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ORIGINAL PAPER

Preserved Self-other Distinction During Empathy in Autism is Linked to Network Integrity of Right Supramarginal Gyrus

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Abstract Autism spectrum disorder (ASD) shows deficits in self-other distinction during theory of mind (ToM). Here we investigated whether ASD patients also show difficulties in self-other distinction during empathy and if potential deficits are linked to dysfunctional resting-state connectivity patterns. In a first study, ASD patients and controls performed an emotional egocentricity paradigm and a ToM task. In the second study, resting-state connectivity of right temporo-parietal junction and right supramarginal gyrus (rSMG) were analysed using a large-scale fMRI data set. ASD patients exhibited deficient ToM but normal emotional egocentricity, which was paralleled by reduced connectivity of regions of the ToM network and unimpaired rSMG network connectivity. These results suggest spared self-other distinction during empathy and an intact rSMG network in ASD.

Keywords Autism spectrum disorder · Self-other distinction · Empathy · Emotional egocentricity · Resting-state functional connectivity · Right supramarginal gyrus

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Introduction

Autism spectrum disorder (ASD) is an early-onset neurodevelopmental disorder characterized by impairments in social communication, interaction, and stereotyped or repetitive behaviors and interests (Association Psychiatric Association 2013). Already in his original paper, Asperger (1944) described the children he studied as being "egocentric to the extreme". Consequently, one of the most consistently reported social cognition deficits in ASD has been in theory of mind (ToM) (Baron-Cohen et al. 1985; Castelli et al. 2002; Frith and Frith 2012; Happé 1994), the socio-cognitive ability to understand the mental states of others, such as beliefs and intentions (Premack and Woodruff 1978). When engaging in ToM tasks, higher egocentrism of individuals with ASD compared to non-autistic individuals is for example evidenced by their increased difficulty in passing false-belief tasks (Baron-Cohen et al. 1985; Begeer et al. 2012; Senju et al. 2010; Senju et al. 2009).

It has been proposed that the underlying problem in ToM and in particular false-belief understanding for individuals with ASD is difficulties in differentiating between perspectives of self and other, also known as self-other distinction (Lombardo and Baron-Cohen 2011). Human interpersonal understanding often relies on mechanisms of self-projection and simulation (Bastiaansen et al. 2009; Brass et al. 2009; Decety and Lamm 2007; Gallese 2001, 2007; Gallese and Goldman 1998; Mitchell 2009; Nickerson 2001; Silani et al. 2013; Singer 2012; Singer et al. 2004; Steinbeis et al. 2014; Van Boven and Loewenstein 2003). However, such projection mechanisms fail in making sense of other's mental states in situations where mental states of self and other clearly differ, such as in typical false-belief tasks, eventually leading to egocentrically biased judgments (Birch and Bloom 2007; Pronin 2008; Royzman et al. 2003). To avoid egocentrically biased judgments, a mechanism differentiating between self and other perspectives has to be in place. In sum, context appropriate differentiation of self and other might be at the core of many social cognition deficits displayed in ASD.

While self-other distinction during ToM seems to be crucial, recent evidence has shown that self-other distinction is of equal importance during empathic judgments (Silani et al. 2013; Steinbeis et al. 2014). Empathy involves sharing the emotional state of others while being aware that the other is the source of that state (Singer and Lamm 2009), requiring emotional self-other distinction particularly in cases, where emotional states of self and other are incongruent. Failure of self-other distinction during empathy results in egocentric emotional responses, e.g. failure to share sadness of a friend when being in a good mood. While deficits in ToM in ASD have been consistently reported, it remains less clear whether individuals with ASD also have difficulties in empathy, particularly when emotional states between oneself and others differ in valence. There is indeed good evidence that empathy may be intact in ASD (Bird et al. 2010; Hadjikhani et al. 2014; Jones et al. 2010; Lockwood et al. 2013; Rogers et al. 2007). While some studies have reported lower empathy in ASD (Dapretto et al. 2005; Minio-Paluello et al. 2009), there is increasing evidence that such deficits might arise more from comorbid alexithymia, i.e. inability to identify and describe one's own emotions, as opposed to autism-specific deficits per se (Bird and Cook 2013). As empathy might thus be intact in ASD, it is still an open question if, in case of incongruent emotional states, individuals with ASD would display increased emotional egocentricity, indicating poor self-other distinction also in the affective domain. In Study 1 our main aim was therefore to test for increased emotional egocentricity bias (EEB) in ASD compared to normal controls. We used the EEB touch-paradigm (ETOP, Silani et al. 2013), in which emotions are induced via tactile stimulation. The ETOP allows to measure pure empathic judgments under different conditions, when emotional states of self and other are congruent or incongruent, thus assessing simple empathic judgments, but also self-other distinction during empathic judgments.

A key brain region adequately suited for self-other distinction in the cognitive domain, being a hub of both interoceptive and exteroceptive information pathways is the so-called temporo-parietal junction (TPJ). TPJ has shown to be consistently recruited during ToM (Aichhorn et al. 2006; Decety and Lamm 2007; Ramsey et al. 2013; Sommer et al. 2007). In particular the right TPJ, has been suggested to play a major role during ToM, especially when self-other distinction is required (Aichhorn et al. 2006; Sommer et al. 2007). It has however been argued that rTPJ plays a more general role in self-other distinction in the cognitive as well as motor domain, based on results from meta analyses and single studies, showing a relation between the inhibition of spontaneous imitation tendencies (i.e. self-other distinction in the motor domain) and ToM abilities (Santiesteban et al. 2012b; Spengler et al. 2009, 2010). A recent study using transcranial direct current stimulation (tDCS) of rTPJ provided strong evidence that rTPJ is causally involved in differentiating self and other during imitation inhibition and ToM (Santiesteban et al. 2012a). Thus rTPJ might help to differentiate self and other perspectives during ToM but also during imitation. In the case of ASD, structural and functional abnormalities of rTPJ have been linked to social cognition deficits (Castelli et al. 2002; David et al. 2014; Kana et al. 2012; Lombardo et al. 2011; Mueller et al. 2013; Pitskel et al. 2011; Washington et al. 2013). Conversely, recent research suggests that self-other distinction in the emotional domain may be subserved by brain regions that are part of the temporo-parietal cortex, but slightly more anterior to TPJ, namely the right supramarginal gyrus (rSMG). Thus, using a design structurally analogous to classic false-belief tests of ToM in which another's belief will be counter to one's own tasks but substituting beliefs for emotional experience a study by Silani et al. (2013) demonstrated that adults when judging the emotional state of another person incongruent to their own provide judgments skewed in favour of their own experience, something also known as EEB. Crucially, the rSMG was functionally implicated in overcoming EEB. Peaks of this activation were distinct from other subregions of temporo-parietal cortex involved in ToM. In line with these findings, a study by Steinbeis et al. (2014) showed in a resting-state connectivity analysis that rSMG showed stronger functional connectivity to regions of the empathy network, such as the middle cingulate cortex, bilateral anterior insulae (AI), extending to inferior frontal gyrus (IFG) and bilateral dorsolateral prefrontal cortex (DLPFC), while rTPJ showed stronger functional connectivity to nodes of the ToM network, including the medial prefrontal cortex (MPFC) and the precuneus. Taken together these findings corroborate the hypothesis that the broader area usually referred to as temporal parietal cortex consists of important subdivisions that in turn might subserve different functions in the context of social cognition respectively such as self-other distinction during empathy on the one hand as compared to ToM on the other. It has to be explicitly noted here that while rTPJ and rSMG seem to have crucial functions during social cognitive processes as described above, both regions are involved in numerous other non-social cognitive functions (Carter and Huettel 2013; Geng and Mangun 2011; Menon et al. 1997; Stoeckel et al. 2009).

