Developmental Cognitive Neuroscience 34 (2018) 139–147

Contents lists available at ScienceDirect

Developmental Cognitive Neuroscience

journal homepage: www.elsevier.com/locate/dcn

Do you know what I'm thinking? Temporal and spatial brain activity during a theory-of-mind task in children with autism

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ARTICLE INFO

Keywords:
Autism
Children
False belief
Magnetoencephalography
Theory of mind

ABSTRACT

The social impairments observed in children with autism spectrum disorder are thought to arise in part from deficits in theory of mind, the ability to understand other people's thoughts and feelings. To determine the temporal-spatial dynamics of brain activity underlying these atypical theory-of-mind processes, we used magnetoencephalography to characterize the sequence of functional brain patterns (i.e. when and where) related to theory-of-mind reasoning in 19 high-functioning children with autism compared to 22 age- and sex-matched typically-developing children aged 8–12 during a false-belief (theory-of-mind) task. While task performance did not differ between the two groups, children with autism showed reduced activation in the left temporoparietal junction between 300–375 and 425–500 ms, as well as increased activation in the right inferior frontal gyrus from 325 to 375 ms compared to controls. The overlap in decreased temporoparietal junction activity and increased right inferior frontal gyrus activation from 325 to 375 ms suggests that in children with autism, the right inferior frontal gyrus may compensate for deficits in the temporoparietal junction, a neural theory-of-mind network hub. As the right inferior frontal gyrus is involved in inhibitory control, this finding suggests that children with autism rely on executive functions to bolster their false-belief understanding.

1. Introduction

Theory of mind (ToM) is typically defined as the ability to attribute mental states to others, or the understanding that others may have thoughts, feelings, and perspectives independent from our own (Premack and Woodruff, 1978). ToM is frequently impaired in individuals with autism spectrum disorder (ASD), which is thought to contribute to their social cognitive deficits (Baron-Cohen et al., 1985; Perner et al., 1989). Since ToM requires the orchestration of numerous brain regions to rapidly detect and integrate complex social cues, identifying both the timing and location of brain areas that are involved in this intricate process will allow the determination of when and where differences in brain activity occur in ASD.

Functional MRI studies have demonstrated a network of brain regions involved in ToM reasoning in typically-developing (TD) individuals, including the precuneus, the middle temporal gyrus (MTG), the temporoparietal junction (TPJ), and the medial prefrontal cortex (mPFC) (Carrington and Bailey, 2009; Van Overwalle and Baetens, 2009; van Veluw and Chance, 2014). Both children and adults with ASD have demonstrated atypical activity in many of these areas, although there is mixed evidence as to the nature of these differences. Some studies have shown that those with ASD exhibit increased activity in certain brain areas of the ToM network (Kim et al., 2016; White et al., 2014), while others have found decreased activity in these same regions (Kana et al., 2015; Murdaugh et al., 2014; O'Nions et al., 2014). Individuals with ASD also have been found to recruit additional brain

Abbreviations: ADOS, Autism Diagnostic Observation Schedule; AG, angular gyrus; ASD, autism spectrum disorder; EEG, electroencephalography; FB, false belief; IFG, inferior frontal gyrus; ITG, inferior temporal gyrus; MEG, magnetoencephalography; MFG, middle frontal gyrus; MOG, middle occipital gyrus; mPFC, medial prefrontal cortex; MTG, middle temporal gyrus; NEPSY-II, Developmental Neuropsychological Assessment, Second Edition; ROI, region of interest; RT, response time; TB, true belief; TD, typically developing; ToM, theory of mind; TPJ, temporoparietal junction; WASI, Wechsler Abbreviated Scale of Intelligence; WMTB-C, Working Memory Test Battery for Children

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https://doi.org/10.1016/j.dcn.2018.08.001
Received 8 June 2017; Received in revised form 15 August 2018; Accepted 30 August 2018
Available online 29 September 2018

areas during ToM reasoning, such as the bilateral inferior frontal gyri (IFG), which may act as compensatory mechanisms in those who are able to perform similarly to their TD peers on behavioural tests of ToM (Colich et al., 2012; Libero et al., 2014).

To date, the temporal dynamics of ToM-related brain activity remain virtually unexplored in the ASD population. Although knowledge of how the neural mechanisms underlying ToM differ in children with and without ASD would greatly benefit our understanding of the atypical development of social cognition in ASD, current research has focused primarily on TD individuals. Using electroencephalography (EEG) and its more spatially precise counterpart, magnetoencephalography (MEG; Hämäläinen et al., 1993), researchers have found that during ToM reasoning, TD children and adults activate the precuneus between 275–325 ms post-stimulus onset (Mossad et al., 2016), the TPJ as early as 150–225 ms and at 400–450 ms (McCleery et al., 2011; Mossad et al., 2016; Vistoli et al., 2011), and the mPFC from 350 to 450 ms to as late as ~800 ms (Liu et al., 2009; Pylkkänen and McElree, 2007; Sabbagh and Taylor, 2000). One MEG study by Hasegawa et al. (2013) investigated ToM in control adults and related their findings to the ASD population by correlating brain activity with Autism Spectrum Quotient (AQ) scores, which is used to screen individuals for ASD traits (Baron-Cohen et al., 2001; Woodbury-Smith et al., 2005). They found greater activity in an area adjacent to the TPJ between 150–250 ms, which positively correlated with AQ scores. However, these results cannot be generalized to the ASD population, as individuals with ASD undergo atypical neural development (Lange et al., 2015).

The present study is the first to explore, at the behavioural and neurophysiological level, the complex temporal-spatial neural underpinnings of ToM in children with ASD. We used MEG, which has excellent temporal and spatial resolution (Hari et al., 2010), to record brain activity during a task that has been shown to elicit activity in ToM-related brain regions in adults (Mossad et al., 2016). The task assessed understanding of false belief (FB), which refers to the idea that another person could believe something that is untrue or that differs from one's own beliefs (Baron-Cohen et al., 1985). We used FB as a measure of ToM, as recognizing FB is thought to be a common marker of attaining ToM competence (Wellman et al., 2001), and studies have shown that children with ASD consistently fail tests of FB compared to age-matched TD peers (Baron-Cohen et al., 1985; Begeer et al., 2012; Peterson, 2005).

