2024). The study instructed participants to rate the intensity of the emotions and to identify them (= empathic accuracy). The researchers found that empathic accuracy was less regarding autistic narrators compared to non-autistic narrators, especially when it came to the emotions happiness and sadness. On the other hand, the felt intensity of emotions was higher regarding autistic narrators compared to non-autistic, especially with anger and fear (Cheang et al., 2024). With this study the authors could show that neurotypical people indeed

struggle more to read emotional states of autistic people, supporting the double empathy problem.

Baron-Cohen et al. (2001) used his ‘Reading the Mind in the Eyes’ Test on ‘high-functioning’ autistic individuals and adults with Asperger and non-clinical adult control groups measuring advanced ToM. The test in general shows several photos of eyes and participants have to describe in two words what the person in the picture is thinking or feeling (Baron-Cohen et al., 2001). As predicted, they discovered a significantly worse performance in assessing mental states in the autistic group. They could also see that in the control groups, females were generally more accurate than males. Scores of the AQ were as expected negatively correlated with performance in the ‘Reading the Mind in the Eyes’ Test (Baron-Cohen et al., 2001).

Cognitive vs. affective empathy in ASD

Since the current study looked at brain regions associated with cognitive- as well as affective empathy, study findings about these two kinds of empathy are summarized in the following part. McKenzie et al. (2022) investigated empathic accuracy and cognitive and affective empathy in young autistic adults compared to neurotypicals. They used the Interpersonal Reactivity Index (IRI) to measure trait empathy, as well as the Empathic Accuracy Task. McKenzie et al. (2022) operationalized cognitive empathy as the number of correctly identified emotions and affective empathy as the match between the felt emotion by the participant and the shown emotion in the stimuli. Looking at the IRI scores, they found that the ASD group had significantly lower scores on the subscale *perspective taking* than the control group as well as on the subscale *empathic concern*, measuring affective empathy. In contrast, in the experimental task, they could not find significant differences between the autistic and the non-autistic group: overall they had similar scores in correctly identified emotions (cognitive empathy) and matches of felt emotions (affective empathy). Only concerning anger, empathic accuracy was less for the ASD group (McKenzie et al., 2022).

Shirayama et al. (2022) tested associations between personality traits, autistic traits and cognitive and affective empathy in autistic adults. Relevant methods they used were the AQ assessing autistic traits, the Questionnaire of Cognitive and Affective Empathy (QCAE) and the IRI as an additional tool to assess empathy. In comparison with a non-autistic control group, the researchers found out that the ASD

group scored significantly lower on the subscales *perspective taking* and *online simulation* (both cognitive) and *peripheral responsivity* (affective) of the QCAE. They did not find any significant differences for *emotion contagion* and *proximal responsivity* (both affective). Calculating the subscales together and comparing only cognitive and affective empathy, the ASD group had significantly lower cognitive empathy scores while affective empathy scores were similar to those of the control group (Shirayama et al., 2022). Analyzing the IRI, the authors found that the autistic group scored lower on the subscales *perspective taking* (cognitive) and *empathic concern* (affective) but not on *personal distress* (affective) and *fantasy* (cognitive) compared to the controls. Total AQ scores correlated significantly negative with cognitive empathy (Shirayama et al., 2022).

Furthermore, a meta-analysis by Song et al. (2019) showed that trait-cognitive empathy, trait-empathic concern, state-cognitive empathy and state-empathic concern were atypical in ASD compared to neurotypical individuals. However, the researchers could also see that empathic accuracy (i.e. correctly identifying the emotion felt by another person) was similar or even higher in autistic individuals. They investigated possible factors moderating these results and found significant effects for gender in favor of females and for age in a way that with higher age, impairments in affective empathy are compensated (but not in cognitive empathy).

Fatima and Babu (2023) investigated in another meta-analysis cognitive and affective empathy in autistic people. They found a huge positive effect for cognitive empathy, i.e. the control group scored significantly higher in cognitive empathy than the ASD group. For affective empathy, they detected the same but with a medium positive effect. When looking at potential influencing factors on these findings, the researchers found no significant moderation of age or culture. They were able to find a significant effect for the measure used to assess empathy, but only for cognitive empathy, not for affective.

Neuropsychological findings

Contributing to findings from self-report measurements and behavioral tasks, the question arises how brain regions for empathy differ in autistic individuals compared to typically developed ones. A review by Harmsen (2019) suggested that certain brain regions including those involved in empathy are smaller in autistic individuals than in neurotypicals; these are the anterior cingulate, the superior

temporal gyrus and the prefrontal cortex. ‘High functioning’ autistics score higher in empathy due to the influence of better executive functions and increased vocabulary (Harmsen, 2019). As mentioned earlier, autism goes along with altered brain connectivity. The author cited Hoffmann et al. (2016) who found out that reduced connectivity is associated with deficits in ToM. Harmsen (2019) summarized evidence about the *broken mirror hypothesis* claiming that dysfunctions in the mirror neuron system lead to impaired mimicry and thus empathy deficits. The review stated that results for this hypothesis are mixed and rather suggests that the system for basic mimicry is actually intact in autistic individuals. The *social motivation hypothesis of autism* says that the reward system works differently in autistic people in a way that social marks like eye contact or facial expressions are less rewarding and hence are less processed (Harmsen, 2019). According to the paper, there were several results speaking for this hypothesis, and it could add to difficulties in reading facial emotions.

In a research article by Schulte-Rüther et al. (2013), age-dependent changes of empathic brain areas in ASD were discussed. They did a fMRI study during which participants were presented with various emotional facial expressions and were instructed either to match and judge the emotion on the picture or to report their own felt emotions when looking at it (other- vs. self-task). When comparing the autistic group to the non-autistic group looking at the behavioral level, age was not interacting with the results in the self-task but group membership was: the control group gave significantly more congruent answers than the ASD group (Schulte-Rüther et al., 2013). In the other-task they found neither a significant age effect, nor a group effect, meaning both groups performed as well. When they looked at the neural level, the age group (children, adolescents, adults) interacted significantly with the results only in the self-task. They found different activation patterns influenced by age in the two groups in the right dorsolateral prefrontal cortex, the right medial prefrontal cortex, the right inferior parietal cortex, the right anterior insula and the occipital cortex. While in the control group activation in those areas generally decreased with higher age, in the ASD group, activation increased or stayed the same with higher age (Schulte-Rüther et al., 2013). The authors argued that decreased neural activation in certain areas of neurotypicals is a sign of being able to cognitively control activation dependent on a specific task. On the other hand, they

speculated that increasing brain activation in ASD depending on age could be a sign of developing compensatory mechanisms.

Komeda et al. (2014) aimed to investigate empathy mechanisms between autistic individuals. They split participants into an ASD group and a neurotypical control group; in a fMRI scanner they were then confronted with statements either matching autistic- or non-autistic characteristics and participants had to indicate if they agree with them (yes/no). Additionally, statements were either from a self-perspective or about another person and the researchers chose to look at the activation of the ventromedial prefrontal cortex (vmPFC) as a region of interest. On the behavioral level, they saw a significant interaction between group membership and character type. The ASD group agreed with autistic traits statements significantly more than the control group, while the control group agreed more on non-autistic statements (Komeda et al., 2014). Komeda et al. (2014) showed that participants generally agreed faster on self-statements than other-statements for both autistic- and non-autistic traits. When they looked at brain activation, for both groups the same areas were activated when the ASD group rated autistic traits statements and the control group rated non-autistic traits statements. These were the inferior frontal gyrus (IFG), postcentral gyrus, paracentral lobule, precuneus, cuneus, lingual gyrus, cerebellum, fusiform and superior frontal gyrus and the vmPFC (Komeda et al., 2014). The authors found different functional connectivity patterns between the ASD and the control group: activation in the vmPFC in autistic individuals went along with activation in other brain structures than in typically developing controls. These results showed that autistic individuals are able to empathize with other autistic individuals, even though brain networks for that differ from those of neurotypical individuals (Komeda et al., 2014). Furthermore, this study gave insight into the neural correlates of the double empathy problem.

More on the affective side of empathy, Hadjikhani et al. (2014) did a fMRI study about emotional contagion for pain in ASD. They compared a ‘high functioning’ adult ASD group to a control group while showing them videos of painful facial expressions. Since the researchers were interested in implicit empathic reactions, there were no specific tasks like rating the emotions etc. When the authors looked at the scores of the Empathy Quotient (EQ), filled out beforehand, they found significantly lower scores in the ASD group in total and on the subscales *cognitive* and *social EQ*. The fMRI data revealed that in both groups the same areas

responsible for emotional processing and emotional attribution were activated (amongst others): the orbito-frontal cortex and the amygdala, the inferior frontal gyrus, the temporoparietal junction (TPJ) and the STS (Hadjikhani et al., 2014). They found some regions additionally activated that differed between the ASD and control group, e.g. the premotor cortex in the ASD group and the mPFC in the control group. All in all, the researchers could not detect any significant differences in brain activation between the groups during the perception of painful faces, showing that autistic individuals are generally capable of experiencing affective empathy and emotional contagion.

Social cognition, emotion recognition and empathy

Sugranyes et al. (2011) conducted a meta-analysis about the neural correlates of social cognition in ASD, specifically about ToM and emotion recognition. They included fMRI studies with autistic participants and matched controls doing either a ToM or a facial emotion recognition (FER) task. Sugranyes et al. (2011) found in the FER tasks that in comparison to non-autistic people, autistic people showed significantly higher activation in temporal areas close to the STS and less activation in the primary somatosensory cortex of the postcentral gyrus. Regarding ToM task results, they found that autistic individuals showed significantly less activation than controls in “six clusters located in the medial prefrontal and in the anterior cingulate cortex, the amygdala and in primary and secondary somatosensory areas in the precentral gyrus, the STS, and in the TPJ within the inferior parietal lobule” (Sugranyes et al., 2011, p.7). Summarizing this, regions for mentalizing (mPFC), mirroring (somatosensory cortices) and processing social information like emotions (amygdala, TPJ) were atypical in ASD. The authors explained the overactivation in autistic participants during the FER tasks in regions around the STS due to more logical than social processing of faces, since this area is primarily involved in memorizing and identifying faces not in processing emotional states.

Sucksmith et al. (2013) investigated the relationship between emotion recognition and empathy in autistic people. Their study included autistic adults and a non-autistic control group who filled out the EQ and did a facial emotion recognition task showing several photos of the six basic emotions and neutral expressions from which they had to choose the right emotion. The researchers found significant group

differences for the EQ, with lower scores in the autistic group and generally higher scores in females than males, but only in the control group. Looking at emotion recognition scores, they saw that the ASD group was significantly less accurate than the control group in rating happiness, anger, fear, disgust and neutral faces. In addition, they could see that autistic participants had overall higher response times than non-autistic participants. When correlating EQ scores with the results of the emotion recognition task, they found a significant negative correlation between EQ scores and accuracy-adjusted response time in the FER task, indicating that lower empathy goes along with lower emotion recognition skills.