Nonetheless, the aim of our complementary Study 2 was to test for differences in the associated brain regions supporting the function of self-other distinction in the emotional domain on the one hand and in the cognitive domain on the other. In order to do so we conducted a resting-state analysis using previously identified brain regions critically involved in emotional egocentricity and ToM as seed regions. Because no resting-state data were available for the same set of participants as in Study 1, we analyzed resting-state functional connectivity data in an independent large multi-center sample of individuals with ASD and matched healthy controls; seeding from rSMG, a region directly implicated in overcoming emotional egocentricity and rTPJ, a region commonly shown to play a crucial role during ToM.

To sum up we aimed to investigate whether individuals with ASD relative to healthy controls would show normal emotional egocentricity, differentiating emotional states of self and other during empathic judgments, associated with intact functioning of the rSMG-related brain network. In contrast we hypothesized that individuals with ASD relative to healthy controls would exhibit known deficits in ToM possibly linked to problems differentiating self and other perspectives in the cognitive domain related to aberrant functioning of the rTPJ-related brain network.

Methods

Participants

Behavioral Sample

For Study 1, 32 with ASD and 26 healthy controls were recruited. In the case of the ASD patients, 4 participants showed abnormal emotional responses to the stimuli (e.g., rated positive stimuli as negative and vice versa) and were excluded from further analysis. 3 ASD patients and 1 healthy control participant were later excluded, showing abnormal emotional egocentricity, with ratings above two standard deviations. Subsequently the final sample included 25 adults with ASD and 25 IQ and gender-matched neurotypical participants (see Table 1). ASD patients were relatively high-functioning. ASD participants were recruited through the outpatient clinic of the Charité University Medicine Berlin, or were referred to us by specialized clinicians. Diagnoses according to DSM-IV criteria (American Psychiatric Association 2000) for Asperger disorder and autistic disorder without intellectual disabilities were based on expert clinical opinion, the Autism Diagnostic Observation Schedule (ADOS, Lord et al. 2000), and the Autism Diagnostic Interview-Revised (ADI-R, Lord et al. 1994), if parental information was available

Table 1 Demographic and clinical characteristics of the behavioral sample (Study 1)

Behavioral sample	ASD M (SD)	HC M (SD)	ASD versus HC
Sample size	25	25	
Gender	18 Males	18 Males	
Age	32.6 (8.5)	32.4 (8.5)	$p = .960$
Full IQ	115.8 (9.1)	112.8 (8.0)	$p = .246$
AQ	36.9 (8.0)	13.7 (4.7)	$p < .001$***
TAS-26	54.2 (10.0)	37.4 (7.8)	$p < .001$***
ADOS	11.0 (3.8)		

Statistics applied: independent *t* test

* $p < .05$; ** $p < .01$; *** $p < .001$

(n = 14). ADOS—scores are used as a measure for symptom severity throughout this article. Healthy control (HC) participants with no history of psychiatric or neurological disorders were recruited by public notices and from project databases of the Freie Universität Berlin and the Max Planck Institute for Human Cognitive and Brain Sciences Leipzig, Germany. Crystalline and fluid intelligence levels were estimated by means of a verbal intelligence German vocabulary test/Mehrfach-Wortschatz-Test (MWT, Lehrl et al. 1995) and a strategic thinking test (LPS, subscale 4, Horn 1962) respectively, and combined to yield a full scale IQ (FIQ). Autistic traits and Alexithymia were assessed in both groups using the Autism Quotient and the Toronto Alexithymia Scale (TAS-26, Taylor et al. 1985), respectively. Participants LPS, subscale 4 gave informed consent prior to participation and received payment. The study was approved by the ethics committee of the German Society for Psychology (DGPs).

Imaging Sample

For Study 2, we studied a subsample of 163 (84 ASDs, 79 healthy controls) male participants from the Autism Brain Imaging Data Exchange (ABIDE) database, a publically available multi-center aggregate of previously collected structural and functional MRI data from individuals with ASD and healthy controls (see http://fcon_1000.projects.nitrc.org/indi/abide/). ASD patients were relatively high-functioning. After the exclusion of individuals with too much head-motion the final sample consisted of 155 male participants (78 ASDs, 77 healthy controls, see Table 2 and "Methods"). ASD diagnoses according to DSM-IV criteria (American Psychiatric Association 2000) were based on expert clinical opinion, the Autism Diagnostic Observation Schedule, ADOS (n = 63) (Lord et al. 2000), and/or the Autism Diagnostic Interview-Revised, ADI-R (n = 27)

Table 2 Demographic and clinical characteristics of the fMRI sample (Study 2)

fMRI sample	ASD M (SD)	HC M (SD)	ASD versus HC
Sample size	78	77	
Gender	All males	All males	
Age	25.4 (6.9)	25.5 (6.1)	$p = .956$
Full IQ	108.1 (16.3)	115.6 (11.9)	$p = .001$**
ADOS (N = 63)	12.6 (3.9)		

Statistics applied: independent *t* test

* $p < .05$; ** $p < .01$; *** $p < .001$

(Lord et al. 1994). Note that for the individuals of the behavioral sample no resting-state scans were available. To further inform the behavioral results, however, we chose to analyze resting-state data from the ABIDE database. The selected sample was chosen to be within the same age range as the behavioral sample (20–55 years), and ASD participants of both samples did not differ in terms of symptom severity as measured through the ADOS. Within the imaging sample, individuals with ASD only differed in terms of full scale IQ to the healthy controls. Subsequently, full scale IQ was used as a covariate of no interest in the resting-state analysis.

The sample selected for this study consisted of male participants from 7 different sites: (1) California Institute of Technology (n = 26, 13 ASDs, 13 healthy controls); (2) University of Leuven (n = 27, 13 ASDs, 14 healthy controls); (3) Olin, Institute of Living at Hartford Hospital (n = 10, 5 ASDs, 5 healthy controls); (4) University of Pittsburgh, School of Medicine (n = 21, 10 ASDs, 11 healthy controls); (5) Trinity Centre for Health Sciences (n = 19, 9 ASDs, 10 healthy controls); (6) University of Utah, School of Medicine (n = 5, 5 healthy controls) and (7) University of Michigan (n = 55, 34 ASDs, 21 healthy controls).

EEB Touch-Paradigm (ETOP)

The design and procedure of this paradigm was identical to that reported in Silani et al. (2013). Participants unknown to each other were assigned pairwise to an experimental session. Sitting back to back in front of a touch screen (800 × 600 pixels resolution, 15 in. screen, viewing distance ~40 cm) they were asked to rate the pleasantness or unpleasantness of tactile stimulation of their left palm hidden behind a curtain preventing them to observe the different stimulation materials. Before the start, participants were familiarized with the rating scale which was presented on the touch screen and performed 6 practice trials for each experimental condition. Participants started with the individual conditions instructed to either judge the pleasantness of their own touch stimulation (individual self condition) or the pleasantness of the tactile stimulation for the other person (individual other condition). The individual conditions were blocked and counterbalanced. In the individual self condition a picture (size 336 × 336 pixels) appeared on the screen accompanied by a corresponding tactile stimulation of the participant's left hand at 1 Hz for 3000 ms (e.g. a picture of a rose while the participant was touched by a silky object). Immediately after the stimulation phase participants judged the pleasantness or unpleasantness of the tactile experience by using the touch screen rating scale (ranging from −10 to 10), within 3000 ms response time. In the individual other condition, the trial structure remained the same, but the participant did not receive any tactile stimulation. Instead, he was instructed to judge the pleasantness of the tactile experience for the other participant based on the picture indicating what tactile stimulation the other participant received. Each run consisted of 30 pseudo-randomized trials, with 10 pleasant, 10 neutral and 10 unpleasant visuo-tactile stimuli. This resulted in a three-factorial mixed design with the two within-subjects factors *target* (self, other judgment) and *valence* (pleasant, neutral and unpleasant stimulation) and the between-subjects factor *group* (healthy controls and ASD).