More specifically, we examined whether brain activity differed between situations involving FB and those involving true belief (TB), in which a person's belief is in line with reality. While both FB and TB engage ToM processes, we chose to compare FB to TB based on the ToM model proposed by Dennis et al. (2013), which demonstrated that TB conditions serve as an appropriate control for FB tasks, as TB involves many of the same cognitive components, but does not have the added requirement of distinguishing between one's own thoughts and another's – a crucial feature that defines FB and ToM. Hence, our comparison allows for a more precise measurement of FB processes, as functions not specific to FB reasoning (i.e., present in both FB and TB scenarios), such as processing one's own viewpoint, are cancelled out. Several studies have found significant neural differences when contrasting FB with TB (Bardi et al., 2017; Hyde et al., 2015; Kovács et al., 2014; Mossad et al., 2016; Sommer et al., 2007), while others have found comparable activation of the TPJ in FB and TB conditions (Döhnel et al., 2012; Schneider et al., 2014). Although this discrepancy questions the contrast of FB to TB, it is suggested that these inconsistencies may be due to differences in task design and demands (Saxe, 2009), and given the similarity of our task to previous work demonstrating differential TPJ activity in FB versus TB tasks (Sommer et al., 2007), we predict that our analyses will reveal changes in TPJ activity for FB compared to TB.

We hypothesized that children with ASD would show reduced activation in a ToM network of brain areas, namely the precuneus, TPJ, MTG, and mPFC, and that they would draw on additional brain regions to compensate. We predicted, based on previous EEG and MEG studies, that these regions of the ToM network would be recruited between 300–400 ms in TD children, and children with ASD would show delayed activation of these regions.

2. Materials and methods

2.1. Participants

We tested 22 typically-developing (TD) children (19 males, mean age = 10.34 ± 1.32 years) and 19 children with autism spectrum disorder (ASD) (16 males, mean age = 10.52 ± 1.45 years) between ages 8 and 12. (Initially, 44 TD children and 34 children with ASD were recruited, but 11 TD and 15 ASD participants were excluded from analyses due to artefacts in the MEG, excessive movement, or poor (≤ 55%) task performance. An additional 11 TD participants were excluded after matching the groups on age and sex.) The age range of 8–12 years was chosen based on previous related neuroimaging work using similar age ranges (e.g., Gweon et al., 2012; Kobayashi et al., 2007; Saxe et al., 2009), and after piloting the task in a small cohort of children to determine the ages at which children (under 13) would be capable of meeting the study demands. Inclusion criteria were full scale (two-subtest) IQ > 70 and no history of psychological, neurological, or developmental disorders (except in the ASD group). Participants with ASD all had a primary diagnosis of ASD from a clinical expert, confirmed by the Autism Diagnostic Observation Schedule (ADOS-G or ADOS-2; Lord et al., 2000, 2012). The two groups did not differ in age ($t(39) = 0.42$, $p = 0.67$) or sex ($X^2(1) = 5.29 \times 10^{-31}$, $p = 1$). The mean ADOS calibrated severity score in the ASD group was 6.79 ± 2.04. Informed assent was given by all children, and informed consent was obtained from their parents. Testing took place at the Hospital for Sick Children in Toronto, Canada. This study was approved by the institutional Research Ethics Board.

2.2. Experimental design

2.2.1. Neurocognitive assessments

We administered psychological tests to assess a variety of cognitive skills. The Vocabulary and Matrix Reasoning subtests of the Wechsler Abbreviated Scale of Intelligence (WASI; Wechsler, 1999) were used to estimate IQ. The Forward and Backward Digit Recall subtests of the Working Memory Test Battery for Children (WMTB-C; Pickering and Gathercole, 2001) measured working memory capacity. The Inhibition and Theory of Mind subtests of the Developmental Neuropsychological Assessment, Second Edition (NEPSY-II; Korkman et al., 2007) evaluated their respective titular domains.

2.2.2. False-belief MEG task

Children completed a pictorial false-belief task adapted from Dennis et al. (2012) for MEG and used recently by Mossad et al. (2016) to investigate ToM in adults (Fig. 1A). In each trial, children saw two consecutive images concerning two characters, Jack and Jill. Jill first sees Jack holding a ball over one of two hats, then Jack either drops the ball into the hat or *switches* his decision and drops it into the other hat, and Jill either *witnesses* the ball's placement or does not. Children were told explicitly that Jack wanted to put the ball into the hat that he held it over in the first picture, and that he changes his mind when he drops the ball into the other hat. The second picture therefore revealed one of four outcomes, three of which contained true beliefs (TB; i.e. Jill's belief about the ball's location is correct), while the remaining one involved a false belief (FB; i.e. Jill's belief is incorrect because Jack moves the ball without Jill's knowledge; see Fig. 1). Upon seeing the second picture, children indicated, using a button box, in which hat Jill thinks the ball is. They received feedback in the form of a green checkmark for a correct answer or a red cross for an incorrect answer.

