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Research Report

Beta oscillations precede joint attention and correlate with mentalization in typical development and autism

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ABSTRACT

A precursor of adult social functioning is joint attention (JA), which is the capacity to share attention on an object with another person. JA precedes the development of the capacity to attribute mental states to others (i.e., mentalization or theory of mind). The neural mechanisms involved in the development of mentalization are not fully understood. Electroencephalographic recordings were made of children while they watched stimuli on a screen and their interaction with the experimenter was assessed. We tested whether neuronal activity preceding JA correlates with mentalization in typically developing (TD) children and whether this activity is impaired in children with autistic spectrum disorder (ASD) who evidence deficits in JA and mentalization skills. Both groups exhibited JA behavior with comparable frequency. TD children displayed a higher amplitude of negative central (Nc) event-related potential preceding JA behavior (~500 msec after stimuli presentation), than did the ASD group. Previous to JA behavior, TD children demonstrated beta oscillatory activity in the temporoparietal region, while ASD children did not show an increase in beta activity. In both groups, the beta power correlated with mentalization, suggesting that this specific neuronal mechanism is involved in mentalization, which used during social interaction.

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1. Introduction

The extremely complex social functioning of adults arises from several early onset behaviors that are the building blocks for social behavior. One of these social precursors is the capacity to share attention on an object with another person. This is called joint attention (JA) (Charman et al., 2000; Happé & Frith, 2014; Soto-Icaza, Aboitiz, & Billeke, 2015). JA can be responding or initiating (Mundy & Jarrold, 2010). The former refers to the ability to follow the direction of the gaze and gestures of others in order to share a common point of reference; the latter refers to the ability to spontaneously point, show and use eye contact to share the experience of an object. While evidence shows that responding JA emerges at around 6–9 months of age (Butterworth & Cochran, 1980; Kopp & Lindenberger, 2011; Morgan, Maybery, & Durkin, 2003; Striano, Reid, & Hoehl, 2006), initiating JA does not appear until around the end of the first year of life/beginning of the second year of life (Mundy, 2017). While it is agreed that responding JA encompasses a visual readjustment of a child's gaze in accordance with the change in the focus of visual attention of an adult (Butterworth & Jarrett, 1991), there is no consensus on whether initiating JA involves a child's understanding of other's intentions and thoughts (Moore, 1994). In spite of this controversy, it is well-known that JA plays a key role in social development, since it results in a coordinated social behavior where the child shares a common experience with his/her partner without obtaining something from him/her (Mundy, Card, & Fox, 2000), revealing its reciprocity quality (Oberwelland et al., 2017). Moreover, several studies have shown that JA ability precedes the development of the capacity to attribute mental states to others, that is, mentalization (Happé & Frith, 2014; Krall et al., 2016; Mundy et al., 2000; Soto-Icaza et al., 2015).

Studies of neurobiological JA mechanisms have provided two main lines of evidence. One is that JA is related to brain activity associated with attention control in typically developing (TD) infants, which involves the dorsal attentional network and the fronto-parietal network (Eggebrecht et al., 2017; Mundy, 2017). These networks include lateral parietal areas (the intraparietal sulcus, IPS) and lateral frontal areas (dorsolateral prefrontal cortex and frontal eye fields, FEF; Eggebrecht et al., 2017; Power et al., 2011). During JA, these networks probably participate in the cognitive control related to exterocentive processing and contribute to maintain the attentional focus. Specifically, studies have demonstrated that responses to JA modulate the negative central (Nc) component (Kopp & Lindenberger, 2011; Striano et al., 2006), which refers to a negative deflection in frontocentral electrodes. It is thought that the Nc reflects the reorientation of the attention to salient stimuli (Richards, Reynolds, & Courage, 2010; Striano et al., 2006). Increases in the amplitude of this component have been interpreted as attention activation (arousal), elicited by an environmental stimulus (Soto-Icaza et al., 2015; Striano & Reid, 2006). Increases in arousal influence subsequent cognitive processes like memory and learning (Hirotani, Stets, Striano, & Friederici, 2009; Richards, 2003). For example, attended objects elicit a greater Nc amplitude, which correlates with the subsequent recognition of these objects (Hirotani et al., 2009; Kopp & Lindenberger, 2011; Richards, 2003). Several studies have shown a strong association between Nc amplitude and recognition processing (De Haan, Johnson, & Halit, 2007; Luyster, Powell, Tager-Flusberg, & Nelson, 2014; Nelson & McCleery, 2008; Webb, Long, Nelson & Webb, 2005). The Nc seems to be generated in frontal cortical areas, such as the anterior cingulate cortex (ACC) and the inferior frontal gyrus (IFG) (Richards et al., 2010). Additionally, using oscillatory activity measures by EEG and metabolic activity by near-infrared spectroscopy (NIRS), other studies have demonstrated a crucial participation of lateral frontal and lateral parietal regions during JA in infants (Grossmann & Johnson, 2010; Grossmann, Johnson, Farroni, & Csibra, 2007; Kühn-Popp, Kristen, Paulus, Meinhardt, & Sodian, 2016; Mundy et al., 2000).

The other line of evidence associates JA with the mentalization network in both adults and children that pass false belief tests (the main marker of mentalization or theory of mind; Soto-Icaza et al., 2015). The mentalization network consists of several cortical areas that show consistent activation when people figure out other people's intentions. In typical adults, this network involves the temporoparietal junction (TPJ), the posterior superior temporal sulcus (pSTS), the dorsomedial prefrontal cortex (dmPFC), the precuneus, the temporal pole, and the inferior frontal gyrus (Schurz, Radua, Aichhorn, Richlan, & Perner, 2014). Neuroimaging research in children, adolescents and adults shows that JA activates a brain network that overlaps with the mentalization network, particularly in the TPJ, STS (Oberwelland et al., 2017) and dmPFC (Caruana, Brock & Woolgar, 2015; Oberwelland et al., 2016; Redcay et al., 2010; Saito et al., 2010; Schilbach et al., 2010). It has been proposed that practicing JA provides a unique brain–behavior experience that is necessary for sculpting the components of the neural systems for mentalization (Mundy, 2017; Mundy, Sullivan, & Mastergeorge, 2009).

A candidate structure for developing mentalization from JA is the TPJ. Comparing JA studies between infants and children, TPJ becomes specifically activated in children with mentalization capacity (Mundy, 2017). In healthy adults and adolescents this area (especially the posterior segment involving the angular gyrus; Igelström, Webb, Kelly, & Graziano, 2016; Lee & McCarthy, 2016) is a key node of the network that participates in attributing mental states to others and perceiving the perspectives and preferences of others during social interactions (Oberwelland et al., 2016; Van Overwalle, 2009). Indeed, using non-invasive brain stimulation, recent studies have demonstrated the causal role of the TPJ in mentalization processes (Donaldson, Kirkovski, Rinehart, & Enticott, 2017; Hill et al., 2017). Additionally, the TPJ (probably the anterior segment involving the supramarginal gyrus (Igelström et al., 2016; Lee & McCarthy, 2016)) is also part of the right-lateralized ventral attentional network related to detecting task-relevant stimuli and reorienting attentional responses (Chang et al., 2013; Corbetta, Patel, & Shulman, 2008; Zamorano et al., 2017). Developmental changes in the function of this cortical area could underlie the emergence of complex mentalization skills from attentional processes. In fact, the structural connectivity between the temporoparietal and frontal regions correlates with the achievement of mentalization during childhood (Wiesmann, Schreiber, Singer, Steinbeis, & Friederici, 2017).