In the following simultaneous conditions both participants in the room received tactile stimulation simultaneously, and were instructed to either judge the pleasantness of their own tactile experience (simultaneous self condition) or judge the pleasantness of the tactile experience for the other person (simultaneous other condition). The simultaneous conditions were blocked and counterbalanced. In these conditions two pictures appeared on the screen, while the left picture with the label "Self" on top corresponded to the tactile stimulation the participant received, the right picture with the label "Other" corresponded to the touch the other person received. The touch experiences of the two participants could be either affectively congruent (e.g. both touched by pleasant materials, e.g. silk and fur) or incongruent (e.g. one gets touched by a pleasant, the other by an unpleasant material, e.g. silk and rubber spider). The EEB was defined as the difference between ratings in incongruent and congruent trials when judging the other, as compared to the difference when judging one's own feelings. In this way, the simultaneous self condition served as a control for general perceptual or cognitive confounds—such as visual and affective stimulus comparison, detection of incongruency, or overcoming general response conflict. For the simultaneous conditions each run consisted of 40 pseudo-randomized trials, with 20 pleasant (10 congruent/10 incongruent) and 20 unpleasant

(10 congruent/10 incongruent) visuo-tactile stimuli. This resulted in a four-factorial mixed design with the three within-subjects factors *target* (self, other judgment), *valence* (pleasant, unpleasant stimulation), and *congruence* (congruent, incongruent stimulation of participant and other) and the between-subjects factor *group* (healthy controls and ASD). The significant triple interaction of *target* × *congruency* × *group* would be indicative of a significant group difference in the size of the EEB. Data analysis was performed using the IBM SPSS statistics software, version 19.0.

Movie for the Assessment of Social Cognition (MASC)

During the MASC participants are watching a 15 min movie about four characters spending an evening together, which is stopped 45 times for questions about the actors' mental states. Correct responses were computed to a total score. The MASC has proven internal consistency, sensitivity, stability over time (Dziobek et al. 2006) and has been used with different patient populations (Montag et al. 2010, 2011; Ritter et al. 2011).

Resting-State Connectivity Analysis

Data were processed using the data processing assistant for the resting-state fMRI toolbox (DPARSF) (DPARSF, Song et al. 2011) for Matlab. The toolbox is based on the Statistical Parametric Mapping toolbox (SPM8, http://www.fil.ion.ucl.ac.uk/spm). In brief, preprocessing discarded the first 10 volumes, performed slice time correction, motion correction and realignment, and co-registered the functional time series to the corresponding T1-weighted MRI. Images underwent DARTEL-based segmentation and registration, followed by nuisance covariate regression to remove effects of average WM and CSF signal, as well as six motion parameters (three translations and three rotations). To deal more thoroughly with possible differential motion artifacts in our samples we included the scrubbing approach advocated by Power et al. (2012), which models bad time points [based on the framewise displacement threshold, FD (Power), of .5 mm or higher; together with one time point before and one time point after each such time point] as separate regressors during the nuisance covariate correction. Time series were band-pass filtered to be within the .01 and .08 Hz band (Satterthwaite et al. 2013), normalized to MNI space, resampled to 3 mm voxels, and spatially smoothed using a 8 mm full-width-at-half-maximum (FWHM) isotropic Gaussian kernel. In the resting-state sample, 6/84 individuals with ASD and 2/79 of healthy controls showed head-motion beyond 3 mm translation or 3 degrees of rotation and were excluded from all further analysis. Functional connectivity maps were generated for both rSMG and rTPJ, based on the overlap of activations from two separate fMRI experiments (Silani et al. 2013) using the ETOP ($MNI_{xyz} = 65, -37, 33$) and a coordinate-based meta-analysis of rTPJ-activation in ToM studies by Mar (2011) respectively ($MNI_{xyz} = 51, -52, 21$). The rSMG region from these fMRI experiments was based on the contrast (Other Judgment: Incongruent > Congruent) > (Self Judgment: Incongruent > Congruent). The rTPJ region was based on meta-analytic activation of rTPJ in story-based and nonstory based ToM studies. Results of this analysis were provided as a NIfTI File thresholded at $p = .01$, FDR-corrected. Both regions the rSMG and rTPJ region were adjacent to each other but spatially non-overlapping (Fig. 1). Functional connectivity was calculated as the time series correlation between the mean time series of the seed region and the time series of all brain voxels. Time-series correlation coefficients underwent a Fisher r-to-z transformation to render the data more normally distributed. Group differences in functional connectivity were analyzed with SPM8 using random-effects models, assessing interactions between within-subject difference of rTPJ to rSMG connectivity and the group. For ASD patients whole-brain correlations were run using the ADOS social score, as a measure of symptom severity. The number of sites, age and FIQ were included in the model as covariates of no interest. Using Monte Carlo simulation correcting for multiple comparison cluster size corrected results are reported (voxel-wise p value of .01 combined with an extent threshold of 77 voxels corresponded to cluster-wise family-wise error rate of .05).

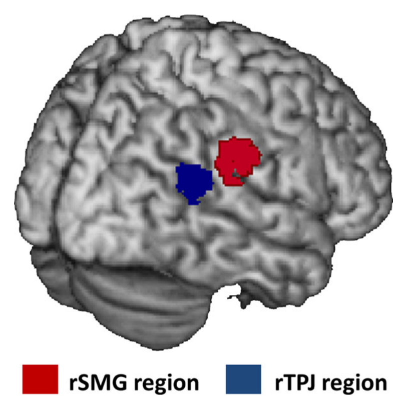

Fig. 1 Display of adjacent but non-overlapping rSMG and rTPJ regions used for functional connectivity analysis in a large independent sample of individuals with ASD and healthy controls. The rSMG region consists of an overlap of activations of two fMRI experiments ($MNI_{xyz} = 65, -37, 33$) looking at the neuronal basis of the EEB using the ETOP (Silani et al. 2013). The rTPJ region ($MNI_{xyz} = 51, -52, 21$) was taken from a meta-analytic activation during ToM (Mar 2011)

Results

EEB Touch-Paradigm (ETOP)

Individual Conditions

Investigating whether the emotion induction worked for both groups the individual conditions were analysed with an analysis of variance (ANOVA) on the affective ratings with target (self vs. other) and valence (positive, neutral, negative) as within-subjects factors and group (ASD vs. healthy controls) as between-subjects factor.

The results revealed a significant main effect of valence ($F_{1,96} = 221.48$, $p < .001$, $\eta^2_p = .822$), and target ($F_{1,48} = 8.33$, $p < .001$, $\eta^2_p = .148$), as well as a significant target by valence ($F_{1,96} = 13.09$, $p < .001$, $\eta^2_p = .214$) and valence by group interaction ($F_{1,96} = 6.40$, $p < .001$, $\eta^2_p = .118$). There was no significant main effect of group or further significant interaction with group (Fs < 1.84, ps $> .164$). Post-hoc tests showed that the ASD group rated the negative and positive emotions less intense for self and other. After controlling for alexithymia, no group differences remained, indicating equally effective emotion induction for both groups by means of visuo-tactile stimulation.

Simultaneous Conditions

To investigate whether ASD participants and healthy controls display different emotional egocentricity an ANOVA on the affective ratings with target, congruency and valence as within-subjects factors and group as between-subjects factor was performed.