Stimuli were presented using *Presentation 18.1* (Neurobehavioral Systems Inc., https://www.neurobs.com/presentation) and back-projected onto a screen at a viewing distance of 80 cm. Each trial began

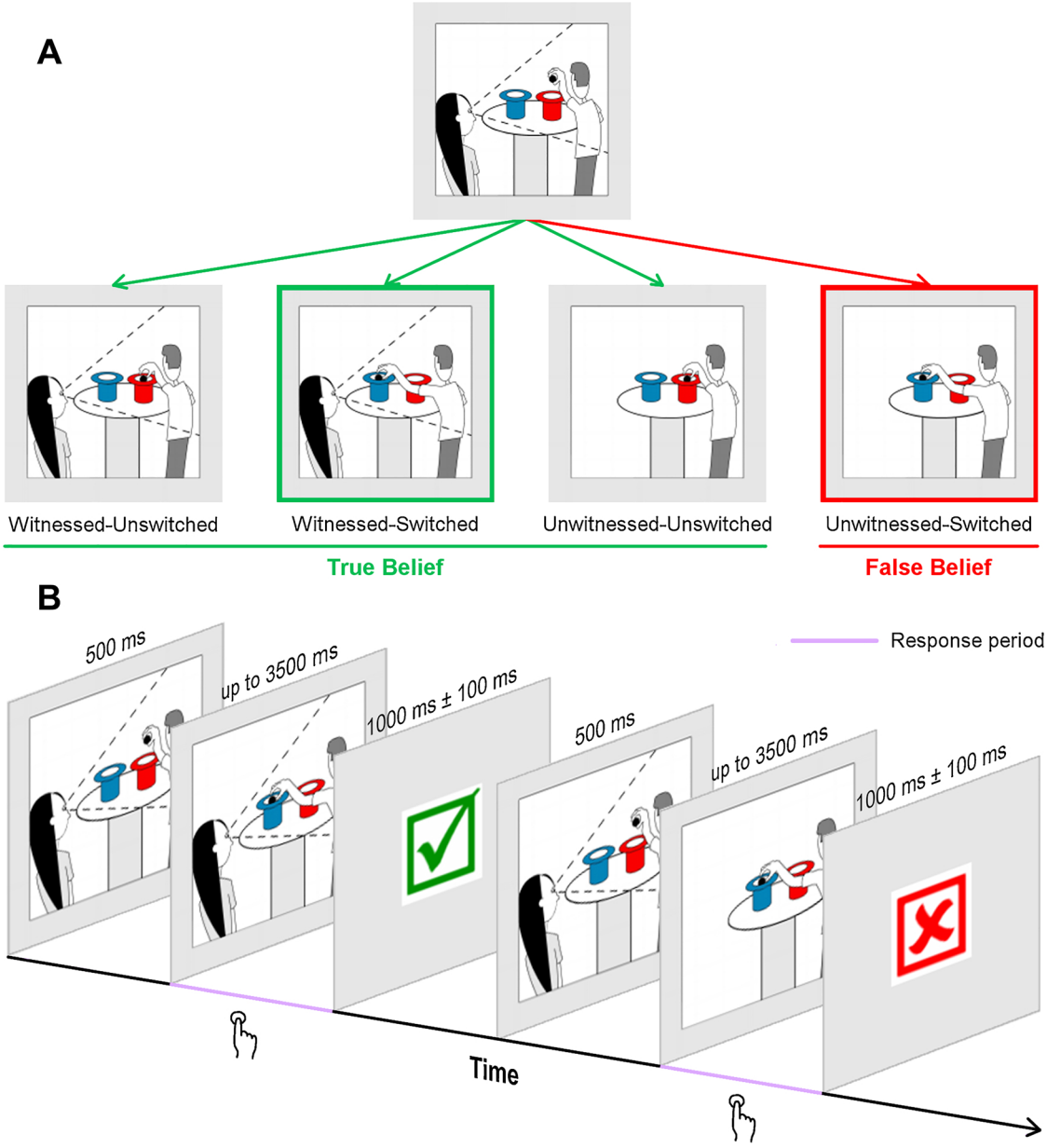

Fig. 1. Stimuli for the false-belief MEG task, adapted from Dennis et al. (2012). (A) An example of the four trial types. The first picture of the trial (top) showed Jill seeing Jack hold a ball over one of two hats. The second picture (bottom row) presented one of four scenarios: (1) Witnessed-Unswitched, where Jill watches (Witnessed) as Jack puts the ball in the same hat he was holding it over in the first picture (Unswitched); (2) Witnessed-Switched, where Jill watches (Witnessed) as Jack changes his mind and puts the ball in the other hat (Switched); (3) Unwitnessed-Unswitched, where Jill goes away (Unwitnessed), and then Jack puts the ball in the same hat (Unswitched); and (4) Unwitnessed-Switched, where Jill goes away (Unwitnessed), and then Jack puts the ball in the other hat (Switched). The first three conditions contain true beliefs, as Jill knows the correct location of the ball, whereas the fourth – Unwitnessed-Switched – involves a false belief, as Jack switches the ball without Jill's knowledge. Analyses focused on the Witnessed-Switched and Unwitnessed-Switched conditions, outlined in green and red, respectively. (B) The timing of the stimuli in the task. The first picture of each trial appeared for 500 ms, followed by the second picture, which was shown for 3500 ms or until the child responded, whichever came first. Feedback then appeared for 1000 ± 100 ms, and the next trial began immediately after. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article).

with the presentation of the first image for 500 ms, then the second picture for a maximum of 3500 ms, or until the child responded, followed by feedback, which was displayed for 1000 ms with a jitter of ± 100 ms (Fig. 1B).

All children completed a practice session outside the MEG. They then performed the task in the MEG scanner, which involved 100 FB (Unwitnessed-Switched) and 200 TB (100 Witnessed-Switched, 50 Unwitnessed-Unswitched, and 50 Witnessed-Unswitched) trials, presented pseudo-randomly. Therefore, children saw Jill in the second image 50% of the time, and the ball being switched 66% of the time. The task ended when either the child correctly completed 300 trials, or after 15 min had passed.

2.2.3. MEG data acquisition

A CTF MEG system (151 axial gradiometers; Coquitlam, British Columbia, Canada) within a magnetically shielded room was used to acquire the MEG data. Fiducial coils were placed on the nasion and the left and right pre-auricular points to track head position. Children lay supine with their head in the MEG dewar during the task. Data were sampled at 600 Hz with continuous head localization. To optimize the signal-to-noise ratio, a third-order spatial gradient was used, and an anti-aliasing low-pass filter of 150 Hz was applied to the signal.