Oscillatory activity in the temporoparietal region could be a neuronal marker of mentalization network activity. Studies using electroencephalogram (EEG) in humans have shown that the power of beta and gamma bands in temporoparietal regions is greater when a subject points to an object to share attention on this object with another person (declarative pointing) than when the subject points to solicit a desired object from another person (imperative pointing) (Brunetti et al., 2014). Evidence indicates that the power and synchrony of brain oscillations in TD infants at rest correlate with subsequent initiation of JA (Henderson, Yoder, Yale, & McDuffie, 2002; Mundy et al., 2000). Furthermore, oscillatory brain activity in the alpha and beta ranges in the right temporoparietal region correlates with the anticipation and prediction of another person's responses and preferences (Billeke, Zamorano, Cosmelli, & Aboitiz, 2013; Park, Kim, Sohn, Choi, & Kim, 2018). Although the spatial resolution of the EEG activity is poor, source analyses have revealed that a candidate area for this activity is the TPJ (Billeke et al., 2015; Melloni et al., 2016). Hence, temporoparietal oscillatory activity in the alpha and beta ranges could reflect mentalization processing related to JA.

Our main hypothesis is that JA behavior involves the activity of attentional networks (fronto-parietal and dorsal attentional networks) and that during development this activity gradually extends into the mentalization network. The latter can be reflected in a greater power of alpha/beta oscillatory activity in the TPJ in children that have developed mentalization skills than in children that have not. Specially for initiating JA, we expect that this neural processing related to the wanting/seeking another person's attention should occur before the execution of the JA behavior. To test this hypothesis, we first tested neuronal activity in TD children preceding (and eventually triggering) JA, including attentional and mentalization networks, to determine if the activity of the latter is greater in children that have already correctly solved the false belief test. We studied EEG activity preceding JA behavior and used the Nc component as a marker of attentional control processing and oscillatory activity in the temporoparietal region as a marker of mentalization network activity. An experimental paradigm was designed to facilitate initiating children's contact with the experimenter (initiating JA, see the Methods section for further details). Generally, the paradigms designed to test responding JA are based on experimental settings where the experimenter alternates his/her gaze between the child and the object on a computer screen (Striano et al., 2006). In contrast, to test the capacity to produce social communication instead of the capacity to respond to it (initiating JA rather than responding JA), our experimental paradigm presented a novel stimulus on a computer screen while the researcher waits for the child to initiate interaction. Using this paradigm, we studied children between 3 and 4 years of age because at this age children begin to solve social problems using explicit mentalization skills.

We then studied a group of children with atypical development to obtain further insights into the neurobiological mechanisms underlying JA and mentalization, including the typical mechanisms of cognitive alterations, and the pathophysiology of atypical development (Kennedy & Adolphs, 2012). In this context, autistic spectrum disorder (ASD) plays a crucial role because alterations to JA and mentalization have been described as key features of this disorder (Mundy, 2017). Studies have demonstrated that infants with high risk of developing autism show alterations in their capacity to attend to social stimuli (Chawarska, Ye, Shic, & Chen, 2016; Jones & Klin, 2013), and that children with ASD have difficulty socializing, showing altered mentalization development, like not understanding irony, metaphors, jokes, other people's intentions and emotions in social situations (Hamilton, 2009; Kana, Libero, Hu, Deshpande, & Colburn, 2014). Neuroimaging studies have shown that subjects with ASD evidence a reduced neural response in both the TPJ and the medial prefrontal cortex during mentalization tasks (Castelli, Frith, Happé, & Frith, 2002; Kana et al., 2014; Lombardo, Chakrabarti, Bullmore, & Baron-Cohen, 2011; O'Nions et al., 2014). Children and adults with ASD exhibit an altered pattern of oscillatory brain activity, both at rest (Cornew, Roberts, Edgar, Blaskey, & Edgar, 2012; van Diessen, Senders, Jansen, Boersma, & Bruining, 2015) and during sustained attention and memory tasks (Larrain-Valenzuela et al., 2017; Stroganova et al., 2007). Adolescents with ASD present altered patterns of alpha synchronization between right temporal and central electrodes during JA, reflecting altered social performance (Jaime et al., 2016). More specifically, evidence has shown that children with ASD have deficits in initiating JA, but not necessarily in responding JA. This has been interpreted as a lack of spontaneous initiation of intentional communication (Caruana, Brock, et al., 2015; Mundy, 2017). Responding and initiating JA are also neuroanatomically differentiated. A fMRI study in typically developing children and adolescents showed that initiating JA recruited the social brain network (Oberwelland et al., 2016). In addition, a recent study showed that whereas initiating JA recruits frontal and parietal brain areas in typically developed adolescents, this was not the case for adolescents with ASD. Furthermore, those participants who showed less severe symptomatology evidenced more frontal activation in self initiating JA (Oberwelland et al., 2017). Specifically, the TPJ, which is part of this social cognition network, showed a distinct pattern of functioning from childhood to adolescence (Oberwelland et al., 2016). Interestingly, a study in adults with ASD showed a significantly different response in this brain network during responding JA condition when compared to typically developing participants. This difference was absent in initiating JA condition (Redcay et al., 2013). There are also other neuropsychiatric diseases, such as schizophrenia and frontotemporal dementia, that are associated with alterations in alpha and beta oscillation in the temporoparietal region, which correlate with social deficits (Billeke et al., 2015; Melloni et al., 2016). However, the precise neuronal impairments related to alterations in initiating JA and mentalization in ASD subjects remain unclear. In this context, identifying a potential neuronal marker of social deficit in ASD could be useful to elucidate the underlying pathophysiology. With the ASD group we explored the neuronal signatures of attentional and mentalization processing that precede JA behaviors and tested if these are differentially affected by the disorder. Specifically, we investigated whether the activity level of the mentalization network is lower in children with ASD than in TD children, which would correlate with reduced social skills.

2. Materials and methods

2.1. Participants

All methods and the experimental protocols were approved by two Ethics Committees (those of the Universidad del Desarrollo and the Pontificia Universidad Católica de Chile) and met the principles of the Declaration of Helsinki and the Local Ethical Guidelines for Research Involving Human Subjects (Chilean National Scientific and Technological Research Commission, CONICYT). All parents gave written informed consent to participate with their children in the study. The experiments were carried out at the Laboratorio de Electroencefalografía y Neuromodulación of the Division de Neurociencia del Centro de Investigación en Complejidad Social (neuroCICS) of the Universidad del Desarrollo, Santiago, Chile.