The results showed significant main effects of target ($F_{1,48} = 11.14$, $p = .002$, $\eta^2_p = .188$), valence ($F_{1,48} = 16.92$, $p < .001$, $\eta^2_p = .261$), and group ($F_{1,48} = 6.10$, $p = .013$, $\eta^2_p = .113$), and significant interactions of target and valence ($F_{1,48} = 9.57$, $p = .003$, $\eta^2_p = .166$), and congruency, emotion and group ($F_{1,48} = 4.70$, $p = .035$, $\eta^2_p = .089$). Importantly however, while there was a significant target by congruency interaction ($F_{1,48} = 10.58$, $p = .002$, $\eta^2_p = .181$), there was no significant interaction of target, congruency and group, suggesting no group difference in emotional egocentricity ($F_{1,48} = .17$, $p = .684$, $\eta^2_p = .003$). Both ASD participants and healthy controls however showed a significant EEB ($F_{1,24} = 5.56$, $p = .027$, $\eta^2_p = .188$; $F_{1,24} = 5.27$, $p = .031$, $\eta^2_p = .180$). Including the TAS score as a covariate in the model revealed no significant interaction of target, congruency and group ($F_{1,48} = .02$, $p = .882$, $\eta^2_p = .00047$), ruling out that any possible differences in EEB between healthy controls and ASD participants was being masked by alexithymia in the ASD group. In fact, the EEB of ASD participants and healthy controls was comparable in size ($t_{48} = .41$, $p = .684$, 95 % CI $-.40 \leq \mu1–\mu2 \leq .60$, $d = .11$, 95 % CI $-.44 \leq \Delta \geq .67$) (ASD = .35, healthy controls = .45, Fig. 2a). The EBB in ASD participants was unrelated to symptom severity ($r = -.20$, $p = .365$).

Movie for the Assessment of Social Cognition (MASC)

ASD participants showed significantly lower scores on the MASC ($t_{46} = 2.32$, $p = .025$; Fig. 2b) Scores on the MASC were negatively related to symptom severity as measured by the ADOS ($r = -.69$, $p = .001$). Additionally the MASC score were unrelated to the EEB for ASD patients ($r = .10$, $p = .648$) and healthy controls ($r = -.08$, $p = .712$).

Resting-State Connectivity Analysis

We directly compared the voxel-wise connectivity strength of both regions within subjects combining individuals with ASD and healthy controls. The rSMG showed marked connectivity patterns relative to rTPJ to the lSMG, the bilateral AI extending into IFG, the medial cingulate cortex (MCC) and bilateral dorsal lateral prefrontal cortex (DLPFC). In contrast rTPJ showed significantly stronger functional connectivity compared to rSMG, to the left TPJ, precuneus, posterior cingulate cortex (PCC), and MPFC (see Fig. 3; Table 3). These results of different connectivity profiles for rSMG and rTPJ converge nicely with other parcellations of the temporo-parietal region (Bzdok et al. 2013; Mars et al. 2012). To investigate possible group differences in functional connectivity in the rSMG or the rTPJ network between ASD participants and healthy controls, we tested for an interaction between connectivity difference and group. Findings revealed significant differences in rTPJ (FWE < .05, Fig. 4a) but not rSMG (Fig. 4b) functional connectivity between the two groups (see also Table 3). Individuals with ASD displayed significantly reduced functional connectivity from rTPJ to lTPJ, precuneus, PCC, and MPFC. In addition a regression analysis within the rTPJ network using ADOS social interaction scores revealed that with increasing rTPJ-PCC coupling symptom severity decreased within the ASD sample (Fig. 4c).

Discussion

Previous studies consistently report findings on ToM deficits in ASD, presumably based on altered processes of self-other distinction in the cognitive domain (e.g. Lombardo and Baron-Cohen 2010; Lombardo et al. 2010). Evidence for

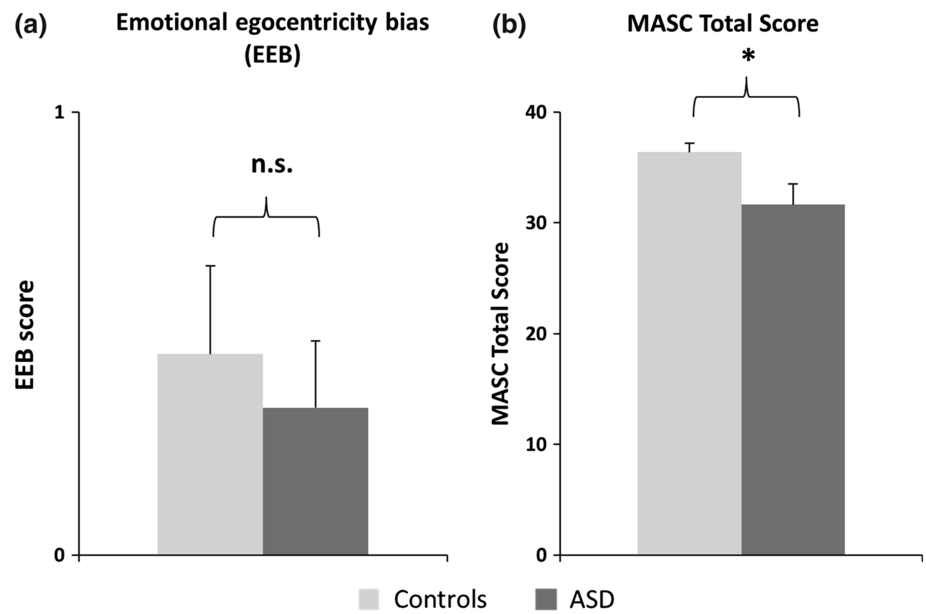

Fig. 2 **a** Display of the emotional egocentricity bias (EEB). Both groups displayed a significant EEB but the size of the EEB was similar for individuals with ASD and healthy controls, suggesting intact self-other distinction during empathic judgments in ASD. **b** MASC Total Score. As expected healthy controls showed a significantly greater MASC score than individuals with ASD, suggesting deficient ToM in ASD

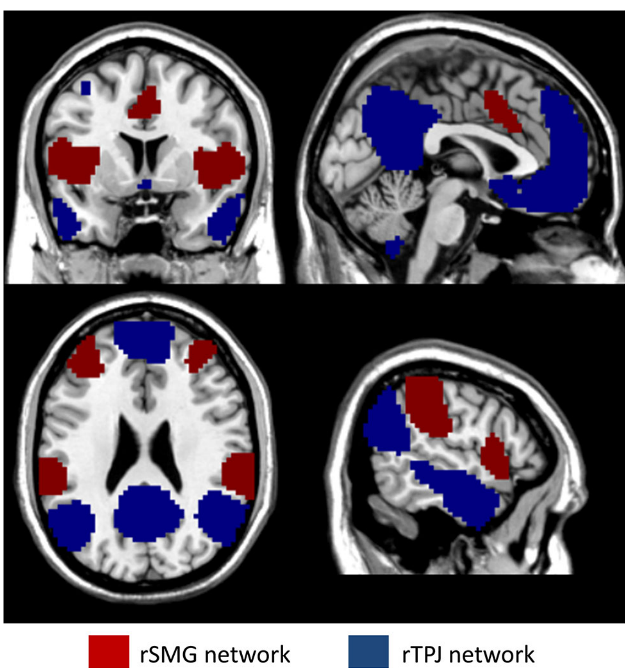

Fig. 3 Display of the rSMG and the rTPJ network. Seed-based resting-state functional connectivity analysis revealed marked divergent connectivity profiles for rSMG and rTPJ (FWE < .05). RSMG shows greater functional coupling compared to rTPJ with lSMG, bilateral AI, IFG, DLPFC and MCC. Whereas rTPJ shows greater functional coupling compared to rSMG with lTPJ, MPFC, PCC, precuneus, temporal poles

comparable deficits in the domain of empathy in ASD has remained inconsistent, thus demanding a more detailed investigation. In Study 1 we focused on behaviorally investigating a more specific socio-affective ability, namely affective self-other distinction as assessed by the ability to overcome emotional egocentricity during empathic judgments in individuals with ASD using the ETOP (Silani et al. 2013), while also assessing ToM abilities with an established task (Dziobek et al. 2006). In addition, Study 2 aimed to shed light on the integrity of neuronal networks associated with overcoming egocentricity (through self-other distinction) during empathy and ToM in ASD.