Summary of the literature review

Different processes of social cognition in autistic individuals compared to neurotypicals are connected to altered connections within brain networks. Although autistic people process faces and emotional expressions differently, findings about the general level of emotion recognition abilities are mixed. When it comes to emotion expression, some studies show that autistic people express less nonverbal emotions or they do it in a seemingly inappropriate way and that they have difficulties with mimicry. Literature suggests strong links between emotion recognition, cognitive empathy and ToM. However, direct correlations between emotion recognition abilities and empathy investigated in studies show rather small effects if any. Looking at emotion expression and empathy, there is some evidence that there is a relationship between the level of empathy and mimicry/emotional contagion. Reflecting on the literature review, it can be reasoned that ToM as a part of cognitive empathy not only plays a role in emotion perception but also in emotion expression. Probably similar mentalizing processes are happening when a person is thinking about how to show an emotion in a way that the other person can clearly recognize it; perspective-taking is needed when imagining how the other person will perceive a certain emotion.

A lack of ToM in autistic and non-autistic social interactions can cause misunderstandings, called the double empathy problem. There is strong evidence that cognitive empathy and ToM are somehow reduced in ASD. Findings for affective empathy are more mixed: one meta-analysis found a significant medium effect while another neuropsychological study found that mechanisms for affective empathy and emotional contagion are not significantly different between autistic and non-autistic

participants. Those empathic impairments can manifest also in autistic brains, e.g. through smaller sizes of empathic areas. Including all of the concepts above, there are study findings showing correlations between ASD and decreased emotion recognition abilities and empathy.

Based on the literature and the theoretical approach connecting emotion expression and empathic processes, my project aimed to answer the following research questions:

RQ1: Is there a difference between autistic and non-autistic people regarding activation in empathy-related brain areas during emotion expression?

RQ2: Is there a difference between autistic and non-autistic people regarding the subjective self-rating of thinking of others? (note: this refers to a measurement of empathic reactions and is explained in the *Methods* section)

RQ3: Is there a correlation between subjective self-rating of thinking of others and activation in empathy-related brain regions?

In response to the mixed study results about altered levels of empathy (especially regarding affective empathy) and therefore neural differences in autistic people, the first hypothesis is non-directional and does not specify in which way brain regions would differ in their activation. Since literature does suggest lower levels of cognitive empathy in autistic people and the self-rating of thinking of others measured in the direction of cognitive components, the second hypothesis is directional. Hence, these were my hypotheses that emerged from the theoretical background and were investigated in the following study:

H1: Autistic participants show a different activation in cortical brain areas associated with cognitive and affective empathy than non-autistic participants during the expression of emotions.

H2: Autistic participants have lower scores in the subjective self-rating of thinking of others than non-autistic participants during the expression of emotions.

H3: There is a positive correlation between subjective self-rating of thinking of others and activation in the empathy-related cortical brain regions.

Methods

Study plan and design

The study was part of a larger funded project running over several years on autism and emotion communication. The whole project consists of expression and perception studies (of emotions) and my research took place only in the expression part. Participants with and without diagnosis of autism were recruited and were assigned to the autistic or non-autistic (NA) group. Since there was no randomization, it was a quasi-experimental, cross-sectional, mixed design (group as between-subject factor and test modality as within-subject factor). The presence of an ASD diagnosis, hence group membership, functioned as the independent variable. The study took place in a lab of the psychological faculty at the University of Vienna. All participants went through an art-based, as well as a facial expression task of emotion production while activation of empathy-related brain areas was recorded which was the dependent variable. Additionally, a questionnaire regarding the tasks (including questions like how much the participant thought of the other person who will try to recognize the emotion) was correlated with empathic activation to compare subjective evaluation with neuronal activation.

Participants

A power analysis was performed with GPower. Assuming a power of 0.8 (note: values were adopted from the study plan concerning the whole project, since I had to work with the corresponding sample), alpha of 0.05 and a medium effect size (f=0.25), the sample size required to detect a significant interaction between group and modality was 34 (17 per group). We decided to recruit 30 per group to increase power and account for unusable data.

Participants were recruited through a convenient sampling with online advertisement on social media and the Study Participant Platform of the Vienna Cognitive Science Hub. Also, people were contacted through an existing email list of autistic participants. Potential participants completed an online prescreening on Qualtrics. The prescreening survey collected demographic data including age, gender, presence of autism diagnosis to validate group membership, and presence of other neurological or psychiatric diagnosis. Inclusion criteria were age between 18 and 65, fluency in German and normal to corrected to normal vision. Exclusion criteria were regular use of illegal drugs, presence of intellectual disability, and sensitivity on the

scalp (e.g. eczema). For the autistic group, a clinical diagnosis of ASD was required, and for the non-autistic group, a score below 17 on the AQ-k (Freitag et al., 2007) and the absence of any psychiatric or neurological diagnosis were required. Taking part in the study was compensated with 30€. People joining the study signed an informed consent form describing the aims and the procedure of the study and were told that they can withdraw their data any time by emailing.
Members of the autistic and non-autistic group were age- and gender-matched.

From initially 62 people joining the study, one person and her match were excluded from the data because of a missing diagnosis document. The actual sample consisted of *n* = 60 participants (33 women, 17 men, 10 non-binary/non-conforming participants). There were matching pairs of autistic and non-autistic participants, forming two groups with *n* = 30. The mean age was 29.7 years (*SD* = 10.43, *mode* = 24) with the youngest person being 18 and the oldest person being 65 years old. Reporting levels of education, four people had a compulsory school certificate, three had a vocational training, 21 graduated from high school, 20 had a bachelor degree, six had a master degree, one had a MBA and two had other university degrees. Two participants had a PhD and one preferred not to answer. Most participants were Austrian (*n* = 40), eight were German and 12 indicated other nationalities.

Measurements

The expression of emotions was operationalized by a facial expression task and a novel art paradigm. In the **facial expression task** created by the study team the participant was instructed to show a certain emotion (happiness, sadness, anger or fear) while being filmed by a camera in front of them. Participants were told that another group in the future will try to guess which emotion they are expressing. The task included a baseline (looking towards the camera) and a practice round in the beginning with a neutral emotion (surprise) to avoid priming effects. After that, there were four rounds showing the emotions in random order with questions after each round to assess how the person felt during the task and how they would rate their expression. One of the questions was “How much did you think about the viewer while making the expression?” This question on a 7-point Likert scale (“not at all” to “very much”) should assess empathy in the form of a subjective rating. A high rating indicates higher empathy. Since this question was about the amount of thinking of

others (not feeling with another person) it rather measured components of cognitive empathy.

The **art paradigm** was also created by the study team, and it has been the first time that emotion communication of autistic adults was investigated in such a way. Participants had to make abstract drawings on a tablet depicting either happiness, sadness, anger or fear while the screen was being recorded, and hand movements were filmed. The task consisted of a baseline (holding the pen in a drawing position) and a practice round to get familiar with the drawing and then the actual four emotions in random order. Participants were instructed that drawings must not show representational objects (faces, landscapes, or symbols such as smiley face) and could not be erased. Pen color was black. The same question as in the facial expression task about thinking of the viewer was included in between each round to assess a subjective rating of empathy ("How much did you think about the viewer while drawing?").

Activation of empathy-related brain regions was measured with **functional Near-infrared Spectroscopy (fNIRS)**. This neuroimaging method was discovered by Jobsis in 1977 and allows researchers to track brain activity through blood flow (Li et al., 2022). With a light source that reaches the cortex, the hemodynamic responses, meaning concentration changes of oxygenated hemoglobin and deoxygenated hemoglobin, are measured (Li et al., 2022). propagated light of different wavelengths that is able to go through cortical tissue, hits optical detectors which are as well as the sources placed on the scalp (Li et al., 2022). Benefits of fNIRS are that it is non-invasive and allows the person to move freely instead of having to lie down in a tube, although only cortical regions can be measured since the light cannot penetrate deep enough for deeper structures (Li et al., 2022). The temporal resolution is higher than in fMRI but lower than in Electroencephalography (EEG) (Li et al., 2022). To check the fitting of the optodes on the scalp, a 3D digitizer (Polhemus Patriot) was used to digitize optode locations and head landmarks. We used NIRSport2 devices by NIRx with a 8x8 system, which includes eight sources and eight detectors measuring brain activity over short distance channels. For the caps, standard EEG 10-5 system caps were used. Optodes were placed based on head landmarks, meaning the nasion (Nz), the inion (Iz), and the left and right pre-auricular points (LPA and RPA) (Oostenveld & Praamstra, 2001).

Questionnaires assessing autistic traits and empathy were provided in German. The prescreening included a short form of the **Autism Spectrum Quotient (AQ-k)** which has 33 items (Freitag et al., 2007). Freitag et al. (2007) evaluated and translated the AQ into German for the first time. They calculated three factors (subscales): first, *Social Interaction and Spontaneity*, second, *Fantasy and Imagination* and third, *Communication and Reciprocity*. According to the researchers the items are based on cognitive symptoms of ASD. One example item is “In sozialen Situationen fühle ich mich wohl.” with the four response options „ich stimme eindeutig zu“, „ich stimme ein wenig zu“, „ich stimme eher nicht zu“ and „ich stimme überhaupt nicht zu“ (Freitag et al., 2007; psydix.org). They stated that the self-report questionnaire is suitable for people aged 16 or older with at least an average IQ. After summarizing the total score, a cut-off value from 17 on indicates high likelihood of ASD (Freitag et al., 2007). The authors calculated internal consistencies for all three factors that were between .65 and .87 and the retest reliability which was .79 for the total score. They judged the AQ-k generally as reliable. When calculating the external validity by correlating other questionnaires scoring high when filled out by autistic individuals (e.g. scales of the Young Adult Self Report), they found correlations between .46 and .62. Thus, the external validity is acceptable (Freitag et al., 2007).

The **Comprehensive Autistic Trait Inventory - Revised (CATI-R)** has recently been validated for the German version (Hechler et al., 2024). This self-report measurement of autistic traits was part of the main study. The authors of the revised questionnaire invited autistic individuals to work together on creating items that more accurately represent autistic characteristics. In addition, their inventory considers specific female appearances of ASD characteristics (e.g. masking). The CATI-R has 42 items evenly distributed on the subscales *Social Interactions (SOC)*, *Sensory Sensitivity (SEN)*, *Repetitive Behaviors (REP)*, *Communication Difficulty (COM)*, *Cognitive Rigidity (RIG)* and *Masking (MAS)* (Hechler et al., 2024). One example item is “Meine Sinne werden in manchen Situationen überreizt.” on a 5-point Likert scale with the response options “ich stimme definitiv nicht zu”, “ich stimme eher nicht zu”, “ich stimme weder zu noch stimme ich nicht zu”, “ich stimme eher zu” and “ich stimme definitiv zu” (Hechler et al., 2024). The internal consistency is high, between .84 and .94. There is good convergent validity when correlating the CATI-R with questionnaires measuring similar constructs (e.g. AQ) with Pearson’s

coefficients between .82 and .85. They identified a total cut-off value from 153 on to classify someone as autistic. When distinguishing between gender, the researchers suggested a higher cut-off for non-binary people (163) and a lower value for men (148). Their questionnaire was tested on adults.