Twenty-four TD children between 3 and 4 years of age participated [12 boys, mean age 3.9 years ± .5 (SD), Table 1]. All the children were born at term and were native Spanish speakers. A TD subject was tested but was excluded from the final sample because she could not wear the EEG cap properly and so provided unreliable EEG data (i.e., electrodes should be equally spaced, with symmetry between the two hemispheres).

The exclusion criteria were indications of language impairment, and neurological or psychological/psychiatric diagnosis and treatment associated with mental health problems. Children with a family member diagnosed with ASD were also excluded from the sample. Alterations in communication and social interaction were assessed with the Autism Diagnostic Observation Schedule-2 (ADOS-2) (Gotham, Pickles, & Lord, 2009). TD participants had to score 2 or less on the ADOS-2 severity scale, which indicates no significant alterations in communication, social interaction, play, or restricted/repetitive behaviors.

Twenty children with ASD between 3 and 4 years of age participated in the study [16 boys, 4.16 ± .55 (SD), see Table 1]. Another 6 children were evaluated but were excluded from the final sample because they refused to wear the EEG cap (4 boys), did not pay attention to the stimuli on the screen (1 subject), or did not meet the inclusion criteria (1 subject). All the children with ASD were selected according to the clinical neurological evaluation following the diagnosis criteria of the Diagnostic and Statistical Manual of Mental Disorders, fifth edition (DSM-5) (American Psychiatric Association, n.d.). All ASD participants met the criteria for both persistent deficits in social communication and social interaction, and the presence of restricted, repetitive patterns of behavior, interests, or activities. ASD participants were also assessed according to the ADOS-2 severity scale to determine the severity of the alterations in communication, language, social interaction, social-emotional understanding, and repetitive or stereotyped interests. All ASD participants scored an average of 5 points, with a low to moderate level of symptoms associated with autistic spectrum disorder. Children with a history of auditory processing disorder and/or a syndromic disorder diagnosis were excluded.

TD participants were all recruited from the Center "Semillitas del Oriente" under the authority of the Dirección de Servicio de Salud Metropolitano Oriente, Santiago, Chile. ASD participants were recruited from the therapeutic center "ALTHEA Crecimiento y Desarrollo" (8 children), the Foundation "San Nectario" (1 child), and the "Centro de Rehabilitación Cognitivo, Lingüístico y Sensorial ACOLINSE" (2 children). Another 7 ASD participants were recruited through private therapists. Following the requirements of the Ethics

Table 1 – Table of demographic and behavioral data.

	TD children	TD children v/s children with ASD	*P*-value (<.05)
	TD ($n = 24$)	ASD ($n = 20$)	
Demographic data			
Participants	24 (12 boys)	20 (13 boys)	.33
Mean age (years)	3.9 ± .5 SD	4.16 ± .55 SD	.163
Behavioral data			
Mean ADOS-2 score	1 point ± .20 SD, range 1–2	5 points ± 1.28 SD, range 3–7	
Number of children that correctly solved the False Belief Task	14 (6 boys)	3 (2 boys)	**.00383**[a]
nJA trials (from valid trials[b])	52.7% ± 22.5 SD	61.8% ± 28.3 SD	.1
JA trials (from valid trials[b])	47.3% ± 22.5 SD	38.2% ± 28.3 SD	.13
JA trials from children that correctly solved the False Belief Task	41.9% ± 19.1 SD	22.1% ± 4.8 SD	.09
JA trials from children that did not correctly solve the False Belief Task	54.8% ± 25.6 SD	46.6% ± 20 SD	.38
Mean age of children that correctly solved the False Belief Task	4.1 ± .3 SD	4.69 ± .3 SD	**.0294**[a]
Mean age of children that did not correctly solve the False Belief Task		4.06 ± .5 SD	.0664

SD = standard deviation.
[a] Statistically significant difference ($p < .05$ Wilcoxon test).
[b] Valid trial.

Committees, all ASD participants were under treatment and specialist supervision.

2.2. Experimental procedure

Participation in the study had four parts. First, the parents were interviewed to gain insight into the children's social behaviors, and to obtain the signed informed consent forms. Second, a video recorded encounter was carried out between the experimenter and each individual child to assess how each child solved a classical version of the False Belief Task (Baron-Cohen, Leslie, & Frith, 1985) in order to assess mentalization abilities. The task was carried out in a single trial. If the child correctly identified where Sally would look for the marble, the experimenter asked two control questions: (1) "Where is the marble really?" (Reality Question) (2) "Where was the marble at the beginning?" (Memory Question). The task is considered successfully solved if the child answers both control questions correctly. Third, the children's electrical brain activity was measured by EEG while they solved the JA task; and finally, a certified therapist assessed the children individually, according the ADOS-2.

2.3. Experimental design

We designed an experimental paradigm to facilitate that children initiated JA with the experimenter. Children watched a computer screen in the presence of their mother and an experimenter, and their behaviors in response to novel stimuli on the screen were assessed. The distance between the child and the center of the screen was between 50 and 150 cm (Odom et al., 2010). Since evidence has shown that children with ASD have deficits in initiating JA, but not necessarily in responding JA (Caruana, Brock, et al., 2015; Mundy, Gwaltney, & Henderson, 2010), we tested the capacity for self-initiating JA. Before beginning the task, parents were instructed to remain silent except if the child explicitly looked for the interaction with him/her. The experimenter was sitting next to the child and beside the screen, willing and prepared to interact with the child. Just before the display of the stimuli began, the researcher invited the child to watch what was coming up on the screen. A co-experimenter in the adjacent room, seeing the real time behavior of the child and the experimenter through a video camera system, began each trial by displaying the target stimuli on the screen for 3 sec when the child fixed his/her gaze on the fixation stimulus for at least 1 sec. After the target stimulus was displayed, the co-experimenter manually marked the trial as a JA condition if the following 3 criteria were met: (1) the child shifted its attention from the screen to the experimenter; (2) the child alternated his/her gaze between the stimulus and the experimenter's eyes; and (3) the child either pointed to or verbalized the target object (Kopp & Lindenberger, 2011; Striano et al., 2006). The non-joint attention (nJA) condition was defined as any trial in which the child did not switch its attention to the experimenter after the target stimulus was displayed, thus maintaining the attentional focus on the target stimuli (see Fig. 1). Any trial that did not meet the criteria for nJA or JA was marked as invalid and was not used for subsequent analysis. Using the child's responses recorded on a video camera, another experimenter blind to the child diagnosis revised the co-experimenter's scoring. The reliability was high with a mean of 99.1% per subject. Inconsistent trials were not included in subsequent analyses.