Study 1 found in line with the literature, that individuals with ASD show deficits in ToM (Baron-Cohen et al. 1985, 2001; Castelli et al. 2002; Dziobek et al. 2006; Happé 1994; Klin 2000). However, using the previously established ETOP, both, ASD participants and healthy controls, showed a significant EEB, comparable in size, suggesting no enhanced emotional egocentricity in ASD, and implicating relatively intact self-other distinction during empathic judgments. Thus, according to these results, individuals with ASD are not more prone to project their own feelings onto others/bias their perception of feelings of others towards their own feelings than neurotypical individuals. Additionally, in ASD participants, ToM abilities were significantly related to symptom severity, while the EEB was not. These behavioral findings suggest that individuals with ASD might have specific deficits in ToM but not in self-other distinction during empathic judgments, extending previous findings of intact empathy, that this is also the case even when emotional states between oneself and others are incongruent. The finding of an equally sized EEB in ASD as compared to healthy controls suggests that a more detailed account of social cognition deficits in ASD is required, as is also suggested by previous findings of spared socio-affective functions (Bird et al. 2010; Hadjikhani et al. 2014; Jones et al. 2010; Lockwood et al. 2013; Rogers et al. 2007).

Table 3 Peak coordinates from significant clusters observed in all analyses in Study 2

Anatomical label	MNI coordinates			Cluster size	T value
	x	y	z		
rSMG > rTPJ					
Right SMG	63	−36	33	609	42.92
Left SMG	−63	−39	36	426	16.87
Right AI/IFG	48	12	0	869	10.34
Left DLPFC	−42	45	30	326	9.95
Left AI/IFG	−33	18	9	478	9.91
MCC	6	18	30	226	6.55
Right cingulate gyrus	12	−27	42	14	5.74
Left superior frontal gyrus	−24	42	−15	2	4.57
rTPJ > rSMG					
Right TPJ	51	−54	21	6125	39.54
Left TPJ	−48	−60	24	1048	15.59
Left middle temporal gyrus	−60	−3	−21	1252	15.32
Right superior frontal gyrus	21	36	48	5226	14.91
Left cerebellum	−30	−78	−33	525	9.37
Left cerebellum	−6	−54	−45	184	9.09
Right cerebellum	30	−78	−33	430	8.57
Left middle frontal gyrus	−33	54	−6	2	4.72
HC > ASD > rSMG > rTPJ[a]					
					N.S.
HC > ASD > rTPJ > rSMG[a]					
Left TPJ	−42	−66	21	311	3.61
Left middle frontal gyrus	−21	21	39	170	3.37
Left precuneus	−3	−63	27	611	3.29
Right superior frontal gyrus	6	66	0	236	3.15
Right TPJ	39	−60	27	147	3.08
Right middle frontal gyrus	27	33	45	86	3.01
ADOS social interaction regression in rTPJ network[a]					
Left middle temporal gyrus	−30	−66	21	211	4.31
Right posterior cingulate	18	−54	18	167	4.28

[a] Results are cluster-wise corrected at FWE < .05

The analyses of Study 2 of networks underlying overcoming emotional egocentricity during empathy and ToM, showed that rSMG and rTPJ display highly specific resting-state connectivity profiles, further supporting the view of a functional segregation of these two networks (Silani et al. 2013). The rSMG, relative to rTPJ, was significantly connected to bilateral AI and MCC, i.e., regions which have been consistently shown to play a crucial role in emotion processing such as during interoception and empathy (Lamm et al. 2011; Singer et al. 2004, 2009). The rTPJ, relative to the rSMG, was in contrast predominantly connected with PCC, precuneus, MPFC, and lTPJ, all regions commonly associated with cognitive processes such as attentional processing, default mode brain function, as well as ToM (Buckner et al. 2008; Carter and Huettel 2013; Frith and Frith 2006; Gusnard et al. 2001; Van Overwalle 2009). These differing resting-state profiles of rSMG and rTPJ are in accordance with previous parcellations of the TPJ (Bzdok et al. 2013; Mars et al. 2012).

More importantly, the direct comparison of these networks between the healthy control and ASD samples revealed that in line with the behavioral patterns observed in Study 1, ASD participants displayed abnormal resting-state connectivity in the ToM network with significantly decreased functional connectivity of the rTPJ to the MPFC, PCC and lTPJ, but no significant functional connectivity decrease in the rSMG network. Additionally, symptom severity was shown to correlate negatively with increasing rTPJ/PCC coupling, speaking to the importance of the ToM network abnormalities in contributing to autistic symptomatology. These findings are in accordance with influential “disconnection theories” of ASD, suggesting that a

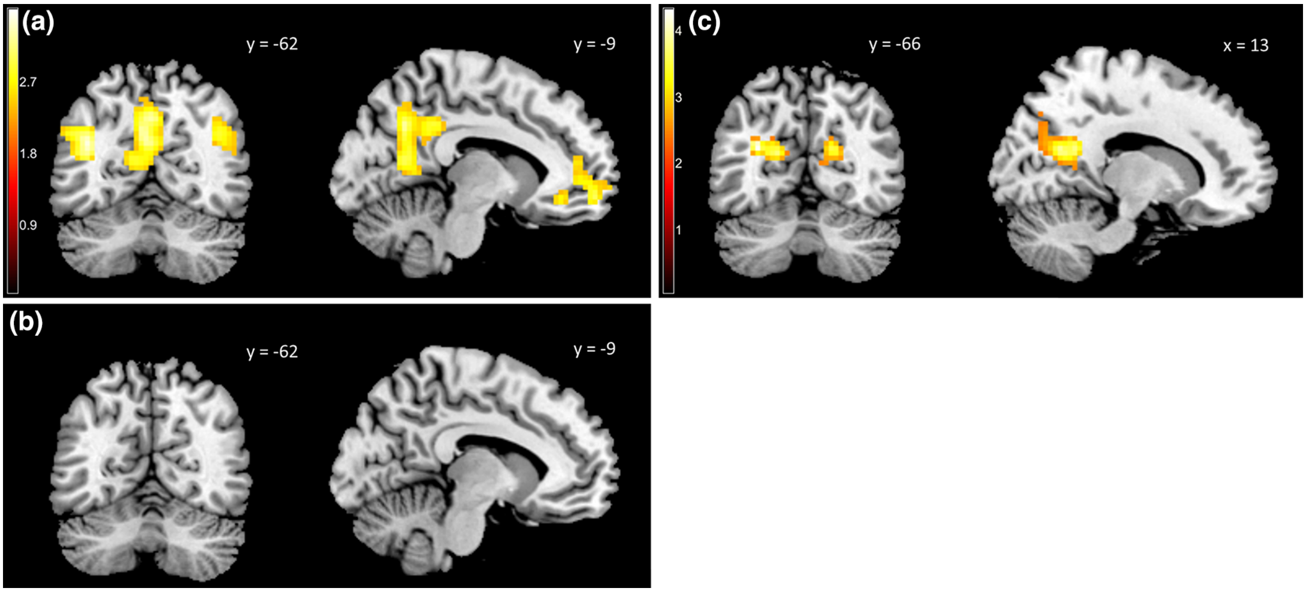

Fig. 4 **a** Display of significant group difference in resting-state connectivity in the rTPJ network. Findings revealed significant differences in rTPJ but not rSMG functional connectivity between the two groups, with individuals with ASD showing reduced functional connectivity from rTPJ to lTPJ, precuneus, PCC and MPFC (FWE < .05, cluster corrected). **b** No significant group difference in resting-state connectivity in the rSMG network (FWE < .05, cluster corrected). **c** Brain regions showing increased coupling during rest with rTPJ with decreasing symptom severity (ADOS social interaction) within the ASD sample (FWE < .05, cluster corrected). Stronger connectivity between regions of the PCC with rTPJ in individuals with ASD predicts smaller ADOS social interaction scores

disruption of a combination of frontotemporal, frontolimbic, frontoparietal and interhemispheric connections might be at the heart of the autistic condition (Belmonte et al. 2004; Courchesne and Pierce 2005; Geschwind and Levitt 2007; Just et al. 2007, 2012). One problem of the “disconnection theories” of ASD is the lack of specificity, being short of explanation to why some abilities in ASD are deficient, some remain spared and some even seem to be enhanced (Geschwind and Levitt 2007). Our results suggest that in the case of the temporo-parietal cortex in ASD, disrupted functional connectivity to other regions of the brain might be highly specific to TPJ but not the adjacent SMG, indicating that underconnectivity in ASD might just pertain to very specific brain networks. The finding of an intact rSMG but deficient rTPJ connectivity in ASD can also be seen as in accordance with findings suggesting that the ventral attention network of which rSMG represents a hub, is generally less impaired in ASD in contrast to the default mode network (Cherkassky et al. 2006; Kennedy and Courchesne 2008; von dem Hagen et al. 2013). In sum, these resting-state connectivity findings complement our behavioral findings of unaffected emotional self-other distinction during empathy in ASD but deficient ToM. They suggest that intact functioning of the rSMG network links with intact self-other distinction during empathy in ASD, while aberrant functioning of the rTPJ network possibly contributes to ToM deficits, which in turn add to the autistic symptomatology.