The **Saarbrücker Persönlichkeitsfragebogen zu Empathie (SPF-IRI)** is based on the Interpersonal Reactivity Index (IRI) and measures trait empathy (Paulus, 2009). It has 16 items divided into four subgroups: *perspective taking (PT)*, *fantasy (FS)*, *empathic concern (EC)* and *personal distress (PD)* (Paulus, 2009). The first factor measures cognitive empathy while the other factors measure affective components (Paulus, 2009). Internal consistency was judged by the researcher as satisfactory with a total value of .78; intercorrelations of the subtests were at a medium level. The item discriminability, or the correlation of an item with the total score of the SPF-IRI, is mostly usable (correlations between .27 and .52). Calculating the external validity, Paulus (2009) found significant positive correlations with other self-report empathy questionnaires between .31 and .78. All his participants were over 18. One example item is “Ich versuche, bei einem Streit zuerst beide Seiten zu verstehen, bevor ich eine Entscheidung treffe.” on a 5-point Likert response scale including the options “nie”, “selten”, “manchmal”, “oft” and “immer” (Paulus, 2009).

Procedure

Data acquisition took place between August 2024 and February 2025. The whole study took approximately 3 hours. Only the procedure relevant for my research questions is explained in detail.

Participants were invited to the department of Psychology at the University of Vienna after filling out the online prescreening questionnaire. Study appointments were scheduled either in the morning or in the afternoon. After they were given general information about the study, they signed the consent form. Their head was measured to choose a fitting fNIRS cap. The cap was put on and 3D coordinates of the brain were measured with the digitizer while the participant was sitting on a chair. Following that, the optodes were fitted and people joining the study were instructed to not move their head too much during the whole experiment.

Signal optimization using Aurora fNIRS (a software by NIRx Medical Technologies) default signal optimization and PHOEBE (Placing Headgear Optodes

Efficiently Before Experiment) on Matlab was carried out testing if the optodes were communicating with each other. Possible adjustments of the optodes on the scalp were made. Brain activation was recorded during the whole study, except in the part where questionnaires were filled out. The baseline brain activation was measured by instructing the participant to look at a fixation cross on the screen in front of them for one minute while not thinking about something specific. The facial expression task followed: participants were seated against the wall, so they faced a camera filming them. They were instructed to take off their glasses if they wore any and to look towards the camera without covering the face with their hands. For better lighting, two soft diffuse lightings were used standing next to the camera.

The study started with a 30 second baseline of just looking towards the camera to measure the general level of brain activation as a reference for task induced brain activation. Then the practice round followed with surprise as emotion which could be repeated as often as participants wanted. Before each emotion that they should express was revealed, a fixation cross was shown for five seconds. As soon as the emotion was revealed, they had unlimited time to think about the emotion (e.g. feeling into the emotion, when they felt it before). When participants were ready to start the experiment, they pushed a button and after a 5 second timer, a beep sound indicated the start of facial expression. They had 8 seconds time, and each round ended with another beep sound. After each round, six self-rating questions about the task were presented on the screen and participants had to answer on a 7-point Likert scale amongst others how much they thought of the viewer while expressing the emotion. There were four rounds for each emotion and including the practice round, the task required about 15 minutes in total. A curtain was drawn between experimenters and participants while their facial expressions were filmed so they did not feel observed.

After that, the abstract drawing task started with a 30 second baseline where participants were instructed to lean forward and only hold the pen over the tablet. Instructions were shown on the PC screen and for the actual drawing participants had to switch between the tablet tabs. There was a practice round lasting 90 seconds where they should draw the emotion surprise on the tablet. Then the same procedure as in the facial expression task followed (fixation cross, revealing the four emotions in random order, self-rating questions in between) but participants had 90 seconds to draw each shown emotion in an abstract way. They were told that they

could skip to the next round when they are done before the 90 seconds by pressing a button, but they should draw at least for one minute, so enough brain data is recorded. In total the drawing task lasted about 20 minutes with the screen being recorded the whole time as well as hand movements being filmed. After these tasks the participants had a break before the experiment continued with the questionnaires. The recording was stopped, and the devices were taken off. On the computer, participants filled out the German version of the CATI-R and the SPF-IRI. The CATI-R took approximately four minutes and the SPF-IRI took about two minutes (Hechler et al., 2024; Paulus, 2009). The experiment ended with questions about current age, country of origin and if they wanted to be contacted in the future. All in all, the steps described above took about one hour and 40 minutes.

Analysis

Analyzing fNIRS data included preprocessing in Matlab with the Homer3 toolbox and using the statistics software R to calculate linear mixed models (LMMs). Preprocessing consisted of cleaning the data, so removing bad channels, and z-scoring. Packages used in R were *lme4*, *lmerTest, car, ggplot2, psych, gridExtra, moments* and *dplyr*. For the first hypothesis regarding possible differences in empathy-related brain activation between the ASD and the non-autistic group, *brain activity* (BA) functioned as the dependent variable. Fixed categorical predictor variables were the *group* and *modality* (drawing or facial expression task). A random predictor variable took *individual* differences (ID) into account. The interaction between modality and group was examined additionally. The general formula in R was lmer ((BA ~ group + modality + group*modality + (1 | ID)). For the second hypothesis concerning possible group differences in the subjective empathy rating, again LMMs were used with the rating scores (*thought_of_others*) as dependent variable and *group* as fixed categorical predictor variable. *Individual* was the random predictor variable. In the formula, brain activation was replaced by *thought_of_others* and the modality was left out. For the third hypothesis, regarding the general correlation between empathy-related brain activation and subjective ratings, *brain activity* was the dependent variable and rating scores was the fixed factor. Again, *individual* variations functioned as a random factor. In the LMM formula *thought_of_others* was included in addition to brain activation. Since the facial

expression task took eight seconds, only the first eight seconds of the drawing task were analyzed regarding brain activation ensuring comparability across tasks.

16 channels were summarized into four regions of interest (ROIs). The ROIs measured with fNIRS were the medial prefrontal cortex (mPFC); summarizing study findings above it is part of the emotional brain, is involved in ToM and social interactions and predicts empathic accuracy, meaning correctly guessing an emotional state of a person. According to the section *Neural correlates* of empathy*,* another ROI was the right inferior frontal gyrus (rIFG) which is responsible for affective empathy in addition to the right dorsolateral prefrontal cortex (rdlPFC) which is responsible for components of cognitive empathy. The last ROI was the bilateral temporoparietal junction (right and left TPJ) that is responsible for emotional processing and attribution (see study findings of Sugranyes and Hadjikhani). For each ROI the formulas described above were adjusted.

For deepening insights, correlations were calculated between empathic brain activation and scores of ASD and empathy questionnaires. The CATI-R subscales and the SPF-IRI subscales were analyzed for each ROI and included in the LMM formula. Possible influences of group and/or modality on the correlation were examined. Since this was not part of the main research questions, those correlations functioned as additional exploratory results.

Results

Results are reported in the order of tested hypotheses. In the beginning, distributions and descriptive statistics of brain data and the self-rating scale are presented. The results follow for the first, second and third hypothesis. The section closes with the exploratory results showing values of ASD and empathy questionnaires and correlations of them with empathy-related brain activation.

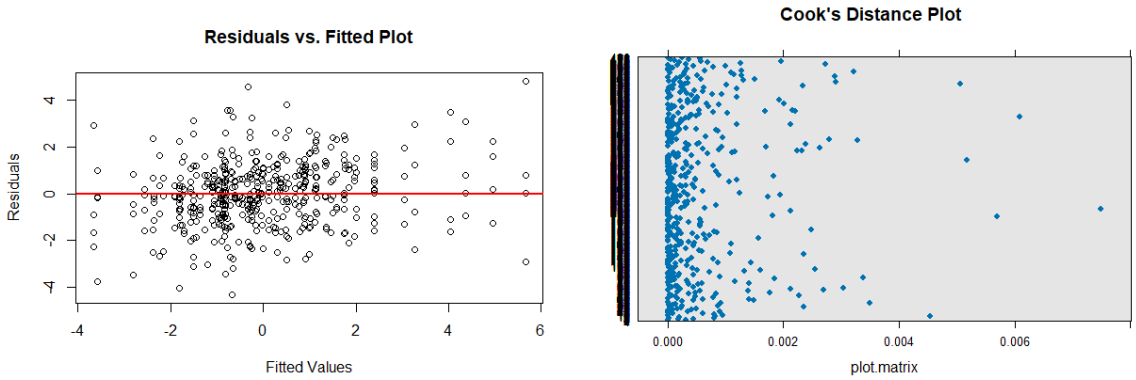
Assumptions and model diagnostics

Statistical requirements for using LMMs are linearity, homoscedasticity, and normality of residuals and low multicollinearity (Gelman & Hill, 2006). In addition, explained variance of random effects and outliers in the data were checked. When plotting the model, residuals showed a random scatter which means assumptions of linearity and homoscedasticity were fulfilled. Normality of residuals was given since the Q-Q plot points followed the reference line in a linear way. For examining

multicollinearity, the variance inflation factor (*VIF*) was calculated. For each predictor a *VIF* below 10 is acceptable (Field, 2018). For *group* this was 4.57, for *modality* it was 9.41 and for the interaction (*group*modality*) it was 13.32. Although the *VIF* for the interaction term (13.32) was above the conventional threshold, it is common for interactions to show elevated *VIF*s. The key predictors themselves (group and modality) had acceptable *VIF*s, which supports the overall assumption of low multicollinearity. Individual differences as a random factor showed moderate to high correlations with *modality* (r = - .42) and *group* (r = .58), hence random effects added to explained variance. Plotting brain activation data (histograms) separated per group and ROI, they all showed normal distributions and only few outliers. Examining influential cases with a residuals vs. fitted- and a Cook's Distance plot for each ROI, it could be concluded that those outliers did not influence the model fit in a significant way. Values for Cook's distance should lay below one (Field, 2018) and the residuals vs. fitted plot presented a random scatter.

Figure 1

Plots for potentially influential cases



Note. Example of mPFC; plots for other ROIs looked similar.

Finally, no imputation methods were necessary for missing data since LMMs are able to estimate parameters even with incomplete data sets (Field, 2018). Few values were missing in the brain data due to unusable data or measuring error.

Descriptive analysis of the self-rating scale *thought of others*

For each modality, participants had to state on a 7-point Likert scale how much they thought of the viewer while expressing the emotion. Higher values meant

that they thought more about the future viewer. Data was analyzed separately for group and modality. The autistic group (n = 30) had a mean of 2.6 in the modality drawing (SD = 1.52, Mdn = 2.38) and a mean of 3.09 when it comes to the facial expression task (face) (SD = 1.42, Mdn = 3). The non-autistic group (n = 30) had a mean of 2.48 in the modality drawing (SD = 1.26, Mdn = 2.25) and a mean of 2.88 in the modality face (SD = 1.37, Mdn = 2.75). Values between one and three were chosen most frequently. Data was distributed ordinally and looking at the histograms, a right skewness was apparent. Skewness was computed with the sample skewness coefficient $G1$. Values greater or less than zero suggest a non-symmetrical distribution (Doane & Seward, 2011). Skewness values were between .22 and .89 for all four distributions. Although the self-rating scores were distributed non-normally, they could be integrated in the LMM (second and third hypothesis) since the model is sufficiently robust (Norman, 2010). Furthermore, with five to seven response options, ordinal data can be treated as continuous data (e.g. Rhemtulla et al., 2012; Sullivan & Artino, 2013).