2.2.4. MRI data acquisition

A 3.0T MRI scanner (MAGNETOM Tim Trio, Siemens AG, Erlangen, Germany) with a 12-channel head coil was used to acquire anatomical MRIs for accurate localization of sources of MEG activity. Radio-opaque markers were placed at the same sites as the fiducial coils used in the MEG to ensure accurate MEG-MRI co-registration. T1-weighted MRI scans were acquired sagittally using the 3D SAG MPRAGE sequence (GRAPPA = 2, TR/TE/FA = 2300 ms/2.96 ms/9°, FOV = 192 × 240 × 256 mm, voxel size = 1.0 mm isotropic).

2.3. Statistical analysis

2.3.1. Behavioural data

Scores on the neurocognitive assessments (WASI, WMTB-C, and NEPSY-II) and the participants' mean response times (RTs) and accuracy on the task were analyzed using R 3.2.3 (R Core Team, https://www.r-project.org/). T-tests were used to compare the two groups' scores on each of the cognitive assessments. For the WMTB-C, composite scores were created from the sum of the scaled scores of the Forward and Backward Digit Recall tests. The NEPSY-II Inhibition vs. Naming scaled scores were taken as a measure of inhibition, controlled for participants' ability to name objects. Total raw scores on the NEPSY-II ToM subtest were compared, as the NEPSY-II does not provide scaled scores for the ToM subtest for children aged 8–12 years.

For our experimental FB task, linear mixed-effects models were fit to determine differences in accuracy and mean RT, with group (TD or ASD), switching (Switched or Unswitched), and witnessing (Witnessed or Unwitnessed) as predictors, and IQ and age as covariates. Main effects of group, switching, witnessing, IQ, and age were included in the analysis, as well as the interactions between group, switching, and witnessing, and group and IQ. To account for the repeated measurement of each participant over the different conditions, participants were included as random effects.

Post-hoc analyses, as well as our neuroimaging analyses, focused on differences between the Unwitnessed-Switched and Witnessed-Switched conditions. Therefore, although our task paradigm contains three different TB scenarios, only the Witnessed-Switched condition was analyzed in comparison to the FB (Unwitnessed-Switched) condition. Thus, the Witnessed-Switched condition will hereafter be referred to as the TB condition, and the Unwitnessed-Switched condition as the FB condition. Significant behavioural results are reported at $p < 0.05$.

2.3.2. MEG data

MEG data were analyzed with SPM12 (FIL Methods Group, www.fil.ion.ucl.ac.uk/spm/) in MATLAB 2014b (The MathWorks, www.mathworks.com/products/matlab/). Signals were filtered offline between 1–50 Hz with a fifth-order Butterworth bandpass filter. Trials were epoched from -200 to 600 ms relative to the presentation of the second stimulus and baseline corrected. Head motion artefacts were controlled by discarding trials in which the child moved 5 mm within a trial or 10 mm between trials. Independent component analysis, as implemented by FieldTrip (Oostenveld et al., 2011), was used to detect and remove heartbeat and eyeblink artefacts in the data. Remaining artefacts were excluded by rejecting trials in which the signal exceeded 2500 fT at any of the MEG sensors, and by discarding sensors in which more than 20% of trials surpassed this threshold. Data were then averaged across only the correct trials for each condition and participant.

Co-registration of MEG data and corresponding anatomical MRIs was performed for each child using the fiducial points. The forward model was calculated based on the single-shell model for computing the lead field matrix (Nolte, 2003), and the inverse model was generated using the minimum norm estimation method in SPM (Litvak et al., 2011). Results from the inversion were averaged over a 50 ms sliding time window, with an overlap of 25 ms, between 100–500 ms post-stimulus, leading to 15 time windows (e.g. 100–150 ms, 125–175 ms, etc.) for each participant and condition, which were then exported as 3D NIFTI images in MNI space. These images were spatially smoothed by a 12 mm full-width, half-maximum Gaussian kernel before being input into a 2 × 2 × 2 (group*switching*witnessing) factorial ANCOVA to model the effects of group and each condition on brain activity, with age and the interaction between group and IQ as nuisance covariates.

Planned comparison t-tests were performed to evaluate specific within- and between-group effects of interest, namely (i) brain regions that were activated more strongly in the FB than the TB condition within each group (TD, FB > TB and ASD, FB > TB); and (ii) brain regions that were differentially active in the ASD compared to the TD group (ASD < TD, FB > TB and ASD > TD, FB > TB), which were obtained by examining the interaction between the group, switching, and witnessing factors. To control for task effects unrelated to the FB > TB contrast, such as different visual or attentional demands between the two conditions, a mask was computed for each time window by contrasting the remaining two TB conditions, Unwitnessed-Unswitched > Witnessed-Unswitched. Any significant voxels ($p < 0.05$, uncorrected) in the masks were removed from our main FB > TB contrast. This comparison served as an ideal control, as the two conditions are visually and conceptually similar to those in the FB > TB contrast. For instance, in both the FB and the Unwitnessed-Unswitched conditions, Jill is not present in the second frame, so her initial belief of the ball's location must be inferred, and in both the TB and Witnessed-Unswitched conditions, Jill is present and sees where the ball is placed. Therefore, use of these masks mitigated any effects due to differences between the stimuli in the FB and TB conditions.

2.3.2.1. Region of interest (ROI) analyses. We selected regions of interest (ROIs) from a meta-analysis by Schurz et al. (2013) of 25 functional neuroimaging studies of false belief. They found six areas commonly active for these tasks: the right precuneus [6 -59 35], the left and right TPJ (LTPJ: [-57 -65 27]; RTPJ: [62 -45 21]), the left mPFC [-5 60 21], and two regions of the right superior temporal gyrus ([51 -9 -9] and [46 11–24]). For our analyses, we focused only on the precuneus, bilateral TPJ, and mPFC, as our *a priori* hypotheses addressed solely these four areas.

ROIs were defined as 10-mm spheres centred on each of these peaks. Significant activations in these ROIs are reported at $p_{svc} < 0.05$ corrected for multiple comparisons over the ROI volume using small volume Gaussian random field correction (Worsley et al., 1996).