Taken together, this study provides novel evidence that self-other distinction deficits and resulting egocentricity in ASD are mainly present in the cognitive domain, not extending into the affective domain of empathy. The finding of intact emotional self-other distinction is in accordance with other findings showing partly intact empathic responding in ASD without comorbid alexithymia (Bird et al. 2010; Hadjikhani et al. 2014; Silani et al. 2008). Thus, importantly individuals with ASD exhibit even intact empathic judgments, when self and other are in different emotional states, which represents another spared socio-affective ability in ASD. Together this study and previous ones point to the need to closely reconsider the exact features of the social deficits portrayed in ASD and to strive for a more fine-grained characterization of this developmental disorder. Identifying areas of intact functioning in ASD could help to inform targeted-intervention programs and in the case of spared socio-affective abilities could play a major role as compensatory mechanisms in therapy.

This study also has some limitations. It would have been favorable to perform the resting-state connectivity analyses on the behaviorally tested sample. Unfortunately however, as mentioned in the method section, resting-state scans were not available for the behavioral sample. On the upside the use of a large independent sample for the resting-state connectivity analyses, diminishes concerns about possible power issues for detecting effects. In addition, while functional connectivity differences at rest as reported in this study, do not necessarily also reflect functional connectivity differences during tasks (Hasson et al. 2009; Mennes et al. 2013), there is strong evidence that task-related functional connectivity in the brain is primarily composed of intrinsic functional connectivity (Cole et al. 2014; Smith et al. 2009). Nevertheless, future studies should also test the ETOP with healthy controls and individuals with ASD directly in the scanner, to investigate whether the rSMG network is also intact in ASD during the task itself.

In conclusion, this study demonstrated that while individuals with ASD exhibited expected deficits in ToM, emotional egocentricity was comparable to that of healthy controls, suggesting intact self-other distinction during empathic judgments. Importantly, via brain analyses we were able to associate self-other distinction during empathy on the one hand and ToM on the other to clearly divergent resting-state connectivity profiles with two adjacent seed regions in the right temporo-parietal junction, the rTPJ and rSMG, thus replicating previous findings. Importantly, and in line with the behavioral results, only the connectivity from rTPJ and not rSMG was significantly reduced for ASD patients compared to controls and correlated with symptom severity. This suggests that unlike ToM and its associated underlying rTPJ network, self-other distinction during empathy and its underlying rSMG network remain spared in individuals with ASD. These findings provide further detail for a more fine-grained characterization of social deficits in ASD, providing evidence for spared social-affective functioning, but deficiencies in socio-cognitive functioning.

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References

Aichhorn, M., Perner, J., Kronbichler, M., Staffen, W., & Ladurner, G. (2006). Do visual perspective tasks need theory of mind? *Neuroimage, 30*(3), 1059–1068.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders, text revision* (4th ed.). Washington, DC: American Psychiatric Association.

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington, VA: American Psychiatric Association.

Asperger, H. (1944). Die "Autistischen Psychopathen" im Kindesalter. *European Archives of Psychiatry and Clinical Neuroscience, 117*(1), 76–136.

Baron-Cohen, S., Leslie, A. M., & Frith, U. (1985). Does the autistic child have a "theory of mind"? *Cognition, 21*(1), 37–46.

Baron-Cohen, S., Wheelwright, S., Hill, J., Raste, Y., & Plumb, I. (2001). The "Reading the Mind in the Eyes" test revised version: A study with normal adults, and adults with Asperger syndrome or high-functioning autism. *Journal of Child Psychology and Psychiatry, 42*(2), 241–251.

Bastiaansen, J., Thioux, M., & Keysers, C. (2009). Evidence for mirror systems in emotions. *Philosophical Transactions of the Royal Society B: Biological Sciences, 364*(1528), 2391–2404.

Begeer, S., Bernstein, D. M., van Wijhe, J., Scheeren, A. M., & Koot, H. M. (2012). A continuous false belief task reveals egocentric biases in children and adolescents with Autism Spectrum Disorders. *Autism, 16*(4), 357–366.

Belmonte, M. K., Allen, G., Beckel-Mitchener, A., Boulanger, L. M., Carper, R. A., & Webb, S. J. (2004). Autism and abnormal development of brain connectivity. *The Journal of Neuroscience, 24*(42), 9228–9231.

Birch, S. A., & Bloom, P. (2007). The curse of knowledge in reasoning about false beliefs. *Psychological Science, 18*(5), 382–386.

Bird, G., & Cook, R. (2013). Mixed emotions: The contribution of alexithymia to the emotional symptoms of autism. *Translational Psychiatry, 3*(7), e285.

Bird, G., Silani, G., Brindley, R., White, S., Frith, U., & Singer, T. (2010). Empathic brain responses in insula are modulated by levels of alexithymia but not autism. *Brain, 133*(5), 1515–1525.

Brass, M., Ruby, P., & Spengler, S. (2009). Inhibition of imitative behaviour and social cognition. *Philosophical Transactions of the Royal Society B: Biological Sciences, 364*(1528), 2359–2367.

Buckner, R. L., Andrews-Hanna, J. R., & Schacter, D. L. (2008). The brain's default network. *Annals of the New York Academy of Sciences, 1124*(1), 1–38.

Bzdok, D., Langner, R., Schilbach, L., Jakobs, O., Roski, C., Caspers, S., et al. (2013). Characterization of the temporo-parietal junction by combining data-driven parcellation, complementary connectivity analyses, and functional decoding. *Neuroimage, 81*, 381–392.

Carter, R. M., & Huettel, S. A. (2013). A nexus model of the temporal–parietal junction. *Trends in Cognitive Sciences, 17*(7), 328–336.

Castelli, F., Frith, C., Happé, F., & Frith, U. (2002). Autism, Asperger syndrome and brain mechanisms for the attribution of mental states to animated shapes. *Brain, 125*(8), 1839–1849.

Cherkassky, V. L., Kana, R. K., Keller, T. A., & Just, M. A. (2006). Functional connectivity in a baseline resting-state network in autism. *NeuroReport, 17*, 1687–1690.

Cole, M. W., Bassett, D. S., Power, J. D., Braver, T. S., & Petersen, S. E. (2014). Intrinsic and task-evoked network architectures of the human brain. *Neuron, 83*(1), 238–251.

Courchesne, E., & Pierce, K. (2005). Why the frontal cortex in autism might be talking only to itself: Local over-connectivity but long-distance disconnection. *Current Opinion in Neurobiology, 15*(2), 225–230.

Dapretto, M., Davies, M. S., Pfeifer, J. H., Scott, A. A., Sigman, M., Bookheimer, S. Y., & Iacoboni, M. (2005). Understanding emotions in others: Mirror neuron dysfunction in children with autism spectrum disorders. *Nature Neuroscience, 9*(1), 28–30.