2.4. Stimuli

The experiment consisted of 100 consecutive trials. In each trial, a target stimulus was randomly presented on a computer screen. The target stimuli consisted of color drawings of common toys and objects displayed in an open window. The objects were selected from open access websites and matched for overall luminance, contrast and size. The fixation stimulus was a color drawing of a closed window. Faces were not included as target stimuli because studies indicate that ASD affects facial perception and recognition (Luyster et al., 2014). We thus avoided findings due to differences in stimulus processing. Evidence indicates that the amplitude of the Nc component is affected by stimulus familiarity (De Haan et al., 2007), so each target stimulus was unique and was displayed only once during the whole task. We intendedly avoided black and white contrast, drawings with eyes or any configuration similar to eyes (e. g., the car front area with its front lights, animals, animated drawings or with human characteristics) and disturbing or frightening objects. Furthermore, knowing that children with ASD might present symptoms of hyper- or hyporeactivity to sensory inputs or unusual interests in sensory aspects of the environment (e.g., adverse response to specific sounds or visual fascination for lights or movement), our stimuli were static and noiseless. Thus, the used drawings were from diverse categories, such as clothes, fruits, transports, toys, etc. Fixation stimuli and target stimuli were aligned in the center of the screen (Fig. 1).

2.5. EEG recordings and analysis

Continuous EEG recordings were obtained with a 64-electrode Geodesic EEG System (Net Station Acquisition). Impedances were kept below 100 kΩ. Electrodes were referenced to the Cz electrode during acquisition, and the signal was digitized at 1 kHz. EEG data was then re-referenced offline to the average electrodes, and .4–30 Hz band-pass filtered. The baseline was chosen from −500 msec to 0 msec before stimulus onset for time-frequency (TF) analysis and −300 msec to 0 msec for ERP analysis, and a time-window from 0 msec to 1000 msec after stimulus onset was analyzed. Artifacts were first automatically detected using a threshold of 150 μV and a power spectrum greater than 2 SD for more than 10% of the frequency spectrum (.4–30 Hz). Trials that included blinking and other artifacts detected by visual inspection of the signal were eliminated. The mean of artifact-free trails was 58.5 for TD children [36–78] and 62.5 for children with ASD [27–89] (Wilcoxon test, $p = .3$, $n = 44$). An average of 26 [7–59] JA trials per subject were analyzed for TD children, while an average of 21.3 [7–55] JA trials per subject were analyzed for ASD children (Wilcoxon test, $p = .2$, $n = 44$). There was no difference in artifact free trials between JA and nJA conditions (TD: .63 and .58, $p = .4$, ASD: .60 and .62 $p = .5$).

For the modulation of the Nc amplitude we explored the area under the curve in frontal and central electrodes, based

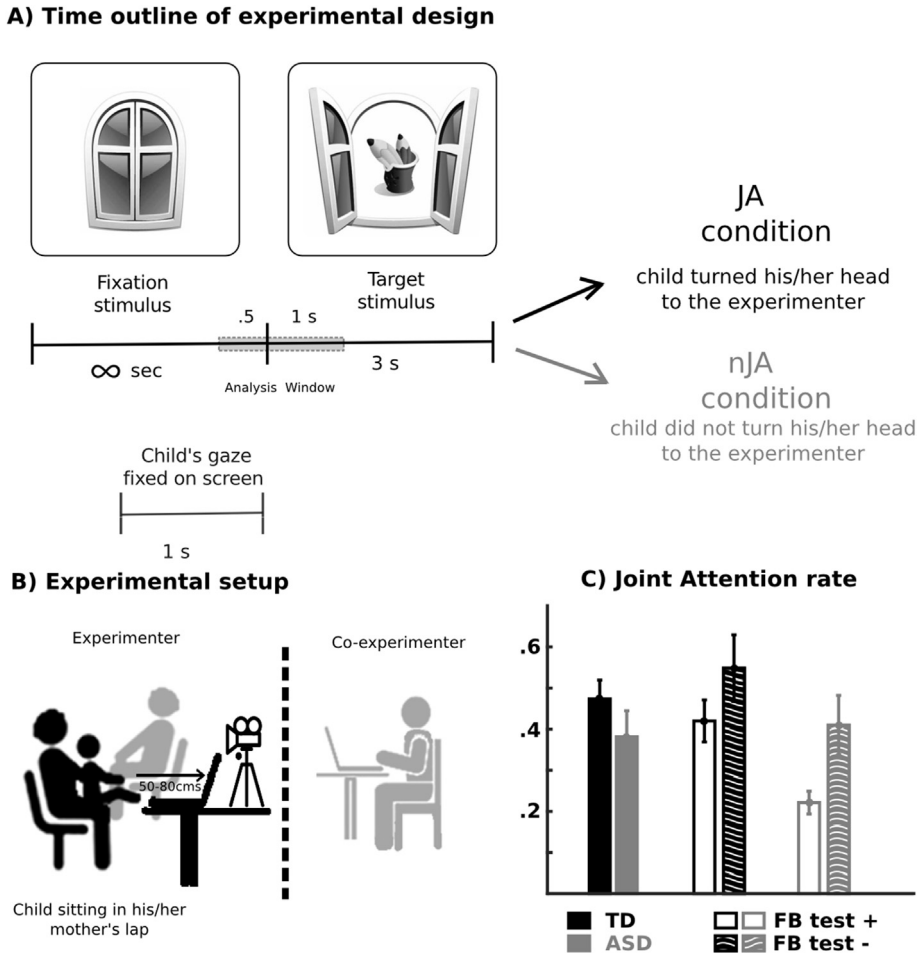

Fig. 1 – (A) Time outline of experimental design. Each trial began with a fixation stimulus displayed until the child fixed its gaze on the stimulis for at least 1 sec, whereupon the target stimulus appeared for 3 sec. The co-experimenter registered the trials in which the child shifted its attention to the experimenter. The experiment consisted of one block of 100 trials with unique target stimuli, which were presented to each child in random order. (B) Experimental setup. Children were sitting in their mother's lap while watching a computer screen. The experimenter was sitting next to the child and beside the screen, willing and prepared to interact with the child. A co-experimenter was in the adjacent room looking at the child, the mother and the experimenter through a video camera system in order to present the target stimuli every time the child fixed its gaze on the fixation stimuli for at least 1 sec. The child's responses were marked by the co-experimenter and also video recorded. (C) Rate of Joint Attention behavior separated by groups (in black, TD: typically developed children and, in gray, ASD: Autistic spectrum disorder children) and mentalization achievement (FB test -: Children who did not correctly solve the False belief test, FB test +: Children who correctly solved the False belief test).

on an *a priori* temporo-spatial region of interest (ROI) suggested by previous work (Kopp & Dietrich, 2013; Luyster et al., 2014; Striano et al., 2006; Webb, Long, Nelson, Webb, & N, 2005). The ROIs consisted of bilateral fronto-central electrodes (E14, E15, E53, E57; 10/10 system equivalence: FC3, FC5, FC4, FC6), bilateral central channels (E7, E16, E54, E51; 10/10 system equivalence: FC1, C1, FC2, C2), and midline electrodes (E4, Cz; 10/10 system equivalence: Cz, FCz), analyzed between 300 and 700 msec after presenting stimuli.