2.3.2.2. Whole-brain analyses. We also performed an exploratory whole-brain, voxel-wise analysis to examine whether additional brain areas were activated during our task. Significant peaks are reported at an uncorrected $p < 0.001$ or at $p_{corr} < 0.05$ corrected for multiple comparisons using Gaussian random field theory (Worsley et al., 1996), although relevant areas that appeared at an exploratory threshold of $p < 0.005$ are also reported to broaden our main findings.

3. Results

3.1. Neurocognitive measures

Two-sample, two-tailed t-tests revealed that the TD group had an overall higher IQ (mean = 119.55 ± 9.49; $t(39) = 2.96$, $p = 0.005$) than the ASD group (mean = 109.58 ± 12.05), although both groups had IQs in the normal range. The two groups also differed on the Theory of Mind subtest of the NEPSY-II ($t(37) = 2.94$, $p = 0.006$), with TD children scoring higher (mean = 25.27 ± 1.80) than children with ASD (mean = 22.82 ± 3.34). There were no significant differences between the TD and ASD groups on the WMTB-C, nor on the Inhibition subtest of the NEPSY-II (all $ps > 0.05$; see Table 1).

Table 1
Mean scores and standard deviations on neurocognitive assessments.

	TD Score (N)	ASD Score (N)	*t*	*p*	
WASI	119.55 ± 9.49 (22)	109.58 ± 12.05 (19)	2.96	0.005	*
WMTB-C	216.64 ± 32.71 (22)	204.00 ± 37.01 (19)	1.16	0.25	
NEPSY-II Inhibition	10.95 ± 3.05 (22)	9.82 ± 3.64 (17)	1.06	0.30	
NEPSY-II Theory of Mind	25.27 ± 1.80 (22)	22.82 ± 3.34 (17)	2.94	0.006	*

Scores are given as mean ± SD. Abbreviations: NEPSY-II = Developmental Neuropsychological Assessment, Second Edition; WASI = Wechsler Abbreviated Scale of Intelligence; WMTB-C = Working Memory Test Battery for Children.
* Significant difference between groups ($p < 0.05$).

3.2. Task performance

Linear mixed-effects models showed that accuracy and mean RT differed for switching (accuracy: $F(1,117) = 100.30$, $p < 0.0001$; RT: $F(1,117) = 113.81$, $p < 0.0001$) and witnessing trials (accuracy: $F(1,117) = 83.00$, $p < 0.0001$; RT: $F(1,117) = 145.88$, $p < 0.0001$). There was also an interaction between switching and witnessing for accuracy ($F(1,117) = 17.35$, $p = 0.0001$), but not mean RT ($F(1,117) = 1.15$, $p = 0.29$). A post-hoc *t*-test of our conditions of interest revealed that all children were less accurate on the FB trials (FB mean = 79.71 ± 10.93% correct; TB mean = 92.46 ± 5.85% correct; $t(40) = 8.28$, $p = 1.68 \times 10^{-10}$; Fig. 2).

There was no main effect of group, nor any significant interaction between group and the other variables for either accuracy or mean RT. Including IQ and its interaction with group as covariates in the model did not account for any differences in accuracy or mean RT (all $ps > 0.05$; see Table A.1). However, age did covary with mean RT ($F(1,36) = 19.63$, $p = 0.001$). A Pearson's correlation revealed that age was inversely related to mean RT ($r(80) = -0.52$, $p = 6.26 \times 10^{-7}$), such that older children responded faster.

3.3. Neuroimaging

3.3.1. ROI analyses

TD children activated the precuneus between 350–400 ms, as well as the LTPJ from 300 to 500 ms. Activity in the LTPJ was reduced in the ASD group between 300–375 ms and 425–475 ms (Fig. 3A). However, neither group showed significant activation in the RTPJ or the mPFC to the FB versus TB condition (Table 2).

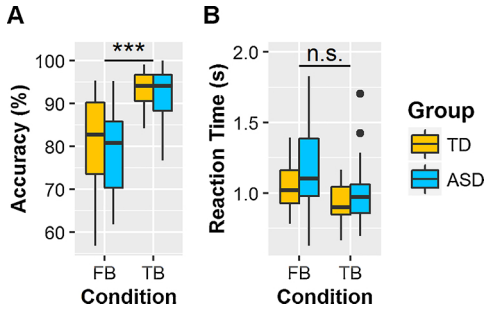

Fig. 2. Accuracy (A) and mean response times (B) for the false-belief MEG task. The two groups did not differ in terms of accuracy on the task, but post-hoc t-tests revealed that all participants performed significantly more poorly ($p < 0.05$) on the FB compared to TB trials. Although this relationship appears to similarly exist for the mean response times, it was not explored, as the interaction between the two factors in our task (switching and witnessing) was not significant ($p > 0.05$).
*** Significant difference between conditions ($p < 0.001$).

3.3.2. Whole-brain analyses

Our exploratory whole-brain analyses showed that the TD group activated the precuneus more for FB than TB from 325 to 425 ms, and the LTPJ, which encompassed the left angular gyrus, middle temporal gyrus, and middle occipital gyrus, from 325 to 475 ms, similar to our ROI analyses. Additionally, TD children activated the right middle frontal gyrus (RMFG) from 275 to 325 ms and 450 to 500 ms, the right middle occipital gyrus (RMOG) from 375 to 450 ms, and the right inferior temporal gyrus (RITG) from 375 to 475 ms.

For FB compared to TB, the ASD group only appeared to recruit the right inferior and middle frontal gyri (RIFG/MFG) from 425 to 475 ms, though a more lenient threshold of $p < 0.005$ demonstrated that this activation extended from 325 to 475 ms.

When comparing the two groups, as with our ROI analyses, children with ASD showed reduced activation in the LTPJ from 325 to 375 ms and 425 to 475 ms, though this was only evident at a lower threshold of $p < 0.005$. However, our whole-brain approach also revealed that children with ASD had increased activation compared to controls in the RIFG from 325 to 375 ms for FB more than TB (Fig. 3B; see Table 3 for a summary of the above results).