David, N., Schultz, J., Milne, E., Schunke, O., Schöttle, D., Münchau, A., et al. (2014). Right temporoparietal gray matter predicts accuracy of social perception in the autism spectrum. *Journal of Autism and Developmental Disorders*, *44*(6), 1433–1446.

Decety, J., & Lamm, C. (2007). The role of the right temporoparietal junction in social interaction: How low-level computational

processes contribute to meta-cognition. *The Neuroscientist, 13*(6), 580–593.
Dziobek, I., Fleck, S., Kalbe, E., Rogers, K., Hassenstab, J., Brand, M., et al. (2006). Introducing MASC: A movie for the assessment of social cognition. *Journal of Autism and Developmental Disorders, 36*(5), 623–636.
Frith, C. D., & Frith, U. (2006). The neural basis of mentalizing. *Neuron, 50*(4), 531–534.
Frith, C. D., & Frith, U. (2012). Mechanisms of social cognition. *Annual Review of Psychology, 63*, 287–313.
Gallese, V. (2001). The' shared manifold' hypothesis: From mirror neurons to empathy. *Journal of Consciousness Studies, 8*(5–7), 33–50.
Gallese, V. (2007). Before and below 'theory of mind': Embodied simulation and the neural correlates of social cognition. *Philosophical Transactions of the Royal Society B: Biological Sciences, 362*(1480), 659–669.
Gallese, V., & Goldman, A. (1998). Mirror neurons and the simulation theory of mind-reading. *Trends in Cognitive Sciences, 2*(12), 493–501.
Geng, J. J., & Mangun, G. R. (2011). Right temporoparietal junction activation by a salient contextual cue facilitates target discrimination. *Neuroimage, 54*(1), 594–601.
Geschwind, D. H., & Levitt, P. (2007). Autism spectrum disorders: Developmental disconnection syndromes. *Current Opinion in Neurobiology, 17*(1), 103–111.
Gusnard, D. A., Akbudak, E., Shulman, G. L., & Raichle, M. E. (2001). Medial prefrontal cortex and self-referential mental activity: Relation to a default mode of brain function. *Proceedings of the National Academy of Sciences, 98*(7), 4259–4264.
Hadjikhani, N., Zürcher, N., Rogier, O., Hippolyte, L., Lemonnier, E., Ruest, T., et al. (2014). Emotional contagion for pain is intact in autism spectrum disorders. *Translational Psychiatry, 4*(1), e343.
Happé, F. G. (1994). An advanced test of theory of mind: Understanding of story characters' thoughts and feelings by able autistic, mentally handicapped, and normal children and adults. *Journal of Autism and Developmental Disorders, 24*(2), 129–154.
Hasson, U., Nusbaum, H. C., & Small, S. L. (2009). Task-dependent organization of brain regions active during rest. *Proceedings of the National Academy of Sciences, 106*(26), 10841–10846.
Horn, W. (1962). Leistungsprüfsystem, LPS: Handanweisung für die Durchführung, Auswertung und Interpretation.
Jones, A. P., Happé, F. G., Gilbert, F., Burnett, S., & Viding, E. (2010). Feeling, caring, knowing: Different types of empathy deficit in boys with psychopathic tendencies and autism spectrum disorder. *Journal of Child Psychology and Psychiatry, 51*(11), 1188–1197.
Just, M. A., Cherkassky, V. L., Keller, T. A., Kana, R. K., & Minshew, N. J. (2007). Functional and anatomical cortical underconnectivity in autism: Evidence from an FMRI study of an executive function task and corpus callosum morphometry. *Cerebral Cortex, 17*(4), 951–961.
Just, M. A., Keller, T. A., Malave, V. L., Kana, R. K., & Varma, S. (2012). Autism as a neural systems disorder: A theory of frontal–posterior underconnectivity. *Neuroscience and Biobehavioral Reviews, 36*(4), 1292–1313.
Kana, R. K., Libero, L. E., Hu, C. P., Deshpande, H. D., & Colburn, J. S. (2012). Functional brain networks and white matter underlying theory-of-mind in autism. *Social Cognitive and Affective Neuroscience, 9*(1), 98–105.
Kennedy, D. P., & Courchesne, E. (2008). The intrinsic functional organization of the brain is altered in autism. *Neuroimage, 39*(4), 1877–1885.
Klin, A. (2000). Attributing social meaning to ambiguous visual stimuli in higher-functioning autism and Asperger syndrome: The social attribution task. *Journal of Child Psychology and Psychiatry, 41*(7), 831–846.
Lamm, C., Decety, J., & Singer, T. (2011). Meta-analytic evidence for common and distinct neural networks associated with directly experienced pain and empathy for pain. *Neuroimage, 54*(3), 2492–2502.
Lehrl, S., Triebig, G., & Fischer, B. (1995). Multiple choice vocabulary test MWT as a valid and short test to estimate premorbid intelligence. *Acta Neurologica Scandinavica, 91*(5), 335–345.
Lockwood, P. L., Bird, G., Bridge, M., & Viding, E. (2013). Dissecting empathy: High levels of psychopathic and autistic traits are characterized by difficulties in different social information processing domains. *Frontiers in Human Neuroscience, 7*, 760.
Lombardo, M. V., & Baron-Cohen, S. (2010). Unraveling the paradox of the autistic self. *Wiley Interdisciplinary Reviews: Cognitive Science, 1*(3), 393–403.
Lombardo, M. V., & Baron-Cohen, S. (2011). The role of the self in mindblindness in autism. *Consciousness and Cognition, 20*(1), 130–140.
Lombardo, M. V., Chakrabarti, B., Bullmore, E. T., & Baron-Cohen, S. (2011). Specialization of right temporo-parietal junction for mentalizing and its relation to social impairments in autism. *Neuroimage, 56*(3), 1832–1838.
Lombardo, M. V., Chakrabarti, B., Bullmore, E. T., Sadek, S. A., Pasco, G., Wheelwright, S. J., et al. (2010). Atypical neural self-representation in autism. *Brain, 133*(2), 611–624.
Lord, C., Risi, S., Lambrecht, L., Cook, E. H., Leventhal, B. L., DiLavore, P. C., et al. (2000). The Autism Diagnostic Obersation Schedule-Generic: A standard measure of social and communication deficits associated with the spectrum of autism. *Journal of Autism and Developmental Disorders, 30*, 205–223.
Lord, C., Rutter, M., & Le Couteur, A. (1994). Autism Diagnostic Interview-Revisited: A revised version of a diagnostic interview for caregivers of individuals with possible pervasive developmental disorders. *Journal of Autism and Developmental Disorders, 24*(5), 659–685.
Mar, R. A. (2011). The neural bases of social cognition and story comprehension. *Annual Review of Psychology, 62*, 103–134.
Mars, R. B., Sallet, J., Schuffelgen, U., Jbabdi, S., Toni, I., & Rushworth, M. F. (2012). Connectivity-based subdivisions of the human right "temporoparietal junction area": Evidence for different areas participating in different cortical networks. *Cerebral Cortex, 22*(8), 1894–1903. doi:10.1093/cercor/bhr268.
Mennes, M., Kelly, C., Colcombe, S., Castellanos, F. X., & Milham, M. P. (2013). The extrinsic and intrinsic functional architectures of the human brain are not equivalent. *Cerebral Cortex, 23*(1), 223–229.
Menon, V., Ford, J. M., Lim, K. O., Glover, G. H., & Pfefferbaum, A. (1997). Combined event-related fMRI and EEG evidence for temporal–parietal cortex activation during target detection. *NeuroReport, 8*(14), 3029–3037.
Minio-Paluello, I., Baron-Cohen, S., Avenanti, A., Walsh, V., & Aglioti, S. M. (2009). Absence of embodied empathy during pain observation in Asperger syndrome. *Biological Psychiatry, 65*(1), 55–62.
Mitchell, J. P. (2009). Inferences about mental states. *Philosophical Transactions of the Royal Society B: Biological Sciences, 364*(1521), 1309–1316.
Montag, C., Dziobek, I., Richter, I. S., Neuhaus, K., Lehmann, A., Sylla, R., et al. (2011). Different aspects of theory of mind in paranoid schizophrenia: Evidence from a video-based assessment. *Psychiatry Research, 186*(2), 203–209.
Montag, C., Ehrlich, A., Neuhaus, K., Dziobek, I., Heekeren, H. R., Heinz, A., & Gallinat, J. (2010). Theory of mind impairments in