H1: Differences in empathy-related brain activation during emotion expression

To examine if there were any differences in empathy-related brain activation between the autistic- and the non-autistic group, a LMM was performed for each ROI. Potential influence of modality was included in the model.

mPFC: When comparing the base model without predictors (only individual differences) with the alternative model including the predictors (group, modality and their interaction), it turned out that the alternative model significantly improved the model fit (χ^2 = 37.15, df = 3, p < .001). The Log-Likelihood was higher for the alternative model than the base model (-1006.5 vs. -1025.1) suggesting that the model with predictors explained the data better. With those results of the ANOVA and the Likelihood Ratio Test it could be concluded that the predictors significantly explained the variance in mPFC activation. When extending the model to a random slope model, meaning putting individual differences and predictors in relation, it turned out to be the best fit in comparison to the other models. Hence, adding the correlation between intercepts and slopes significantly contributed to explained variance (χ^2 = 122.17, df = 9, p < .001).

Chart 1

Averaged activation of mPFC separated for group and modality

group	modality	Mean	SD	N
1	1	-0.48	1.99	30
1	3	0.96	2.63	30
2	1	-0.59	1.75	30
2	3	0.10	2.49	30

Note. Group 1 = autistic, 2 = NA; modality 1 = drawing, 3 = face

The main effect of group was not significant (β = –.09, *SE* = .40, *t* = –.23, *df* = 55, *p* = .82), suggesting no overall difference in mPFC activation between autistic and non-autistic participants. In contrast, the main effect of modality was significant (β = 1.47, *SE* = .46, *t* = 3.21, *df* = 28, *p* < .001), indicating that brain activation was higher in the face modality relative to the drawing modality. The interaction between group and modality was not significant (β = –.78, *SE* = .69, *t* = –1.12, *df* = 56, *p* = .27), suggesting that the effect of modality on mPFC activation did not differ between the groups. Random effects indicated substantial between-participant variability in the intercept (autistic group during the drawing modality) (*Var* = 2.19, *SD* = 1.48), as well as in the effects of group (*Var* = 1.02, *SD* = 1.01) and modality (*Var* = 4.73, *SD* = 2.18). The random intercept and the group slope were moderately negatively correlated (*r* = -.61). The residual variance was 2.32 (*SD* = 1.52).

rIFG: When comparing the base model without predictors with the alternative model including the predictors group, modality, and their interaction, it turned out that the alternative model significantly improved the model fit (χ^2 = 63.47, *df* = 3, *p* < .001). The Log-Likelihood was higher for the alternative model than for the base model (-1260.9 vs. -1292.6), suggesting that the model with predictors explained the data better. When extending the model to a random slope model, meaning allowing individual differences in slopes for group and modality, the model fit improved significantly (χ^2 = 72.55, *df* = 9, *p* < .001). Therefore, as for mPFC, the best-fitting model was the random slope

model with correlated intercepts and slopes, which was chosen for hypothesis testing.

Chart 2

Averaged activation of rIFG separated for group and modality

group	modality	Mean	SD	N
1	1	0.27	2.85	30
1	3	1.98	4.55	30
2	1	-0.25	2.56	30
2	3	2.77	4.01	30

The main effect of group was not significant (β = –0.53, *SE* = 0.53, *t* = –1.00, *df* = 54, *p* = .32), suggesting no overall difference in rIFG activation between autistic and non-autistic participants. In contrast, the main effect of modality was significant (β = 1.69, *SE* = 0.76, *t* = 2.23, *df* = 29, *p* = .033), indicating that rIFG activation was higher in the face modality relative to the drawing modality. The interaction between group and modality was not significant (β = 1.33, *SE* = 0.98, *t* = 1.36, *df* = 57, *p* = .18), suggesting that the effect of modality on rIFG activation did not differ between the groups. Random effects indicated substantial between-participant variability in the intercept (autistic group during the drawing modality) (*Var* = 3.43, *SD* = 1.85), as well as in the effects of group (*Var* = 2.71, *SD* = 1.65) and modality (*Var* = 13.19, *SD* = 3.63). The interaction between group and modality also showed considerable variation across participants (*Var* = 9.09, *SD* = 3.02). The random intercept and the group slope were strongly negatively correlated (*r* = –.80), while the modality slope was moderately negatively correlated with the intercept (*r* = –.53) and positively correlated with the group slope (*r* = .40). The residual variance was 7.51 (*SD* = 2.74).

rdlPFC: When comparing the base model without predictors to the alternative model with predictors, the alternative model significantly improved model fit (χ^2 = 9.32, *df* = 3, *p* = .025). The Log-Likelihood was higher for the alternative model than the base model (-1159.2 vs. -1163.8), suggesting that including predictors explained more variance in rdlPFC activation. Further extending the model to allow random slopes (accounting for individual differences in predictor effects) significantly

improved model fit (χ^2 = 111.1, *df* = 9, *p* < .001). Moreover, removing the correlation between intercepts and slopes led to a worse model fit (χ^2 = 0.00, *df* = 1, *p* = 1.00), confirming that the correlation structure contributed to the explained variance. Based on these comparisons, the model with random intercepts and correlated random slopes was selected for hypothesis testing.

Chart 3

Averaged activation of rdlPFC separated for group and modality

group	modality	Mean	SD	N
1	1	-0.10	2.91	30
1	3	0.57	3.94	30
2	1	-1.05	2.40	30
2	3	-0.51	3.11	30

The main effect of group was not significant (*β* = –.96, *SE* = .57, *t* = –1.69, *df* = 55, *p* = .10), suggesting no overall difference in rdlPFC activation between autistic and non-autistic participants. Similarly, the main effect of modality was not significant (*β* = 0.61, *SE* = 0.65, *t* = 0.95, *df* = 29, *p* = .35), indicating no evidence for an overall difference in activation between the drawing and face modality. The interaction between group and modality was also not significant (*β* = –.06, *SE* = .94, *t* = –0.07, *df* = 58, *p* = .95), suggesting that the effect of modality on rdlPFC activation did not differ between the groups. Random effects indicated substantial between-participant variability in the intercept (autistic group during the drawing modality) (*Var* = 3.97, *SD* = 1.99), as well as in the effects of group (*Var* = 2.83, *SD* = 1.68) and modality (*Var* = 9.36, *SD* = 3.06). The interaction term also showed individual variability (*Var* = 3.16, *SD* = 1.78). The random intercept and the group slope were moderately negatively correlated (*r* = –.56). The residual variance was 4.66 (*SD* = 2.16).

rTPJ: When comparing the base model without predictors with the alternative model including the predictors, the alternative model significantly improved the model fit (χ^2 = 47.40, *df* = 3, *p* < .001). The Log-Likelihood was higher for the alternative model than for the base model (–1103.5 vs. –1127.2), suggesting that the model with predictors explained the data better. Extending the model to include random slopes and correlation structures, further improved the model fit (χ^2 = 91.18,

df = 9, *p* < .001), indicating that modeling individual differences in the effects of group and modality significantly contributed to explained variance. As a result, the model with both random intercepts and slopes, including their correlation, was chosen for hypothesis testing.

Chart 4

Averaged activation of rTPJ separated for group and modality

group	modality	Mean	SD	N
1	1	0.98	2.27	30
1	3	0.98	3.02	30
2	1	-0.35	1.98	30
2	3	1.76	2.84	30

The main effect of group was significant (*β* = -1.33, *SE* = 0.43, *t* = -3.11, *df* = 57, *p* = .003), suggesting that autistic participants showed lower overall rTPJ activation compared to non-autistic participants. In contrast, the main effect of modality was not significant (*β* = -0.16, *SE* = 0.63, *t* = -0.25, *df* = 26, *p* = .80), indicating that there was no general difference in rTPJ activation between the face and drawing modality. However, the interaction between group and modality was significant (*β* = 2.27, *SE* = 0.80, *t* = 2.85, *df* = 50, *p* = .006), suggesting that the effect of modality on rTPJ activation differed between groups. Random effects indicated substantial between-participant variability in the intercept (autistic group during the drawing modality) (*Var* = 2.28, *SD* = 1.51), as well as in the effects of group (*Var* = 1.21, *SD* = 1.10) and modality (Var = 10.25, *SD* = 3.20). The random intercept and group slope were moderately negatively correlated (*r* = -0.60), while the random intercept and modality slope were strongly negatively correlated (*r* = -0.70). The residual variance was 3.49 (*SD* = 1.87).

lTPJ: The model including fixed effects for group, modality, and their interaction provided a significantly better fit than the base model with only a random intercept (χ^2 = 69.26, *df* = 3, *p* < .001), suggesting that the inclusion of these predictors improved explanatory power for analyzing lTPJ activation. Adding correlated random slopes for group and modality further improved the model fit (χ^2 =

87.79, *df* = 9, *p* < .001). Removing the correlation between intercepts and slopes did not improve the model further (χ^2 = 0.00, *df* = 1, *p* = 1.00), confirming that the correlation structure contributed to explained variance. Consequently, the model with correlated random slopes was selected for hypothesis testing.

Chart 5

Averaged activation of ITPJ separated for group and modality

group	modality	Mean	SD	N
1	1	0.05	2.28	30
1	3	1.49	3.69	30
2	1	0.18	2.27	30
2	3	2.67	3.82	30

The main effect of group was also not significant (*β* = 0.15, *SE* = 0.45, *t* = 0.32, *df* = 55.66, *p* = 0.75), suggesting no overall difference in ITPJ activation between autistic and non-autistic participants. In contrast, the main effect of modality was significant (*β* = 1.36, *SE* = 0.50, *t* = 2.70, *df* = 29.20, *p* = 0.011), indicating that brain activation was higher in the face modality relative to the drawing modality. The interaction between group and modality was not significant (*β* = 1.13, *SE* = 0.80, *t* = 1.41, *df* = 55.66, *p* = 0.16), suggesting that the effect of modality on ITPJ activation did not differ between the groups. Random effects indicated substantial between-participant variability in the intercept (autistic group during the drawing modality) (*Var* = 2.51, *SD* = 1.58), as well as in the effects of group (*Var* = 5.29, *SD* = 2.30) and modality (*Var* = 5.19, *SD* = 2.28). The random intercept and the group slope were strongly negatively correlated (*r* = -0.90). The residual variance was 4.69 (*SD* = 2.17).

H2: Differences in the self-rating scores of empathy (*thought of others*)

To detect possible group differences in the scale *thought of others* during the expression of emotions, the LMM was adjusted by substituting ROIs with the self-rating scale and leaving out modality. First, it was tested whether including group as a predictor improved model fit compared to the base model that only included a random intercept for participants. The model with group did not significantly improve fit over the base model (χ^2 = 0.30, *df* = 1, *p* = .59). This suggests that adding group as a predictor did not explain additional variance in *thought of others* ratings. Next, it

was tested whether allowing the effect of group to vary across participants improved model fit. Neither the model with a correlated random slope nor the model with an uncorrelated random slope significantly improved fit over the simpler model (all *p*-values > .97), indicating no meaningful individual variability in the group effect. Despite the non-significant model comparisons, the hypothesis was tested with the model including the group because it allows estimating group differences.

The random effects indicated substantial variation in autistic participants' baseline scores, with an intercept variance of 1.28 (*SD* = 1.13). The residual variance was 1.38 (*SD* = 1.17), suggesting additional within-participant variability. The fixed effects revealed that the intercept was significant (β = 2.85, *SE* = 0.22, *t* = 12.94, *df* = 58, *p* < .001), indicating that autistic participants had a mean *thought of others* score of 2.85. However, the group effect was non-significant (β = -0.17, *SE* = 0.31, *t* = -0.54, *df* = 58 *p* = .59), meaning that non-autistic participants did not significantly differ from autistic participants in their self-ratings of thinking of others.