4. Discussion

Our study investigated the temporal and spatial neural dynamics of FB processing in children with and without ASD. We demonstrate that compared to TD children, children with ASD show increased activity in the RIFG during the same period where they show decreased activity in the LTPJ, an established node of the ToM network. These results complement previous literature that shows atypical activity in the TPJ in individuals with ASD, therefore illustrating the value of MEG in examining higher-level cognitive processes, such as FB understanding.

Our ROI analyses revealed that when comparing FB to TB, children with ASD displayed reduced activity in the LTPJ from 300 to 375 ms and 425 to 500 ms, a result which was supported by our whole-brain analyses. This finding confirms previous studies demonstrating that individuals with ASD show atypical activity in this region of the ToM network (Kana et al., 2015; Oberwelland et al., 2017; von dem Hagen et al., 2014) and adds crucial timing information that is currently lacking. Our results showing the timing of the LTPJ in the TD and ASD groups suggest that rather than having a delayed response in the LTPJ, children with ASD recruit the LTPJ less than controls during FB reasoning, and that they do not activate the LTPJ more for FB compared to TB. The lack of activation differences in the LTPJ between the FB and TB conditions may reflect neural deficits in this area in children with ASD, such that the LTPJ has similar activation profiles for FB and TB due to a non-specific response for beliefs, a hypothesis that will need to be tested in future studies.

Whereas we found decreased activation in the LTPJ in our ASD group, others have generally found diminished activity in the RTPJ in individuals with ASD compared to controls (Kirkovski et al., 2016; Lombardo et al., 2011; Murdaugh et al., 2014; Pantelis et al., 2015),

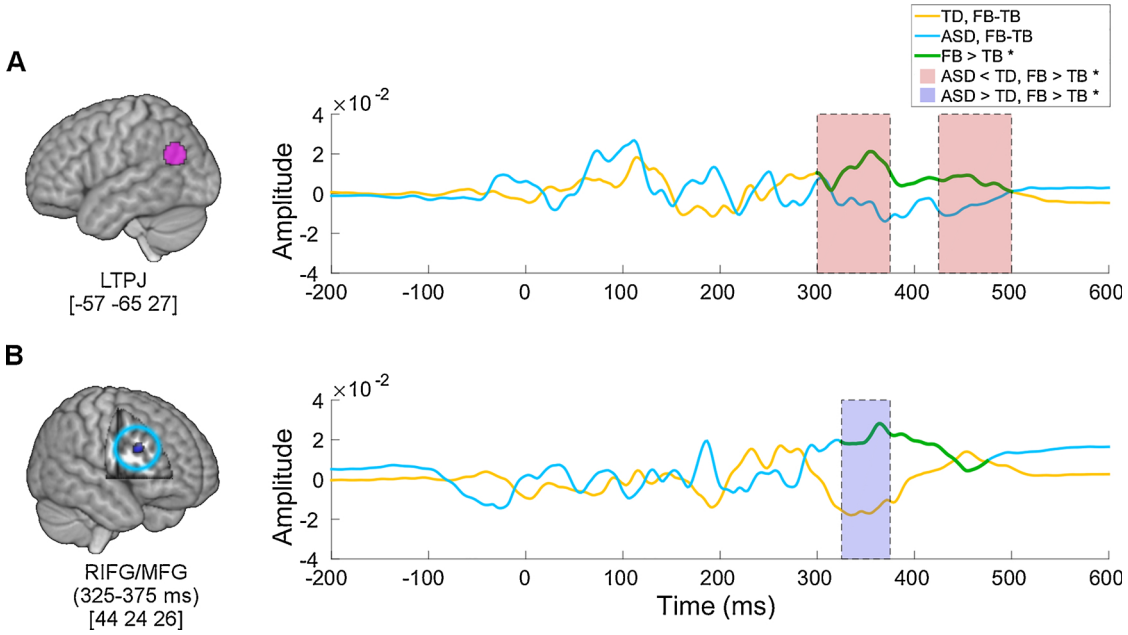

Fig. 3. Time courses of activation for the FB condition, with activity in the TB condition subtracted out, for each group (TD in yellow, ASD in blue). The horizontal axis (time) is scaled relative to the onset of the second picture stimulus. Sections of the time courses that are in green indicate time windows in which brain activity was significantly greater for FB compared to TB. Shaded rectangles denote time windows in which brain activity was significantly greater for one group compared to the other (ASD < TD in red, ASD > TD in dark blue). Coordinates are given in MNI space. (A) Time course for the LTPJ ROI (purple circle), with * $p < 0.05$, corrected for multiple comparisons over the ROI volume. (B) Time course for the RIFG/MFG cluster (dark blue circle) from the whole-brain analyses, with * $p < 0.005$, uncorrected, though the group difference (ASD > TD) is significant at $p < 0.001$, uncorrected. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article).

although a few found decreased activity in both TPJs (Kana et al., 2014; Redcay et al., 2013) or did not find any group differences in either TPJ (Dufour et al., 2013; Kana et al., 2009; O'Nions et al., 2014). We speculate that this discrepancy in laterality may be an effect of development, as our study is one of very few that has studied FB in children with ASD using functional neuroimaging, and more research into the neural development of ToM will help validate the lateralization (or lack thereof) of ToM processing in the TPJ.

Our whole-brain analyses further revealed that children with ASD activated the RIFG significantly more than TD children from 325 to 375 ms, which overlapped with one of the time periods in which children with ASD showed decreased activation in the LTPJ relative to the TD group. This concurrent increased activation in the RIFG and inactivation of the LTPJ implies that the RIFG may be recruited to compensate for deficits in the LTPJ, which has been postulated by other ToM-related studies that have also found increased RIFG activation in ASD (Colich et al., 2012; Martineau et al., 2010; Wang et al., 2006). Since the RIFG is typically and consistently implicated in inhibitory control (Aron et al., 2003; Vidal et al., 2012) and salience detection (Hampshire et al., 2010), we hypothesize that instead of using the LTPJ to understand the thoughts of others, possibly due to impairments in the LTPJ, children with ASD may be employing the RIFG to inhibit their own thoughts in order to reason about another's perspective or mindset to perform equally as well as controls on our FB task.