The TF analysis was made with Wavelet transform, using a five-cycle Morlet wavelet from 1 Hz to 30 Hz using 1 Hz of frequency resolution and 5 msec of temporal resolutions. The spectral power for each subject was baseline-normalized (dB) and used as a dependent variable in a single-trial model (first-level analysis), with two regressors and an interaction given by

$$Power(f,t) = b_1 + b_2\,JA + b_3\,pJA + b_4\,JA{*}pJA \quad (1)$$

for TD study, and

$$power(f,t) = b_1 + b_2\,First\_JA + b_3\,JA \quad (2)$$

for ADS-TD study (see more details in the Supplementary Material).

The source of the EEG signal was determined by applying a weighted minimum norm estimate inverse solution with unconstrained dipole orientations using the ERP signal for each subject and condition, and the single-trial signal. The individual head model was calculated using a tessellated

cortical mesh template derived from a template of 3-year-old children (Neurodevelopmental MRI Database of the University of South Carolina (Sanchez, Richards, & Almli, 2012), 3 × 4000 sources constrained to the segmented cortical surface (three orthogonal sources at each spatial location). Since we did not have the individual anatomy to calculate the head model, the spatial precision of the source estimation is poor. To minimize the possibility of erroneous results, source estimations were only shown if we found both statistically significant differences at the electrode level and differences at the source levels, which survived a multiple comparison correction (False Discovery Rate, FDR, $q < .05$, applied across the vertices). For visualization, all source results were projected on a high-resolution cortical mesh (~180,000 vertex).

We also carried out region of interest analyses for two ROIs, the Angular Gyrus/Posterior Superior Temporal Sulcus (for TPJ ROI), and the Intraparietal Sulcus (for IPS ROI). These ROIs were defined as the intersection between the significant source of the JA regressor for the TD group with the corresponding anatomical regions. These regions were extracted using the Destrieux segmentation provided by FreeSurfer software.

2.6. Statistical analysis

Non-parametric tests were used because most of the tested variables did not meet normal distribution (Kolmogorov–Smirnoff test), and the Wilcoxon test was used to determine differences between means. A random distribution of simulated groups of participants was calculated to estimate the probability that the results were due to chance (permutation test). For ERP analyses, we also computed non-parametric tests (the Wilcoxon signed-rank test for paired comparisons and the Wilcoxon sum-rank test for unpaired comparisons) of the means of the trials of each subject per condition. The results of the analysis without *a priori* hypothesis related to the time or location of the modulation were corrected using a cluster-based permutation test (see below). For the TF analysis, t-value TF charts were used as inputs for the second level analysis for each regressor and subject. The TF charts were then tested using the Wilcoxon test (paired to test if the mean of the t-value was other than zero, or unpaired to test differences between groups). Finally, we corrected these results for multiple comparison using a cluster-based permutation test (Maris & Oostenveld, 2007) (see more details in Supplementary Material). We obtained permutation distributions by randomly permuting the two conditions for each subject (paired test) or permuting the subject for each group (unpaired test), thereby eliminating any systematic difference between the conditions or groups. The same number of trials were used in each permutation as in the original data (for each condition per subject) to control for possible statistical bias due to a different number of trials per condition. After each permutation, a Wilcoxon signed-rank test was computed. After 1000 permutations, we estimated cluster-level significance as the rate of elements of the permutation distribution greater than the observed cluster-level significance. For the source analysis we corrected the results using FDR ($q < .05$) across the vertices.

2.7. Software and data

Behavioral statistical analyses and EEG signal processing were done in MATLAB using in-house scripts (Zamorano et al., 2014) (LAN toolbox, available at http://neuroCICS.udd.cl/), BrainStorm (Tadel, Baillet, Mosher, Pantazis, & Leahy, 2011) and OpenMEEG toolboxes (Gramfort, Papadopoulo, Olivi, & Clerc, 2011). The datasets generated and/or analyzed in this study are available at http://neuroCICS.udd.cl/and from the corresponding authors upon reasonable request.

3. Results

3.1. TD children

3.1.1. Behavioral results

According to the developmental trajectory of mentalization skills, TD children that explicitly evidenced mentalization in the False Belief task had a higher mean age than children that did not (Wilcoxon test, $p = .04$, Spearman rho = .41, d = .89). When solving the JA task during the experimental EEG session, TD children responded to target stimulus with JA behavior and nJA behavior with similar frequency ($p = .37$, Table 1). No significant gender difference nor differences between children who correctly solved and who did not correctly solve the False Belief test were found for the latter parameters ($p > .2$, Table 1).

When children showed JA behavior, this usually was present for several consecutive trials. In other words, once children responded by sharing the target stimuli with the experimenter, this behavior lasted for various consecutive trials. Since consecutive JA trials indicate a different social interaction than those from a single JA trial, we determined the magnitude and frequency of JA trials. This analysis revealed that only approximately 20% of the JA trials occurred in isolation (i.e., a single JA trial without a prior or subsequent JA trial, $p = 8.53e\text{-}5$, Supplementary Figure 1). Specifically, TD children shared an average of 2.75 JA trials consecutively with the experimenter, which is higher than the number by chance ($p < .001$, permutation test). This revealed that TD children often remained engaged in sharing their interest with the experimenter over an extended period of time. No differences were found in the parameters of JA behaviors between children that correctly solved and children that did not correctly solve the false belief task (frequency of JA, Wilcoxon test $p = .18$, Spearman's rho = −.28; mean of consecutive JA: $p = .16$, rho = −.29; frequency of isolated JA behavior: $p = .12$, rho = .3).

3.1.2. ERP results

All the EEG signal were analyses centered above stimuli presentation and not around the execution of JA behavior in order to separate the neuronal activity related to social processing from the one related to movement preparation. We assessed modulation in the Nc component based on prior JA work that has shown that this component is modulated in children (Kopp & Dietrich, 2013; Luyster et al., 2014; Striano et al., 2006; Webb et al., 2005). We first explored modulations in Nc amplitude (area under the