H3: Correlation of empathy-related brain activation and *thought of others* scores

To examine possible correlations between self-rating scores of thinking of others and empathy-related brain activation, the self-rating scale was included in the LMM for each ROI. Group and modality were taken into account.

mPFC: The first model comparison evaluated whether including group, modality, subjective self-ratings of thinking of others, and their interactions improved model fit over the random intercept-only model. The alternative model provided a significantly better fit than the base model (χ^2 = 54.16, *df* = 7, *p* < .001), with lower AIC (2016.0 vs. 2056.2) and BIC (2057.4 vs. 2068.6) values. This indicates that these predictors and their interactions explained additional variance in mPFC activation. The second comparison assessed whether allowing random slopes for the group-by-modality interaction further improved model fit. The model with correlated random intercepts and slopes significantly outperformed the model with only fixed effects (χ^2 = 112.4, *df* = 9, *p* < .001), suggesting substantial between-participant variability in the effect of group and modality on mPFC activation. The uncorrelated random slopes model did not provide additional improvement over the correlated one, and therefore, the model with correlated random slopes was selected as the final model for hypothesis testing.

The fixed effects revealed that the overall relationship between self-ratings of thinking of others and mPFC activation was not significant (β = -0.16, *SE* = 0.13, *t* = -1.23, *df* = 134.14, *p* = 0.22), indicating no significant correlation between subjective self-ratings and mPFC activation across participants. The effect of group on the correlation was not significant (β = -0.60, *SE* = 0.62, *t* = -0.98, *df* = 119.96, *p* = 0.33). This indicated no evidence that group membership (autistic vs. non-autistic) influenced the correlation between self-ratings and mPFC activation. Similarly, the effect of modality on the relationship between self-ratings and brain activation was also not significant (β = 0.16, *SE* = 0.20, *t* = 0.81, *df* = 257.01, *p* = 0.42). This suggests that the type of task modality (drawing vs. face) did not significantly influence the correlation.

rIFG: The alternative model with group, modality and self-rating scores of thinking of others provided a significantly better fit than the base model without the predictors (χ^2 = 65.69, *df* = 7, *p* < .001), with lower AIC (2539.5 vs. 2591.2) and BIC (2581.1 vs. 2603.7) values. This indicates that these predictors and their interactions explained additional variance in rIFG activation. The second comparison assessed whether allowing random slopes for the group-by-modality interaction further improved model fit. The model with correlated random intercepts and slopes significantly outperformed the model with only fixed effects (χ^2 = 71.62, *df* = 9, *p* < .001), suggesting substantial between-participant variability in the effect of group and modality on rIFG activation. The uncorrelated random slopes model did not provide additional improvement over the correlated one, and therefore, the model with correlated random slopes was selected for analysis.

The fixed effects revealed that the overall relationship between self-ratings of thinking of others and rIFG activation was not significant (β = 0.16, *SE* = 0.21, *t* = 0.76, *df* = 89.92, *p* = 0.45), indicating no significant correlation between subjective self-ratings and rIFG activation across participants. The effect of group on the correlation was also not significant (β = 0.03, *SE* = 0.92, *t* = 0.04, *df* = 105.08, *p* = 0.97), suggesting that group membership (autistic vs. non-autistic) did not influence the correlation between self-ratings and rIFG activation. Similarly, the effect of modality on the relationship was not significant (β = 1.47, *SE* = 1.21, *t* = 1.21, *df* = 88.63, *p* = 0.23), indicating that the type of task modality (drawing vs. face) did not significantly influence the correlation.

rdlPFC: The first model comparison evaluated whether including group, modality, subjective self-ratings of thinking of others, and their interactions improved model fit over the base model. The alternative model provided a significantly better fit than the base model (χ^2 = 16.05, *df* = 7, *p* = 0.02). This indicates that these predictors and their interactions explained additional variance in rdlPFC activation. The second comparison assessed whether allowing random slopes for the group-by-modality interaction further improved model fit. The model with correlated random intercepts and slopes significantly outperformed the model with only fixed effects (χ^2 = 106.79, *df* = 9, *p* < .001), suggesting substantial between-participant variability in the effect of group and modality on rdlPFC activation. The uncorrelated random slopes model did not provide additional improvement over the correlated one, and therefore, the model with correlated random slopes was selected as the final model for hypothesis testing.

The fixed effects revealed that the overall relationship between self-ratings of thinking of others and rdlPFC activation was not significant (β = -0.04, *SE* = 0.19, *t* = -0.24, *df* = 121.12, *p* = 0.81), indicating no significant correlation between subjective self-ratings and rdlPFC activation across participants. The effect of group on the correlation was not significant (β = 0.05, *SE* = 0.26, *t* = 0.18, *df* = 236.19, *p* = 0.86). This indicates no evidence that group membership influenced the correlation between self-ratings and rdlPFC activation. Similarly, the effect of modality on the relationship between self-ratings and brain activation was also not significant (β = 0.09, *SE* = 0.28, t = 0.32, *df* = 253.28, *p* = 0.75). This suggests that the type of task modality did not significantly influence the correlation.

rTPJ: The first model comparison evaluated whether including the predictors improved model fit over the base model. The alternative model provided a significantly better fit than the base model (χ^2 = 58.91, *df* = 7, *p* < .001). This indicates that these predictors and their interactions explained additional variance in rTPJ activation. The second comparison assessed whether allowing random slopes for the group-by-modality interaction further improved model fit. The model with correlated random intercepts and slopes significantly outperformed the model with only fixed effects (χ^2 = 81.71, *df* = 9, *p* < .001), suggesting substantial between-participant variability in the effect of group and modality on rTPJ activation. The uncorrelated random slopes model did not provide additional improvement over the

correlated one, and therefore, the model with correlated random slopes was selected as the final model for hypothesis testing.

The fixed effects revealed that the overall relationship between self-rating scores and rTPJ activation was not significant (β = 0.20, *SE* = 0.15, *t* = 1.32, *df* = 99.21, *p* = 0.19), indicating no significant correlation between subjective self-ratings and rTPJ activation across participants. The effect of group on the correlation was not significant (β = -0.16, *SE* = 0.21, *t* = -0.76, *df* = 204.72, *p* = 0.45). This indicates no evidence that group membership influenced the correlation between self-ratings and rTPJ activation. Similarly, the effect of modality on the relationship was also not significant (β = -0.21, *SE* = 0.23, *t* = -0.89, *df* = 272.60, *p* = 0.37). This suggests that the type of task modality did not significantly influence the correlation.

lTPJ: When comparing the model without predictors with the model including the predictors, the alternative model explained the data better than the base model (χ^2 = 71.42, *df* = 7, *p* < .001). This indicates that the predictors and their interactions explained additional variance in lTPJ activation. The second comparison assessed whether allowing random slopes for the group-by-modality interaction further improved model fit. The model with correlated random intercepts and slopes significantly outperformed the model with only fixed effects (χ^2 = 87.78, *df* = 9, *p* < .001), suggesting substantial between-participant variability in the effect of group and modality on lTPJ activation. The uncorrelated random slopes model did not provide additional improvement over the correlated one, and therefore, the model with correlated random slopes was selected as the final model for hypothesis testing.

The fixed effects revealed that the overall relationship between self-ratings of thinking of others and lTPJ activation was not significant (β = -0.02, *SE* = 0.16, *t* = -0.11, *df* = 86.70, *p* = 0.92), indicating no significant correlation between subjective self-ratings and lTPJ activation across participants. The effect of group on the correlation was not significant (β = 0.07, *SE* = 0.23, *t* = 0.31, *df* = 174.41, *p* = 0.76). This indicates no evidence that group membership influenced the correlation between self-ratings and lTPJ activation. Similarly, the effect of modality on the relationship between self-ratings and brain activation was also not significant (β = 0.16, *SE* = 0.25, *t* = 0.63, *df* = 171.01, *p* = 0.53). This suggests that the type of task modality did not significantly influence the correlation.

Exploratory results

Correlations were calculated in the LMM between empathic brain activation and scores of trait empathy- and ASD questionnaire subscales for each ROI, while controlling for group and modality as possible influencing factors on the relationship.

Descriptive statistics of SPF-IRI: For *IRI_Fantasy*, the autistic group had a mean of 11.9 (*SD* = 3.89), with skewness of -0.08 and kurtosis of 2.53. The non-autistic group showed a higher mean score of 13.7 (*SD* = 2.36), with skewness of 0.11 and kurtosis of 1.98. For *IRI_Personal Distress*, the autistic group had a mean of 14.1 (*SD* = 2.40), with skewness of 0.33 and kurtosis of 2.33. In contrast, the non-autistic group had a mean of 10.9 (*SD* = 3.33), with skewness of 0.24 and kurtosis of 2.64. Regarding *IRI_Perspective Taking*, the autistic group's mean was 14.4 (*SD* = 2.90), with skewness of 0.23 and kurtosis of 2.09, while the non-autistic group had a mean of 14.8 (*SD* = 2.74), with skewness of -0.14 and kurtosis of 2.41. For *IRI_Empathic Concern*, the autistic group had a mean of 14.9 (*SD* = 2.74), with skewness of -0.53 and kurtosis of 2.08, while the non-autistic group had a mean of 15.1 (*SD* = 1.79), with skewness of 0.43 and kurtosis of 2.47. To assess the normality of the data, Shapiro-Wilk tests were conducted for each subscale within both groups. The results indicated that all subscales significantly deviated from normality for both groups (p-values < 0.05). Despite the non-normality of the data, IRI subscales were still integrated in the LMM since it is robust enough.

Descriptive statistics of CATI-R: For *CATI_Social Interactions*, the autistic group had a mean of 27.7 (*SD* = 3.91), with skewness of -0.316 and kurtosis of 2.33. The non-autistic group had a lower mean score of 15.6 (*SD* = 4.82), with skewness of 0.221 and kurtosis of 2.31. For *CATI_Communication Difficulty*, the autistic group had a mean of 25.6 (*SD* = 4.65), with skewness of -0.477 and kurtosis of 3.20. In contrast, the non-autistic group had a mean of 12.3 (*SD* = 3.81), with skewness of 0.345 and kurtosis of 2.66. For *CATI_Masking*, the autistic group had a mean of 27.8 (*SD* = 4.42), with skewness of -1.00 and kurtosis of 5.11. The non-autistic group showed a mean score of 17.7 (*SD* = 4.58), with skewness of 0.299 and kurtosis of 2.22. Regarding *CATI_Cognitive Rigidity*, the autistic group had a mean of 29.8 (*SD* = 3.94), with skewness of -0.707 and kurtosis of 2.91. The non-autistic group had a mean of 20.3 (*SD* = 5.46), with skewness of 0.169 and kurtosis of 1.95. For *CATI_Repetitive Behaviors*, the autistic group had a mean of 30.7 (*SD* = 4.13), with skewness of -1.20 and kurtosis of 4.02. The non-autistic group had a mean of 18.5

(*SD* = 5.62), with skewness of 0.281 and kurtosis of 2.94. For *CATI_Sensory Sensitivity*, the autistic group had a mean of 29.1 (*SD* = 5.66), with skewness of -1.39 and kurtosis of 4.77. The non-autistic group showed a mean of 17.0 (*SD* = 6.60), with skewness of 0.529 and kurtosis of 2.79. To assess the normality of the data, Shapiro-Wilk tests were conducted for each subscale within both groups. The results indicated that all subscales significantly deviated from normality for both groups (p-values < 0.05). Due to robustness of the LMM, CATI-R subscales were integrated in the model, despite non-normally distributed scores.