euthymic bipolar patients. *Journal of Affective Disorders, 123*(1), 264–269.
Mueller, S., Keeser, D., Samson, A. C., Kirsch, V., Blautzik, J., Grothe, M., et al. (2013). Convergent findings of altered functional and structural brain connectivity in individuals with high functioning autism: A multimodal MRI study. *PLoS One, 8*(6), e67329.
Nickerson, R. S. (2001). The projective way of knowing: A useful heuristic that sometimes misleads. *Current Directions in Psychological Science, 10*(5), 168–172.
Pitskel, N. B., Bolling, D. Z., Hudac, C. M., Lantz, S. D., Minshew, N. J., Vander Wyk, B. C., & Pelphrey, K. A. (2011). Brain mechanisms for processing direct and averted gaze in individuals with autism. *Journal of Autism and Developmental Disorders, 41*(12), 1686–1693.
Power, J. D., Barnes, K. A., Snyder, A. Z., Schlaggar, B. L., & Petersen, S. E. (2012). Spurious but systematic correlations in functional connectivity MRI networks arise from subject motion. *Neuroimage, 59*(3), 2142–2154.
Premack, D., & Woodruff, G. (1978). Does the chimpanzee have a theory of mind? *Behavioral and Brain Sciences, 1*(04), 515–526.
Pronin, E. (2008). How we see ourselves and how we see others. *Science, 320*(5880), 1177–1180.
Ramsey, R., Hansen, P., Apperly, I., & Samson, D. (2013). Seeing it my way or your way: Frontoparietal brain areas sustain viewpoint-independent perspective selection processes. *Journal of Cognitive Neuroscience, 25*(5), 670–684.
Ritter, K., Dziobek, I., Preißler, S., Rüter, A., Vater, A., Fydrich, T., et al. (2011). Lack of empathy in patients with narcissistic personality disorder. *Psychiatry Research, 187*(1), 241–247.
Rogers, K., Dziobek, I., Hassenstab, J., Wolf, O. T., & Convit, A. (2007). Who cares? Revisiting empathy in Asperger syndrome. *Journal of Autism and Developmental Disorders, 37*(4), 709–715.
Royzman, E. B., Cassidy, K. W., & Baron, J. (2003). " I know, you know": Epistemic egocentrism in children and adults. *Review of General Psychology, 7*(1), 38–65.
Santiesteban, I., Banissy, M. J., Catmur, C., & Bird, G. (2012a). Enhancing social ability by stimulating right temporoparietal junction. *Current Biology, 22*(23), 2274–2277.
Santiesteban, I., White, S., Cook, J., Gilbert, S. J., Heyes, C., & Bird, G. (2012b). Training social cognition: From imitation to theory of mind. *Cognition, 122*(2), 228–235.
Satterthwaite, T. D., Elliott, M. A., Gerraty, R. T., Ruparel, K., Loughead, J., Calkins, M. E., et al. (2013). An improved framework for confound regression and filtering for control of motion artifact in the preprocessing of resting-state functional connectivity data. *Neuroimage, 64*, 240–256.
Senju, A., Southgate, V., Miura, Y., Matsui, T., Hasegawa, T., Tojo, Y., et al. (2010). Absence of spontaneous action anticipation by false belief attribution in children with autism spectrum disorder. *Development and Psychopathology, 22*(02), 353–360.
Senju, A., Southgate, V., White, S., & Frith, U. (2009). Mindblind eyes: An absence of spontaneous theory of mind in Asperger syndrome. *Science, 325*(5942), 883–885.
Silani, G., Bird, G., Brindley, R., Singer, T., Frith, C., & Frith, U. (2008). Levels of emotional awareness and autism: An fMRI study. *Social Neuroscience, 3*(2), 97–112.
Silani, G., Lamm, C., Ruff, C. C., & Singer, T. (2013). Right supramarginal gyrus is crucial to overcome emotional egocentricity bias in social judgments. *The Journal of Neuroscience, 33*(39), 15466–15476.
Singer, T. (2012). The past, present and future of social neuroscience: A European perspective. *Neuroimage, 61*(2), 437–449.
Singer, T., Critchley, H. D., & Preuschoff, K. (2009). A common role of insula in feelings, empathy and uncertainty. *Trends in Cognitive Sciences, 13*(8), 334–340.
Singer, T., & Lamm, C. (2009). The social neuroscience of empathy. *Annals of the New York Academy of Sciences, 1156*(1), 81–96.
Singer, T., Seymour, B., O'Doherty, J., Kaube, H., Dolan, R. J., & Frith, C. D. (2004). Empathy for pain involves the affective but not sensory components of pain. *Science, 303*(5661), 1157–1162.
Smith, S. M., Fox, P. T., Miller, K. L., Glahn, D. C., Fox, P. M., Mackay, C. E., et al. (2009). Correspondence of the brain's functional architecture during activation and rest. *Proceedings of the National Academy of Sciences, 106*(31), 13040–13045.
Sommer, M., Döhnel, K., Sodian, B., Meinhardt, J., Thoermer, C., & Hajak, G. (2007). Neural correlates of true and false belief reasoning. *Neuroimage, 35*(3), 1378–1384.
Song, X. W., Dong, Z. Y., Long, X. Y., Li, S. F., Zuo, X. N., Zhu, C. Z., et al. (2011). REST: A toolkit for resting-state functional magnetic resonance imaging data processing. *PLoS One*. doi:10.1371/journal.pone.0025031.
Spengler, S., von Cramon, D. Y., & Brass, M. (2009). Control of shared representations relies on key processes involved in mental state attribution. *Human Brain Mapping, 30*(11), 3704–3718.
Spengler, S., von Cramon, D. Y., & Brass, M. (2010). Resisting motor mimicry: Control of imitation involves processes central to social cognition in patients with frontal and temporo-parietal lesions. *Social Neuroscience, 5*(4), 401–416.
Steinbeis, N., Bernhardt, B. C., & Singer, T. (2014). Age-related differences in function and structure of rSMG and reduced functional connectivity with DLPFC explains heightened emotional egocentricity bias in childhood. *Social Cognitive and Affective Neuroscience*, *10*(2), 302–310.
Stoeckel, C., Gough, P. M., Watkins, K. E., & Devlin, J. T. (2009). Supramarginal gyrus involvement in visual word recognition. *Cortex, 45*(9), 1091–1096.
Taylor, G. J., Ryan, D., & Bagby, R. M. (1985). Toward the development of a new self-report alexithymia scale. *Psychotherapy and Psychosomatics, 44*(4), 191–199.
Van Boven, L., & Loewenstein, G. (2003). Social projection of transient drive states. *Personality and Social Psychology Bulletin, 29*(9), 1159–1168.
Van Overwalle, F. (2009). Social cognition and the brain: A meta-analysis. *Human Brain Mapping, 30*(3), 829–858.
von dem Hagen, E. A., Stoyanova, R. S., Baron-Cohen, S., & Calder, A. J. (2013). Reduced functional connectivity within and between 'social'resting-state networks in autism spectrum conditions. *Social Cognitive and Affective Neuroscience, 8*(6), 694–701.
Washington, S. D., Gordon, E. M., Brar, J., Warburton, S., Sawyer, A. T., Wolfe, A., et al. (2013). Dysmaturation of the default mode network in autism. *Human Brain Mapping, 35*(4), 1284–1296.