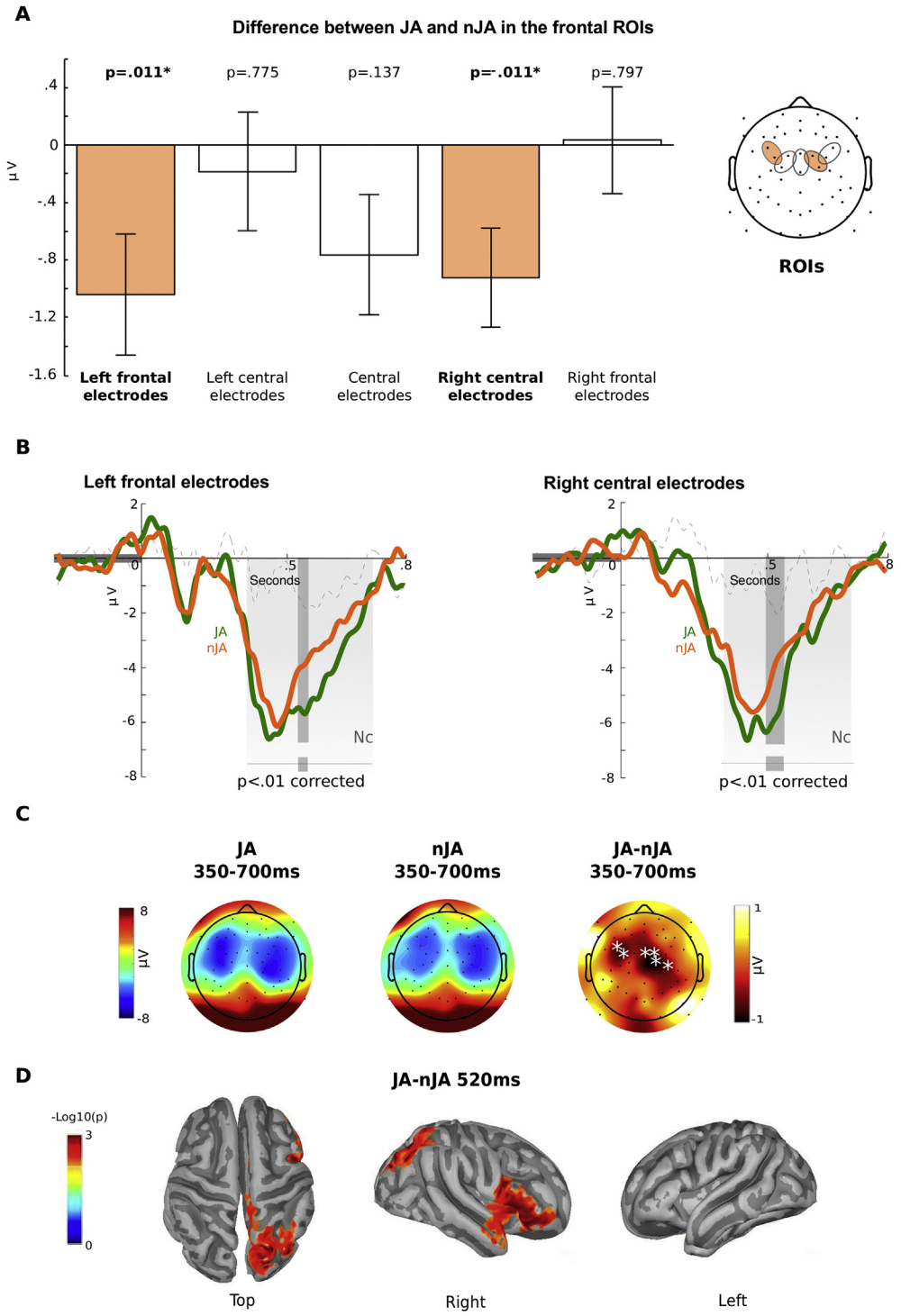

Fig. 2 – Electrical brain activity of the TD children in right central electrodes. (A) Bar plot show the differences in the five frontal ROIs contrasting JA and nJA conditions. (B) ERPs of the two significant ROIs. The gray region indicates a significant area (Cluster-based permutation test). (C) Topographic distribution of the significant cluster of Nc activity (350 msec - 700 msec), as shown in (A–B). Asterisks indicate significant electrodes (Cluster-based permutation test). (D) Estimated sources in the peak of the JA – nJA difference, as shown in (B). Error bars represent 95% confidence interval.

curve) in *a priori* temporo-spatial regions of interest (ROI, see methods and Fig. 2A). We found greater negativity for the JA condition in the right central and left fronto-central ROIs. To avoid false-positive results, these differences were computed in the same spatial *a priori* ROI without the temporal assumption (Wilcoxon test and cluster-based permutation test, Fig. 2B). The statistical differences were likewise computed using the same temporal *a priori* ROI without the spatial assumption (Wilcoxon test and cluster-based permutation test, Fig. 2C). The results were

significant for both analyses. Notably, the differences took place from 490 msec to 560 msec (Fig. 2C). The source analysis showed activity in the right parietal region (including the IPS) and in the right IFG (Wilcoxon test and FDR q < .05, Fig. 2D) during the peak of these differences at 520 msec. Finally, the individual amplitude of the Nc component in both right and left central electrodes (area under the curve between 300 and 700 msec) was isolated and correlated with the two ADOS scores and mentalization. We found no significant correlations in these analyses (Spearman correlation, $p > .2$).

3.1.3. *Time-frequency results*

A single-trial model was used to analyze brain oscillations related to JA (see Equation (1)). Since JA trials are usually consecutive, and the first trial in the sequence can be interpreted as the initiation of social interaction. We included the child's behavior in the preceding trial as a regressor in the model. The model used the behavior in the current trial (JA regressor, accounting for the power modulation when a JA trial occurred), the behavior in the preceding trial (pJA regressor, indicating the power modulation when JA occurred in the preceding trial), and the interaction between them (JA*pJA) as regressors (see Fig. 3A; for further details see Methods and an earlier work, Billeke et al., 2015).

In relation to the JA regressor, there was a significant increase in the power of the beta band (15–25 Hz) between .4 sec and .55 sec after the stimuli in the right parietal electrodes ($p < .01$, Wilcoxon test and cluster-based permutation test, n = 24, Fig. 3B). No significant cluster was found for the alpha band. The source analysis revealed that the most important cerebral region contributing to this power modulation was the right temporoparietal cortex (see Fig. 3D). Concerning the interaction (JA*pJA), that is, when a JA trial was preceded by another JA trial, we found a significant decrease in beta power (15–25 Hz) between .4 and .55 sec in the right parietal electrodes ($p < .01$, Fig. 3C and E).