Model fit comparisons: For the correlation of mPFC activation and the IRI Fantasy subscale the correlated random slopes model significantly outperformed the base model with a decrease in AIC (2026.1 to 1921.8) and BIC (2067.5 to 2000.4), and a significant improvement in model fit (χ^2 = 122.27, *df* = 9, *p* < 0.001). The uncorrelated random slopes model did not improve the model fit, making the correlated model the most appropriate for analysis. For mPFC and IRI Personal Distress the correlated random slopes model significantly outperformed the base model with a decrease in AIC (2026.1 to 1921.8) and BIC (2067.5 to 2000.4), and a significant improvement in model fit (χ^2 = 122.27, *df* = 9, *p* < 0.001). The uncorrelated random slopes model did not improve the model fit, making the correlated model the most appropriate for analysis. For mPFC and IRI Perspective Taking the correlated random slopes model significantly outperformed the base model with a decrease in AIC (2012.3 to 1914.1) and BIC (2053.7 to 1992.7), and a significant improvement in model fit (χ^2 = 116.17, *df* = 9, *p* < 0.001). The uncorrelated random slopes model did not improve the model fit, making the correlated model the most appropriate for analysis. For mPFC and IRI Empathic Concern the correlated random slopes model significantly outperformed the base model with a decrease in AIC (2024.5 to 1923.5) and BIC (2065.9 to 2002.1), and a significant improvement in model fit (χ^2 = 119.00, *df* = 9, *p* < 0.001). The uncorrelated random slopes model did not improve the model fit, making the correlated model the most appropriate for analysis. For all the following correlations the correlated model was always the best fitting model as well. In the next section significant results are summarized (for complete results see *Chart 6*).

mPFC x IRI Perspective Taking: The correlation between mPFC activation and the IRI Perspective Taking subscale was significant ($p = 0.01$), indicating a positive relationship between the two variables. Group had a significant influence on this correlation ($p = 0.04$), suggesting that the relationship between mPFC activation and IRI Perspective Taking differed across groups, with a stronger association in the autistic group.

mPFC x IRI Empathic Concern: The correlation between mPFC activation and the IRI Empathic Concern subscale was not significant but showed a trend ($p = 0.08$). Modality significantly influenced this correlation ($p = 0.04$).

ITPJ x IRI Perspective Taking: The correlation between ITPJ activation and the IRI Perspective Taking subscale was not significant ($p = 0.06$), though there was a trend suggesting a positive association. Group significantly influenced this relationship ($p = 0.01$), indicating that non-autistic participants showed a stronger association between ITPJ activation and perspective-taking tendencies.

mPFC x CATI Sensory Sensitivity: The correlation between mPFC activation and CATI Sensory Sensitivity was significant ($p = 0.04$), indicating a positive association. Group significantly influenced this relationship ($p < 0.05$), suggesting that non-autistic participants showed a stronger association between mPFC activation and sensory sensitivity.

mPFC x CATI Masking: The correlation between mPFC activation and the CATI Masking subscale was significant ($p < 0.001$), suggesting a positive correlation. Group ($p < 0.01$) significantly influenced the relationship, with non-autistic participants showing a stronger correlation between mPFC activation and CATI Masking scores.

rIFG x CATI Social Interactions: The correlation between rIFG activation and CATI Social Interactions was significant ($p = 0.01$), indicating a positive association. Group significantly influenced this relationship ($p = 0.01$), suggesting that non-autistic participants showed a stronger association between rIFG activation and CATI Social Interactions. Modality also had a significant effect ($p = 0.02$),

indicating that the type of task (Face) was associated with a stronger correlation between rIFG activation and CATI Social Interactions.

rIFG x CATI Sensory Sensitivity: The correlation between rIFG activation and CATI Sensory Sensitivity was not significant ($p = 0.09$), though there was a trend towards a positive association. Group significantly influenced this relationship ($p = 0.04$), indicating that non-autistic participants showed a stronger association between rIFG activation and sensory sensitivity. Modality had a significant effect ($p = 0.003$), indicating that the type of task (Face) was associated with a stronger correlation between rIFG activation and sensory sensitivity. The interaction between group and modality was significant ($p = 0.03$), suggesting that the relationship between rIFG activation and sensory sensitivity differed between groups depending on the task modality.

rIFG x CATI Masking: The correlation between rIFG activation and the CATI Masking subscale was significant ($p = 0.01$), indicating a positive relationship. Group ($p = 0.005$) and modality ($p = 0.009$) also significantly influenced the relationship. The association was stronger in the non-autistic group and in the face modality.

rdlPFC x CATI Communication Difficulty: The correlation between rdlPFC activation and the CATI Communication Difficulty subscale was not significant. However, modality ($p = 0.04$) was significantly associated with rdlPFC activation and subscale scores, with a stronger association in the Drawing modality compared to the Face modality.

lTPJ x CATI Sensory Sensitivity: The correlation between lTPJ activation and CATI Sensory Sensitivity was significant ($p < 0.001$), indicating a positive association. Group significantly influenced the relationship ($p < 0.01$), showing that the association was stronger in the non-autistic group.

lTPJ x CATI Masking: The correlation between lTPJ activation and CATI Masking was significant ($p = 0.02$), indicating a positive association. Group significantly influenced the relationship ($p = 0.02$), showing that the association was stronger in the non-autistic group.

Chart 6

Correlations between IRI and CATI subscale scores with activation of each ROI

Correlations	***β***	***SE***	***t***	***p***
mPFC x IRI Fantasy	0.16	0.08	1.98	0.06
* group	3.36	1.79	1.88	0.07
* modality	1.92	1.51	1.27	0.22
* modality * group	-1.68	3.46	-0.49	0.63
mPFC x IRI Personal Distress	-0.09	0.13	-0.70	0.49
* group	-0.93	2.07	-0.45	0.66
* modality	2.58	2.75	0.94	0.36
* modality * group	-1.96	3.30	-0.60	0.56
mPFC x IRI Perspective Taking	0.27	0.10	2.73	0.01
* group	-0.29	0.14	-2.10	0.04
* modality	0.34	2.36	0.14	0.89
* modality * group	2.15	3.74	0.57	0.57
mPFC x IRI Empathic Concern	0.19	0.11	1.80	0.08
* group	4.05	2.74	1.48	0.15
* modality	5.31	2.52	2.10	0.04
* modality * group	-3.61	5.15	-0.70	0.49
rIFG x IRI Fantasy	-0.01	0.11	-0.08	0.94
* group	0.03	2.37	0.01	0.99
* modality	0.40	2.46	0.16	0.87
* modality * group	4.35	4.48	0.97	0.34
rIFG x IRI Personal Distress	0.34	0.17	2.02	0.053
* group	3.57	2.65	1.35	0.19
* modality	3.93	4.56	0.86	0.40
* modality * group	-1.26	5.06	-0.25	0.80
rIFG x IRI Perspective Taking	0.09	0.15	0.60	0.56
* group	2.62	2.80	0.94	0.35
* modality	3.25	3.90	0.84	0.41
* modality * group	3.16	5.21	0.61	0.55
rIFG x IRI Empathic Concern	0.12	0.15	0.79	0.44
* group	5.84	3.54	1.65	0.10
* modality	2.52	4.32	0.58	0.56
* modality * group	-1.12	6.92	-0.16	0.87
rdlPFC x IRI Fantasy	0.18	0.12	1.48	0.15
* group	4.97	2.58	1.93	0.06
* modality	1.49	2.18	0.68	0.50
* modality * group	-4.70	4.55	-1.03	0.31

Correlations	*β*	*SE*	*t*	*p*
rdlPFC x IRI Personal Distress	-0.06	0.18	-0.32	0.75
* group	-0.63	2.88	-0.22	0.83
* modality	-2.77	3.83	-0.72	0.48
* modality * group	1.87	4.50	0.42	0.68
rdlPFC x IRI Perspective Taking	0.28	0.15	1.88	0.07
* group	1.14	2.98	0.38	0.70
* modality	-1.27	3.40	-0.37	0.71
* modality * group	5.46	5.05	1.08	0.28
rdlPFC x IRI Empathic Concern	0.12	0.15	0.78	0.44
* group	1.59	3.98	0.40	0.69
* modality	0.84	3.61	0.23	0.82
* modality * group	1.50	6.86	0.22	0.83
rTPJ x IRI Fantasy	0.01	0.08	0.14	0.89
* group	-2.64	1.97	-1.34	0.19
* modality	-1.83	2.05	-0.90	0.38
* modality * group	6.07	3.52	1.73	0.09
rTPJ x IRI Personal Distress	0.26	0.13	2.04	0.05
* group	0.89	2.06	0.43	0.67
* modality	3.08	3.80	0.81	0.42
* modality * group	-1.12	4.15	-0.27	0.79
rTPJ x IRI Perspective Taking	0.13	0.11	1.18	0.25
* group	1.44	2.27	0.64	0.53
* modality	-0.41	3.30	-0.13	0.90
* modality * group	5.53	4.23	1.31	0.20
rTPJ x IRI Empathic Concern	0.02	0.12	0.14	0.89
* group	-0.97	3.04	-0.32	0.75
* modality	-5.07	3.54	-1.43	0.16
* modality * group	10.58	5.43	1.95	0.06
lTPJ x IRI Fantasy	0.04	0.09	0.43	0.67
* group	3.04	1.99	1.53	0.13
* modality	-1.85	1.51	-1.22	0.23
* modality * group	2.60	4.02	0.65	0.52
lTPJ x IRI Personal Distress	-0.17	0.15	-1.18	0.25
* group	-2.68	2.31	-1.16	0.25
* modality	2.05	3.05	0.67	0.51
* modality * group	-4.77	3.61	-1.32	0.19
lTPJ x IRI Perspective Taking	0.22	0.12	1.94	0.06
* group	6.13	2.27	2.70	0.01
* modality	-2.70	2.49	-1.09	0.29
* modality * group	7.94	4.27	1.86	0.07

Correlations	*β*	*SE*	*t*	*p*
ITPJ x IRI Empathic Concern	0.14	0.13	1.06	0.30
* group	2.91	3.12	0.93	0.36
* modality	-0.72	2.88	-0.25	0.81
* modality * group	7.89	6.05	1.30	0.20