Inhibition is thought to play a major role in FB (Devine and Hughes, 2014), specifically in resolving conflicting perspectives in TD individuals (see Hartwright et al., 2016; Samson et al., 2015; and Schurz and Tholen, 2016 for a discussion). While we did not see significant RIFG activation in our sample of TD children for FB reasoning, it may be that, as suggested by Schurz and Tholen (2016), inhibition is being

Table 2
Significant results from ROI analyses of brain activations in the TD and ASD groups during the theory-of-mind task, for the false belief versus true belief condition (FB > TB).

Time window (ms)	TD, FB > TB						ASD, FB > TB†					Group difference	
	Region		MNI coordinates			Z	Region	MNI coordinates			Z	Direction	d
			x	y	z			x	y	z			
300-350	L	TPJ	−52	−72	28	2.55						ASD < TD	0.39
325-375	L	TPJ	−52	−68	22	3.01						ASD < TD	0.45
350-400	L	TPJ	−54	−68	20	2.73							
	R	Precuneus	10	−62	44	2.57							
375-425	L	TPJ	−50	−66	32	2.37							
400-450	L	TPJ	−50	−68	32	2.48							
425-475	L	TPJ	−50	−70	28	2.92						ASD < TD	0.46
450-500	L	TPJ	−54	−68	20	2.60						ASD < TD	0.38

Reported peaks and group differences are significant at $p < 0.05$, corrected for multiple comparisons over the ROI volume. Effect sizes are reported for any significant group differences. Abbreviations: TPJ = temporoparietal junction.

† No significant effects were found in this contrast in the ASD group.

Table 3
Significant results from whole-brain analyses of brain activations in the TD and ASD groups during the theory-of-mind task, for the false belief versus true belief condition (FB > TB).

Time window (ms)	TD, FB > TB						ASD, FB > TB						Group difference	
	Region		MNI coordinates			Z	Region		MNI coordinates			Z	Direction	d
			x	y	z				x	y	z			
275–325	R	MFG	42	46	4	3.14								
325–375	**R**	**Precuneus**	**8**	**−48**	**70**	**3.88**								
	L	MTG/AG	−50	−70	18	3.13							*ASD < TD*	*0.37*
							R	*IFG*	*44*	*28*	*24*	*2.97*	ASD > TD	0.51
350–400	**R**	**Precuneus**	**8**	**−46**	**72**	**3.95**								
	L	MOG	−54	−76	12	3.10								
							R	*MFG*	*46*	*30*	*22*	*2.65*		
375–425	R	MOG	42	−84	16	3.43								
	R	ITG	48	−44	−16	3.40								
	L	MOG	−48	−88	0	3.31								
	L	Precuneus	−2	−44	72	3.21								
400–450	R	ITG	50	−42	−16	3.68								
	L	MOG	−48	−88	2	3.40								
	R	MOG	34	−78	10	3.12								
							R	*IFG*	*48*	*32*	*12*	*3.00*		
425-475	L	MOG/MTG	−46	−84	12	3.47							*ASD < TD*	*0.45*
	R	ITG	52	−48	−24	3.35								
							R	IFG	50	34	10	3.15		
450–500	R	MFG	38	48	24	3.24								

Reported peaks and group differences are significant at $p < 0.001$, uncorrected. Peaks and group differences in *italics* are significant at $p < 0.005$, uncorrected, and those in **bold** are significant at $p < 0.05$, family-wise error corrected. Effect sizes are reported for any significant group differences. Abbreviations: AG = angular gyrus; IFG = inferior frontal gyrus; ITG = inferior temporal gyrus; MFG = middle frontal gyrus; MOG = middle occipital gyrus; MTG = middle temporal gyrus.

utilized for both the FB and TB conditions. Alternatively, Schulte-Rüther et al., (2008) examined sex differences in recruitment of brain areas for empathy in adults and found greater RIFG activation in females and increased LTPJ activity in males, and since most participants in our study were male, this finding may explain why our group of TD children did not show RIFG activity, as well as why there appears to be a reliance on LTPJ rather than RTPJ.

Nevertheless, the use of the RIFG by children with ASD as a compensatory mechanism for FB reasoning is not implausible, as inhibitory processes mediated by the RIFG appear to be linked to FB understanding. It has been speculated that individuals with ASD with intact executive functions, such as inhibition, may utilize these abilities to mitigate their social deficits (Livingston and Happé, 2017), as executive functions have been shown to predict ToM abilities (Kimhi et al., 2014; Pellicano, 2010) and adaptive skills (Pugliese et al., 2015) in the ASD population. Given that our ASD group had levels of inhibitory control on par with the TD group, as measured by the NEPSY-II, it is likely that their inhibitory skills may have supported their ability to perform well on our FB task.

It has also been proposed that in individuals with ASD who have strong executive function skills, the prefrontal cortex, which includes the RIFG, may help compensate for deficits by recruiting alternative neural systems (Johnson et al., 2015). In our case, the RIFG may play a common role in the ASD group of engaging alternative brain regions for FB processing, the particulars of which may differ between individuals, hence why we only see greater activity in the RIFG in our sample of children with ASD, but not other brain areas. On the other hand, it is also plausible that this increased RIFG activity may indicate higher cognitive demands of this task on inhibitory processes in the ASD group, as individuals with ASD often have difficulties with inhibition (Demetriou et al., 2018; Geurts et al., 2014) and show atypical brain activity during inhibitory control tasks (Duerden et al., 2013; Kana et al., 2007; Solomon et al., 2014). In light of these varying interpretations, future work investigating FB and inhibition concurrently will be needed to tease apart the functional role of the RIFG in FB processing in ASD.