The source analysis revealed that both the right temporoparietal region and the IPS were the main brain regions associated with the beta power modulation (Fig. 3D–E). This suggests that beta power modulation occurred mainly in the first JA trial of a sequence. A ROI analysis of the right TPJ and the right IPS indicated that the beta power was greater in the first JA trial than that in subsequent consecutive JA trials for the TPJ ($p < .05$, Fig. 3G), although not for the IPS ($p > .05$). We determined whether the beta activity modulation found in the

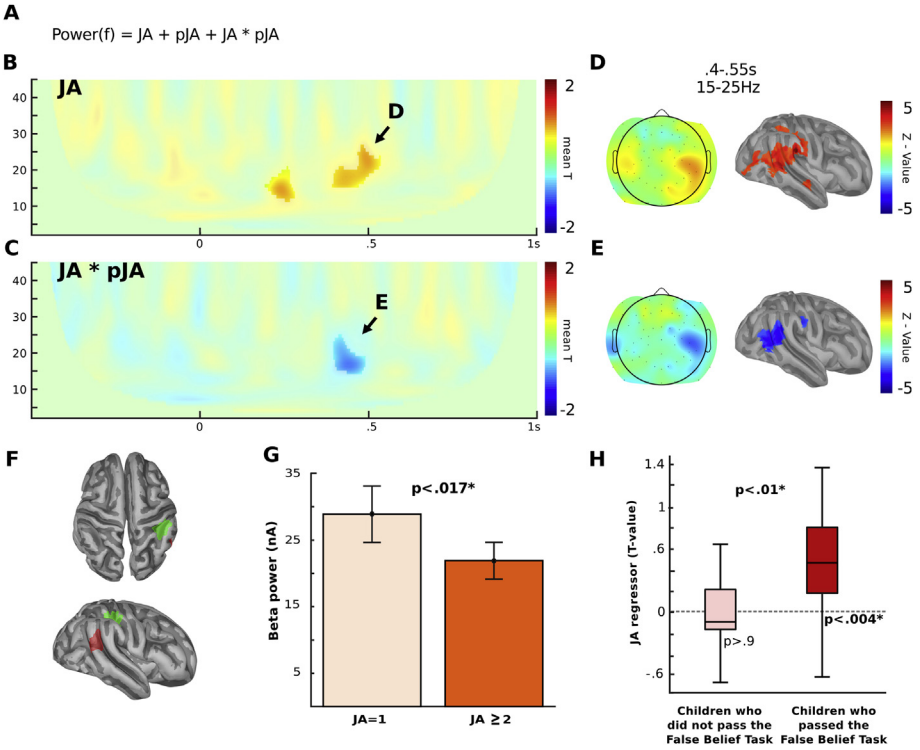

Fig. 3 – Oscillatory brain activity of the TD children. (A) Model used to estimate the activity related to JA in current trials and JA in the preceding trials (pJA). (B) Time–frequency chart of JA regressor, i.e., JA irrespective of preceding behavior. (C) Time–frequency chart of the JA*pJA regressor, i.e., JA preceded by another JA (pJA). (D) Scalp distribution and estimated sources of the significant cluster of beta activity (15–25 Hz, .5–.55 sec) regarding the JA regressor, as shown in (B). (E) Scalp distribution and estimated sources of the significant cluster of beta activity (15–25 Hz, .5–.55 sec) regarding the JA*pJA regressor, as shown in (C). (F) In red, ROI of the maximum activity found in the JA regressor in the inferior parietal region. In green, ROI of maximum activity found in the JA regressor in the intraparietal sulcus. (G) Modulation of beta power between the first and subsequent JAs occurring in sequences. (H) Brain activity in the temporoparietal region during the JA condition in children that evidenced explicit mentalization and those that did not. *$p < .05$ Wilcoxon test. Error bars represent 95% confidence interval.

group analyses was modulated by the development of explicit mentalization by comparing the power in the right TPJ of children that had evidenced explicit mentalization to that of children that had not. Children with mentalization demonstrated significantly greater involvement of beta activity in the right TPJ (Wilcoxon test, $p < .01$, Fig. 3H). Although the correlations between JA behavioral parameters and the mentalization skill were not significant, these can be a confounding factor for the preceding result. For this reason, we additionally calculated a Spearman's partial correlation in order to correct for the covariance between JA parameters and mentalization skill. Interestingly, using the parameter that showed the greatest correlation with mentalization (relative frequency of isolated JA), the correlation between beta activity in TPJ and mentalization is still significant (rho = .44, $p = .03$, d = .97, Supplementary Table 1). Two additional analyses were carried out to test the specificity of this activity, which included IPS ROI activity, the subjects' ages and the frequency of isolated JA. The robust logistic regression indicated that the subjects' ages ($T_{21} = 2.8$, $p = .01$, d = 1.1), IPS activity ($T_{21} = -2.2$, $p = .039$, d = .9) and TPJ activity ($T_{21} = 2.55$, $p = .019$, d = 1) correlated with the development of mentalization. Similar results were obtained using the Spearman partial correlation (TPJ: rho = .58, $p = .005$, d = 1.4; IPS: rho = −.5, $p = .019$, d = 1.1; age: rho = .51, $p = .016$, d = 1.1, frequency of isolated JA: rho = .1, $p = .6$, n = 24). In both analyses, TPJ activity correlated positively with the development of mentalization, while IPS correlated negatively.

Finally, to rule out influence of muscular artifact in the EEG, we carried out a control analysis. We extended the windows of analysis in order to detect (1) when the possible muscular activity became significant, and (2) when muscular activity was biased towards the JA condition (we expected more muscular activity because of the head movement). Following prior work (Muthukumaraswamy, 2013), we explored occipital and temporal electrodes from the edge of the montage and used beta (20–40 Hz) and gamma (60–100 Hz) activity as a proxy for EMG activity (band-pass Zero-phase shift FIR filter and Hilbert transform). The broad band activity became significant after 800 msec post target stimulus and was greater for JA trials around 2 sec post stimulus (Supplementary Figure 2).

3.2. ASD versus TD group

3.2.1. Behavioral results

The ASD group presented an average of 38.2% ± 28.3 (SD) JA trials. There was no significant difference in the number of JA trials between the two groups ($p = .13$, see Table 1). As in the control group, no significant gender difference nor differences between children who correctly solved and who did not correctly solve the False belief test were found for the amount of JA trials ($p > .4$). For children with ASD, about 27% of the JA trials occurred in isolation (see Supplementary Figure 1). For both groups, more JA trials occurred consecutively than in isolation (ASD: $p = .0241$, rho = .5, d = 1.1; Supplementary Figure 1). As in TD children, the mean of consecutive JA trials was above chance ($p = .018$, permutation test, d = 1) for children with ASD (Wilcoxon test, $p = .13$, n = 44). In accordance with other works (Hamilton, 2009; Happé, 1995), we found a statistically significant difference between the number of ASD and TD children that correctly solved the False Belief Task ($p = .0038$, rho = .44, d = .97). The children with ASD that correctly solved the False Belief Task were older than the TD children ($p = .0294$, rho = .43, d = .97, Table 1). Although there is no correlation between frequencies of isolated JA and metalizing skills (Spearman's rho = .1, $p = .6$), the frequencies of isolated JA correlated with ADOS-2 score for ASD group (Spearman's rho = .59, d = 1.4, $p = .007$).

3.2.2. ERP results

When comparing ERP results of TD and ASD group, we found a significant difference in the Nc component in the left frontocentral electrodes. The opposite modulation of the Nc component was found for ASD children when compared to TD children. Specifically, the amplitude of the Nc increased during the nJA condition in ASD children (left frontal ROI, ASD modulation: $p = .008$, n = 20, rho = .58, d = 1.4; ASD-TD difference: $p = 9.2e\text{-}4$, n = 44, rho = .48, d = 1.1, Fig. 4A). The scalp and source analyses of the between-group comparison indicated that the left frontal and the right superior parietal cortices were the main contributors to this difference (see Fig. 4C). JA modulation in left frontal electrodes correlated with the symptomatology of the ASD group (ADOS-2 score, rho = −.45, $p = .044$, n = 20, d = 1), but not with the achieving mentalization ($p > .2$). No significant correlation was found between neither mentalization nor ADOS-2 with NC amplitude in the right frontal electrode where TD children showed significant modulation ($p > .3$).