Correlations	*β*	*SE*	*t*	*p*
mPFC x CATI Social Interactions	0.12	0.08	1.55	0.13
* group	3.77	2.37	1.60	0.12
* modality	4.47	3.30	1.36	0.19
* modality * group	-4.79	3.75	-1.28	0.21
mPFC x CATI Sensory Sensitivity	0.11	0.05	2.20	0.04
* group	3.44	1.67	2.06	0.05
* modality	2.76	2.37	1.16	0.25
* modality * group	-2.89	2.79	-1.04	0.31
mPFC x CATI Repetitive Behaviors	0.08	0.08	0.99	0.33
* group	-0.04	0.09	-0.45	0.65
* modality	-0.03	0.11	-0.24	0.81
* modality * group	-0.02	0.15	-0.17	0.87
mPFC x CATI Communication Difficulty	-0.02	0.07	-0.34	0.74
* group	-1.46	1.95	-0.75	0.46
* modality	-2.06	2.50	-0.82	0.42
* modality * group	2.75	3.08	0.89	0.38
mPFC x CATI Cognitive Rigidity	0.09	0.08	1.07	0.30
* group	1.34	2.76	0.49	0.63
* modality	0.91	3.84	0.24	0.82
* modality * group	0.94	4.35	0.22	0.83
mPFC x CATI Masking	0.22	0.06	3.75	0.001
* group	5.56	1.94	2.87	0.01
* modality	5.60	2.84	1.97	0.06
* modality * group	-2.87	3.52	-0.82	0.42
rIFG x CATI Social Interactions	0.27	0.10	2.80	0.01
* group	7.89	2.93	2.69	0.01
* modality	12.60	5.09	2.48	0.02
* modality * group	-10.12	5.53	-1.83	0.08
rIFG x CATI Sensory Sensitivity	0.13	0.07	1.78	0.09
* group	4.99	2.29	2.18	0.04
* modality	11.40	3.55	3.21	0.003
* modality * group	-8.97	3.96	-2.26	0.03
rIFG x CATI Repetitive Behaviors	0.13	0.10	1.32	0.20
* group	3.31	3.32	1.00	0.33
* modality	6.22	5.69	1.09	0.28
* modality * group	-1.98	6.09	-0.33	0.75

Correlations	*β*	*SE*	*t*	*p*
rIFG x CATI Communication Difficulty	0.15	0.09	1.75	0.09
* group	3.94	2.54	1.55	0.13
* modality	7.80	4.14	1.88	0.07
* modality * group	-5.74	4.67	-1.23	0.23
rIFG x CATI Cognitive Rigidity	0.20	0.10	1.97	0.06
* group	6.21	3.32	1.87	0.07
* modality	3.32	5.86	0.57	0.58
* modality * group	0.47	6.35	0.07	0.94
rIFG x CATI Masking	0.25	0.09	2.92	0.01
* group	8.06	2.71	2.97	0.005
* modality	12.40	4.43	2.80	0.009
* modality * group	-8.48	5.11	-1.66	0.10
rdlPFC x CATI Social Interactions	0.06	0.11	0.50	0.62
* group	0.78	3.41	0.23	0.82
* modality	-5.61	4.65	-1.21	0.24
* modality * group	5.30	5.20	1.02	0.31
rdlPFC x CATI Sensory Sensitivity	0.08	0.08	1.11	0.28
* group	1.48	2.49	0.59	0.56
* modality	-2.19	3.36	-0.65	0.52
* modality * group	1.73	3.86	0.45	0.66
rdlPFC x CATI Repetitive Behaviors	-0.01	0.11	-0.14	0.89
* group	-2.06	3.54	-0.58	0.56
* modality	-8.54	4.56	-1.88	0.07
* modality * group	7.61	5.13	1.48	0.15
rdlPFC x CATI Communication Difficulty	-0.02	0.09	-0.19	0.85
* group	-2.56	2.70	-0.95	0.35
* modality	-7.25	3.29	-2.20	0.04
* modality * group	7.75	4.04	1.92	0.06
rdlPFC x CATI Cognitive Rigidity	-0.00	0.12	-0.03	0.98
* group	-2.96	3.85	-0.77	0.45
* modality	-2.68	5.07	-0.53	0.60
* modality * group	5.39	5.71	0.94	0.35
rdlPFC x CATI Masking	0.14	0.10	1.43	0.16
* group	1.65	3.11	0.53	0.60
* modality	-2.17	4.13	-0.53	0.60
* modality * group	5.50	4.94	1.12	0.27
rTPJ x CATI Social Interactions	0.14	0.08	1.70	0.10
* group	3.32	2.45	1.35	0.18
* modality	6.49	4.45	1.46	0.16
* modality * group	-5.84	4.74	-1.23	0.23

Correlations	*β*	*SE*	*t*	*p*
rTPJ x CATI Sensory Sensitivity	0.06	0.06	1.01	0.32
* group	0.48	1.87	0.26	0.80
* modality	6.38	3.10	2.06	0.05
* modality * group	-3.84	3.38	-1.14	0.26
rTPJ x CATI Repetitive Behaviors	0.09	0.08	1.15	0.26
* group	0.67	2.61	0.26	0.80
* modality	5.27	4.73	1.12	0.28
* modality * group	-1.42	5.01	-0.28	0.78
rTPJ x CATI Communication Difficulty	0.09	0.07	1.35	0.19
* group	1.32	2.04	0.65	0.52
* modality	5.91	3.40	1.74	0.09
rTPJ x CATI Cognitive Rigidity	0.06	0.08	0.71	0.49
* group	1.81	2.72	0.67	0.51
* modality	4.30	4.87	0.88	0.39
rTPJ x CATI Masking	0.07	0.07	0.98	0.34
* group	1.49	2.36	0.63	0.53
* modality	4.47	4.01	1.11	0.28
lTPJ x CATI Social Interactions	0.08	0.09	0.86	0.40
* group	4.59	2.68	1.72	0.10
* modality	4.35	3.62	1.20	0.24
lTPJ x CATI Sensory Sensitivity	0.20	0.05	3.81	0.001
* group	5.95	1.72	3.47	0.01
* modality	4.99	2.58	1.93	0.06
lTPJ x CATI Repetitive Behaviors	0.08	0.09	0.89	0.38
* group	2.62	2.82	0.93	0.36
* modality	5.71	3.75	1.52	0.14
lTPJ x CATI Communication Difficulty	0.10	0.07	1.33	0.19
* group	1.76	2.16	0.81	0.42
* modality	4.64	2.80	1.66	0.11
lTPJ x CATI Cognitive Rigidity	-0.03	0.09	-0.36	0.72
* group	-1.00	2.94	-0.34	0.73
* modality	2.62	3.91	0.67	0.51
lTPJ x CATI Masking	0.19	0.07	2.57	0.02
* group	5.91	2.34	2.52	0.02
* modality	2.25	3.25	0.69	0.49

Discussion

Summary of results

Brain activation in empathy-related areas did not differ significantly between groups across all regions, except for the rTPJ. In this ROI it could be seen that activation was significantly lower in autistic participants than in non-autistic participants, indicating indeed differences in emotional processing between the groups. In addition, the interaction between group and modality was significant, suggesting that the type of modality was somehow influencing rTPJ activation when differentiating between groups, suggesting that the form of emotion communication does make a difference for autistic people. Across groups, the main effect of modality was significant for the mPFC, the rIFG and the lTPJ with higher activation during facial expressions than during drawing. There was no effect of modality for the rdlPFC and the rTPJ. For all regions (except rTPJ), interactions between group and modality were not significant, meaning that both together did not influence brain activation. To summarize, autistic and non-autistic participants showed similar brain activation in empathy-related regions during the expression of emotions. Hence, H1 (that empathy-related brain activation differs between autistic and non-autistic participants) was rejected, due to missing group differences.

Raw scores of the *thought of others* scale (measuring in the direction of cognitive empathy/ToM) were, contrary to the previous assumption, higher in the autistic group than in the non-autistic group. However, the inferential group comparison revealed no significant differences between groups. Generally, participants across groups indicated that they thought little about the other person during emotion expression. Consequently, H2 was rejected.

Correlations between empathy-related brain activation and *thought of others* scores were not significant across all ROIs. Also, group and modality did not have any influence on the relationship, so H3 (assuming a positive relationship) was rejected.

Looking at the SPF-IRI, raw scores were higher in the autistic group for the subscale *Personal Distress*, for the other subscales (*Fantasy, Perspective Taking, Empathic Concern*) the non-autistic group showed slightly higher values. Comparing raw scores of the CATI-R between the groups, the autistic group, as expected, had higher values in all subscales than the non-autistic group. There was a positive

correlation between mPFC activation and the subscale *Perspective Taking* with a stronger association in the autistic group. Regarding the CATI subscales, positive correlations were found for mPFC activation and *Sensory Sensitivity* and *Masking*, for rIFG activation and *Social Interactions* and *Masking* and for lTPJ activation and *Sensory Sensitivity* and *Masking*. However, the associations were all stronger in the non-autistic group. In addition, modality had a significant influence on rIFG activation and *Social interactions* and rIFG activation and *Masking*, with a stronger activation during the face condition.

Interpretation of H1 results

On a neurological level, autistic and non-autistic participants generally did not differ in the level of empathic activation, except for one ROI, revealed by the fNIRS analysis. There was no difference in activation in brain regions associated with affective or cognitive empathy. Surprisingly, there was also no significant difference in mPFC, rdlPFC and lTPJ activation which are highly involved in cognitive empathy. As reviewed above, if there were difficulties with empathy in autistic individuals, literature suggested especially difficulties with cognitive empathy and ToM. In our study, autistic participants had lower activation than non-autistic participants only in the rTPJ during emotion expression. This region is responsible for several cognitive and affective aspects of empathy like self-other discrimination, ToM, morale and emotional mimicry (Ahmad et al., 2021). A fMRI study found hypoactivation of the rTPJ in autistic participants when it comes to socially awkward situations (Pantelis et al., 2015). Even though participants were not directly observed during emotion expression in our study, especially looking towards a camera and being filmed could have evoked a feeling of insecurity. This may have had a stronger effect on the autistic group. Lombardo et al. (2011) examined in their fMRI study the role of the rTPJ in mentalizing and found that rTPJ activation in ASD lacks a specific function for mentalizing but appears as well in other tasks. In addition, their whole-brain analysis revealed that there was a hypoactivation of rTPJ in autistic participants during the mentalizing task compared to non-autistic participants. This finding matches with lower activation of the rTPJ in autistic participants in our study, since the tasks were connected to mentalizing because participants had to think how another person will perceive their emotional expression.

The mPFC, rIFG and lTPJ showed significantly more activation during the facial expression task than during the drawing task across groups. As explained above, the mPFC is strongly connected with the amygdala; both regions are part of the emotional brain. Since the amygdala is active when processing emotional facial expressions and the mPFC is highly involved in ToM, this may explain why mPFC activation was higher in the face modality. The rIFG is involved in facial mimicry and Carr et al. (2003) found indeed activation peaks for the rIFG during the imitation of facial expressions. Because the rIFG is responsible for affective empathy and participants were told before the tasks to feel into the emotion, it stands to reason that the neural process of emotion imitation is similar to the one of feeling into that emotion. Taken together, this may explain the higher activation of the rIFG in the face modality. The lTPJ is mainly involved in ToM (Doricchi et al., 2022) and a study of Gianotti et al. (2017) examined its association with the perception of direct gaze. Again, the task design with the camera in front of participants could have triggered activation of the lTPJ in a way that they might have had a feeling of being looked at. Interestingly, there was no effect of modality for rdlPFC and rTPJ activation, meaning drawing emotions evoked the same activation in these empathy-related areas as making facial expressions. This confirms that it is useful for studies as well as for interventions to include a variety of task modalities in addition to face-to-face interaction or facial expression recognition for examining or training empathy.

Summarizing these results, autistic participants did not show less empathy than non-autistic participants on a neurological level concerning cognitive and affective areas (except in the rTPJ).