In conclusion, the present study provides evidence towards the theory that individuals with ASD have atypical neural activity during FB processing, and demonstrates that these differences are present in childhood. Building upon the current literature, we found that children with ASD have decreased activity in the LTPJ and increased activity in the RIFG compared to TD children. Using MEG's excellent temporal resolution, we demonstrate that these differences occurred simultaneously, likely indicating that the RIFG acts as a compensatory mechanism. Our task differed slightly from traditional FB tasks, since in our task the initial placement of the object is inferred rather than explicitly shown to the character. However, this task was adapted from one that found FB deficits in children with traumatic brain injury (Dennis et al., 2013), and the fact that our task elicited differences in a brain region involved in FB reasoning between children with and without ASD supports its validity in inducing FB processes. Nonetheless, as only children who performed above chance level on our FB task were included in this study, these findings are more representative of high-functioning children with ASD. In addition, while the effects of age and IQ were accounted for in our study, our age range of children (8–12 years) did not allow for consideration of the neurodevelopmental changes that children undergo year-to-year (Schäfer et al., 2014), and future research should examine brain activity in young children over narrower age ranges. We were also unable to conduct brain-behaviour correlations between our neuroimaging findings and an established ToM assessment, as the NEPSY-II ToM does not provide scaled scores for children between the ages of 8–12 years; further research would be important to validate these results. Moreover, as this study is the first to investigate the temporal and spatial correlates of FB in children with ASD, future work is needed to replicate and generalize our findings to other ToM tasks and better understand the relationship between ToM and inhibition throughout development, both in children with and without ASD.

Conflict of Interest

None.

Funding

This work was funded by Canadian Institutes of Health Research grants MOP-119541 and MOP-142379 to M.J.T and a studentship from the Research Training Competition (RESTRACOMP) at the Hospital for Sick Children awarded to V.Y.

Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at https://doi.org/10.1016/j.dcn.2018.08.001.

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Developmental Cognitive Neuroscience

Volume 45, Issue , October 2020, Page

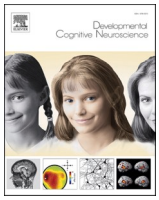
DOI: https://doi.org/10.1016/j.dcn.2020.100842

Developmental Cognitive Neuroscience 45 (2020) 100842

Contents lists available at ScienceDirect

Developmental Cognitive Neuroscience

journal homepage: www.elsevier.com/locate/dcn



Erratum

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The purpose of this publisher correction is to inform readers that the final version of the articles linked with this correction were replaced with a corrected version in March 2019. The corrected version contains a Declaration of Interest statement which the publisher inadvertently omitted from the original version.

The Publisher apologizes for any inconvenience this may cause."

DOIs of original article: https://doi.org/10.1016/j.dcn.2017.08.007, https://doi.org/10.1016/j.dcn.2017.09.008, https://doi.org/10.1016/j.dcn.2017.10.007, https://doi.org/10.1016/j.dcn.2017.11.006, https://doi.org/10.1016/j.dcn.2018.05.004, https://doi.org/10.1016/j.dcn.2018.09.001, https://doi.org/10.1016/j.dcn.2017.07.002, https://doi.org/10.1016/j.dcn.2017.07.007, https://doi.org/10.1016/j.dcn.2017.09.002, https://doi.org/10.1016/j.dcn.2018.06.003, https://doi.org/10.1016/j.dcn.2018.07.003, https://doi.org/10.1016/j.dcn.2018.08.001, https://doi.org/10.1016/j.dcn.2017.02.007, https://doi.org/10.1016/j.dcn.2017.07.006, https://doi.org/10.1016/j.dcn.2017.08.008, https://doi.org/10.1016/j.dcn.2017.10.006, https://doi.org/10.1016/j.dcn.2017.07.001, https://doi.org/10.1016/j.dcn.2017.08.010, https://doi.org/10.1016/j.dcn.2017.10.001, https://doi.org/10.1016/j.dcn.2017.11.009, https://doi.org/10.1016/j.dcn.2017.12.002, https://doi.org/10.1016/j.dcn.2017.02.008, https://doi.org/10.1016/j.dcn.2017.02.010, https://doi.org/10.1016/j.dcn.2016.12.001, https://doi.org/10.1016/j.dcn.2017.07.003, https://doi.org/10.1016/j.dcn.2017.08.003, https://doi.org/10.1016/j.dcn.2017.10.005, https://doi.org/10.1016/j.dcn.2017.09.005, https://doi.org/10.1016/j.dcn.2018.05.008, https://doi.org/10.1016/j.dcn.2017.01.002, https://doi.org/10.1016/j.dcn.2017.01.004, https://doi.org/10.1016/j.dcn.2017.01.010, https://doi.org/10.1016/j.dcn.2017.07.004, https://doi.org/10.1016/j.dcn.2017.08.001, https://doi.org/10.1016/j.dcn.2017.10.003, https://doi.org/10.1016/j.dcn.2017.05.007, https://doi.org/10.1016/j.dcn.2018.03.002, https://doi.org/10.1016/j.dcn.2018.10.002, https://doi.org/10.1016/j.dcn.2017.01.003, https://doi.org/10.1016/j.dcn.2017.10.009, https://doi.org/10.1016/j.dcn.2017.11.011, https://doi.org/10.1016/j.dcn.2018.05.007, https://doi.org/10.1016/j.dcn.2018.07.002, https://doi.org/10.1016/j.dcn.2017.11.001, https://doi.org/10.1016/j.dcn.2018.07.004, https://doi.org/10.1016/j.dcn.2017.01.001, https://doi.org/10.1016/j.dcn.2017.01.005, https://doi.org/10.1016/j.dcn.2017.01.008, https://doi.org/10.1016/j.dcn.2017.08.009, https://doi.org/10.1016/j.dcn.2017.11.003, https://doi.org/10.1016/j.dcn.2017.11.008, https://doi.org/10.1016/j.dcn.2018.02.004, https://doi.org/10.1016/j.dcn.2018.10.001, https://doi.org/10.1016/j.dcn.2018.05.010.

https://doi.org/10.1016/j.dcn.2020.100842

Available online 28 August 2020