Interpretation of H2 results

The *thought of others* scale should measure a component of cognitive empathy (ToM) in a form of subjective self-ratings. Since there was no significant group difference, it can be concluded that autistic and non-autistic participants did not differ in their self-rating of ToM during the expression of emotions. This finding contradicts prior study results claiming that autistic people tend to have lower cognitive empathy, including ToM. However, scores of thinking of others were generally quite low in both groups, so even the non-autistic group failed to show high levels of ToM. A meta-analysis of Murphy and Lilienfeld (2019) showed that only 1% of variance was explained by self-reported cognitive empathy in a behavioral task

performance. Hence, they criticized using self-report measures for assessing the level of ToM and other components of cognitive empathy. This finding could be one explanation for the non-significant result in our study.

Interpretation of H3 results

There were no significant correlations between empathy-related brain activation and the subjective self-rating scale of (cognitive) empathy in all ROIs, contradicting the hypothesis of the higher the scores, the higher the amount of brain activation. Neumann et al. (2015) summarized different approaches to measure empathy and revealed correlations between brain activation and well-known empathy questionnaires. Thus, the non-significant result in our study might be explained by the fact that the *thought of others* scale is not an official instrument for measuring cognitive empathy but was designed by the study team. Additionally, one question about ToM might be insufficient to reach significant correlations with brain activation; results may have looked different with a whole questionnaire.

Interpretation of exploratory results

Since the mPFC is highly involved in ToM, it is reasonable that there was a significant positive correlation with the IRI subscale *Perspective Taking*. This effect was stronger in the autistic group. Chen et al. (2022) argued in their study that higher activation of the mPFC in autistic people could indicate more effort during ToM tasks, meaning they need to increase activation in this region more than non-autistic people to reach the same level of perspective taking. Looking at the CATI-R scores, the subscales *Sensory Sensitivity* and *Masking* correlated most frequently with higher brain activation. These significant positive correlations were found in the mPFC, the rIFG and the lTPJ. Interestingly, the non-autistic group always showed stronger associations. Robertson and Simmons (2012) examined the relationship between sensory sensitivity and autistic traits in neurotypical people. They summarized that other factors than ASD contribute to autistic traits like personality, biological processes and how one interacts socially. They also found positive associations between high level autistic traits and atypical sensory sensitivity. This might explain the results of non-autistic participants showing high levels of autistic traits (sensory sensitivity as well as masking). The link with brain activation being stronger in this group could be justified by the fact that autistic individuals show way higher *Sensory*

Sensitivity and *Masking* scores, suggesting that on a neurological level, they are already used to this (e.g. masking in daily life) and hence responding with less brain activation than neurotypicals.

Additional notes on empathy and autism

Our study was one of many on empathy and no matter how scientific the study design was, they all investigated empathy in a certain context. Social cognitive processes like empathy and emotions highly depend on context, meaning how a specific situation is perceived, understood and interpreted (Chen & Liu, 2016). The authors named different examples for contexts influencing empathic responses like intergroup relationships, interpersonal closeness and causality of events in a situation. They stated that in addition, one's own knowledge and experience play a role in empathic reactions. Context influences empathy also on a neurological level in form of top-down- and bottom-up processes (Chen & Liu, 2016). With this in mind, general conclusions about the level of empathy or emotion recognition skills should be made cautiously. Not only context dependency, but also the complexity of both topics, empathy and ASD, makes it difficult to interpret study results and do research. It is not possible to answer all questions of interest at once, since there are so many definitions and types of empathy as well as a variety of autistic traits, differing from individual to individual. Therefore, researchers should a priori reflect on the limits of their studies and formulate precise research questions.

Several studies reviewed in this thesis used the term ‘high-functioning’ for participants with Asperger autism or average cognitive skills. In our study we also included only autistic people without cognitive disabilities. However, the term can be criticized:

> Rather than using ‘high-functioning’, ‘low-functioning’ or ‘profound autism’, we suggest that researchers, practitioners, and society more broadly use clear brief descriptions, for example ‘autistic person with intellectual disability’, ‘autistic person with minimal verbal language’, or ‘autistic person with extreme anxiety and a co-occurring physical condition’ (in line with Bottema-Beutel et al., 2021; Pukki et al., 2022). (Keating et al., 2023, p.424)

Keating et al. (2023) stated that calling an autistic person ‘high-functioning’ can lead to missing support while the word ‘low-functioning’ can lead to infantilization and the overlooking of strengths. In addition, for autistic people this label is associated with their ‘worth’ in a society created for neurotypicals (Keating et al., 2023). Summarizing this, in order to prevent inaccuracy and stigmatization, we should describe disabilities independently from autistic characteristics.

In a very recent paper by Long et al. (2025) findings for differences in ToM in autistic individuals were critically evaluated. One aspect they pointed out is that there is minimal shared understanding of what the ability of ToM contains, e.g. when it comes to representing mental states. The researchers claimed that theories and studies about ToM should distinguish between representing and correctly inferring mental states, since both are necessary for ToM. As mentioned before, many autistic people actually pass ToM tasks like classical false belief tests and even newer approaches to it. Long et al. (2025) criticized that these tests often not only assess ToM ability but emotion processing which is frequently reduced in autistic individuals due to accompanying alexithymia. Furthermore, fMRI studies showed mixed results concerning differences in activation in regions associated with mental state representation and even when finding significant differences, it remains hard to interpret them (Long et al., 2025). According to the researchers, the reason for that lies in the generally altered structure and neurovascular coupling in autistic brains compared to neurotypical ones. They concluded that there is a need for new ToM tasks targeting the described issues. All in all, universal statements about impaired ToM in autism should be questioned and studies should define which components of ToM were measured exactly.

Study limits and benefits

One major limit of the current study was the brain-imaging method fNIRS in a way that it could only reach cortical areas. However, in addition to the targeted ROIs in our study, subcortical areas like the amygdala and the cingulate cortex play a crucial role in empathic brain activation (Marsh, 2018). Both regions are associated with affective empathy and empathic concern, according to the author. Including these regions in the study would have been helpful to understand the process of feeling into a certain emotion better. Despite the limited range of movement, future

studies might consider examining emotion processing and empathy in ASD using fMRI for a whole-brain analysis.

As mentioned earlier, this study was part of a larger study on emotion expression, as well as emotion recognition and empathy-related brain activation in autistic individuals. Due to time limits, my project took place only in the expression part, hence the link between emotion expression- and recognition is missing in my thesis. The actual goal of the whole study to investigate the double empathy problem and detect possible similarities of brain activation in autistic and non-autistic participants during both, expression and recognition, could not be fulfilled. Instead, the bridge between emotion expression and empathy was built theoretically and a scale measuring in the direction of ToM was included in the current project.

Benefits of the study were the new art paradigm which did not only create interest in participating but also might have had advantages for the autistic group. As already discussed, different forms of communication might be beneficial for autistic people, even concerning the communication of emotions. Additionally, with the feedback from autistic participants on the study design and interviewing them about their own life experiences, we made sure that they felt safe during the study and that their voices were heard. This contributed to a kind of research that values neurodivergence and sees the person behind a diagnosis or a certain label.

Future directions

Future research could extend the current study by discriminating between emotions. For other research questions, it might be interesting how empathy-related brain activation differs between surprise, happiness, sadness, anger or fear. There might be differences for certain emotions between autistic and non-autistic individuals.

Also, it would be worthwhile to investigate empathic activation during real life interactions of autistic and non-autistic people. With fNIRS it would be possible to create a dynamic social setting that allows to generalize study results even further. Participants could move quite freely while interacting with another person (e.g. autistic-autistic, autistic-non-autistic) in tasks that trigger cognitive or affective empathic reactions.

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Index of illustrations

Appendix

Abstract:

The study investigated the relationship between emotion expression and empathy-related brain activation in autistic and non-autistic individuals (n = 30 per group). As previous studies have suggested a link between autism and reduced cognitive empathy (specifically Theory of Mind, ToM) during emotion recognition tasks, the current project should examine if this is also the case during emotion expression. Since research showed mixed findings for atypical affective empathy in autistic individuals, activation in regions for cognitive empathy as well as affective empathy was compared between the groups during a facial expression task and a new art paradigm. In addition, it was tested if self-rating scores of thinking of others during the tasks (measuring in the direction of ToM) differed between the groups and if this subjective self-measurement was correlated with empathic brain activation. The fNIRS analysis revealed no significant differences in empathy-related brain activation between autistic and non-autistic participants in the medial prefrontal cortex, the right inferior frontal gyrus, the right dorsolateral prefrontal cortex and the left temporoparietal junction. Only in the right temporoparietal junction the autistic group showed less activation. Furthermore, no significant differences were found for the self-rating scores of thinking of others between the groups, contradicting the a priori hypothesis that scores may be lower in the autistic group. Additionally, empathy-related brain activation and self-rating scores did not significantly correlate with each other. These findings disprove common stereotypes about lower empathy levels in autistic people. The study contributed to neurodivergence appreciating research by obtaining feedback and interviewing autistic participants.

Keywords: ASD, autism, cognitive empathy, affective empathy, neuroimaging, emotion expression, emotion recognition

Zusammenfassung:
Die Studie untersuchte die Beziehung zwischen Emotionsausdruck und empathiebezogener Gehirnaktivierung bei autistischen und nicht-autistischen Personen (n = 30 pro Gruppe). Da frühere Studien einen Zusammenhang zwischen Autismus und verminderter kognitiver Empathie (insbesondere Theory of Mind, ToM) bei Aufgaben zur Erkennung von Emotionen nahegelegt haben, sollte in diesem Projekt untersucht werden, ob dies auch beim Ausdruck von Emotionen der Fall ist. Da die Forschung gemischte Ergebnisse für atypische affektive Empathie bei autistischen Personen gezeigt hat, wurde die Aktivierung in Regionen für kognitive Empathie sowie für affektive Empathie während einem Gesichtsausdruck-Task und einem neuen Kunst Paradigma zwischen den Gruppen verglichen. Darüber hinaus wurde getestet, ob sich die Selbsteinschätzung des Denkens an andere während der Aufgaben (als Maß für ToM) zwischen den Gruppen unterscheidet und ob diese subjektive Selbsteinschätzung mit der empathischen Hirnaktivierung korreliert ist.
Die fNIRS-Analyse ergab keine signifikanten Unterschiede in der empathiebezogenen Hirnaktivierung zwischen autistischen und nicht-autistischen Teilnehmer*innen im medialen präfrontalen Kortex, dem rechten inferioren frontalen Gyrus, dem rechten dorsolateralen präfrontalen Kortex und der linken temporoparietalen Verbindung. Nur im rechten temporoparietalen Knotenpunkt zeigte die autistische Gruppe eine geringere Aktivierung. Außerdem wurden keine signifikanten Unterschiede zwischen den Gruppen bei der Selbsteinschätzung des Denkens an andere festgestellt, was der a priori-Hypothese widerspricht, dass die Werte in der autistischen Gruppe niedriger sein könnten. Darüber hinaus korrelierten die empathiebezogene Hirnaktivierung und die Selbstbewertungswerte nicht signifikant miteinander. Diese Ergebnisse widerlegen gängige Vorurteile über geringere Empathiefähigkeit bei Autist*innen. Die Studie trug zur wertschätzenden Erforschung von Neurodivergenz bei, indem wir Feedback einholten und autistische Teilnehmer*innen interviewten.