3.2.3. Time-frequency results

We compared the modulations in oscillatory activity in the first JA trials of each sequence using a new model to specifically isolate the activity of the first trials (see Fig. 5A and Equation (2)). The comparison between this model and the model used in Fig. 3A yielded similar results (see Supplementary Figure 4). While the TD group exhibited a significant increase in beta band power (15–25 Hz), around .4 sec and .55 in the right parietal group of channels (Wilcoxon test, cluster-based permutation test, $p < .01$, n = 24), there was no change in this beta band in the ASD group (see Fig. 5C–D). This yielded a statically significant difference between groups (Fig. 5F–G). The source analysis suggested that the right temporoparietal region and the middle/superior frontal gyrus are the main brain regions contributing to the beta power differences between the two groups (15–25 Hz, .4–.55 sec, Fig. 5H). TD and ASD children that correctly solved the False Belief Task also demonstrated significantly greater involvement of beta activity in the right TPJ ROI ($p = .008$, n = 20, rho = .58, d = 1.4, see Supplementary Figure 5). This finding remains significant after controlling for age and IPS activity (robust regression: $T_{17} = 5$, $p = .0001$; Spearman partial correlation rho = .62, $p = .005$, n = 20, d = 1.5). Neither mentalization nor TPJ activity correlated with the ADOS-2 score [abs (rho) < .08, $p > .7$; see Supplementary Table 3]. However, this result needs to be considered with caution given the small number of children with ASD that correctly solved the False Belief Task (n = 3). In view of this limitation, we tested the robustness of the relationship between mentalization ability and TPJ activity using a regression pooling the two groups of children. Again, age and

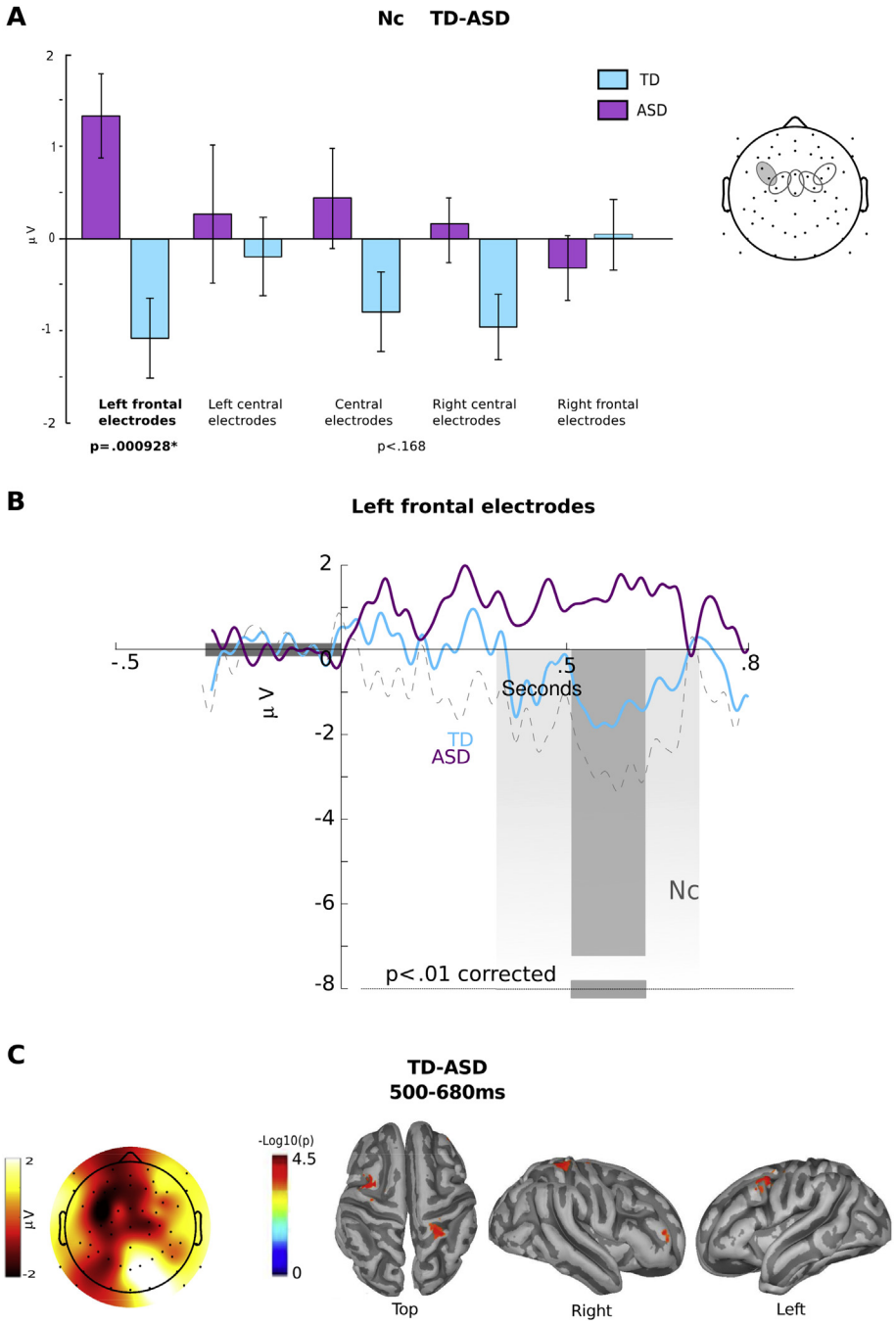

Fig. 4 – Differences in electrical brain activity of TD and ASD children. (A) Bar plot shows the differences in the five frontal ROIs that contrast JA condition – nJA condition in both groups. (B) Differences in the Nc component (JA-nJA) in left central electrodes of the TD and ASD groups. (C) On the left, topographic distribution of the significant cluster of differences between the two groups in Nc modulations (500 msec –700 msec), as seen in (B). On the right, sources of the significant cluster of differences between groups as shown in (B). Error bars represent 95% confidence interval. See Supplementary Fig. 3.

TPJ activity correlated with explicit mentalization ability (see Table 2). Finally, the ERP and oscillatory activity of the ASD group were assessed together using a partial Spearman correlation. We found that both activities maintained the relationship with behavioral scores (ERP–Ados-2 score: rho = –.48, p = .043, d = 1; beta oscillation–mentalization: rho = .66, p = .002, n = 20, d = 1.7; see Supplementary Table 3).

4. Discussion

This study assessed the neurobiological mechanisms that precede and trigger initiating JA behavior in children with and without ASD by recording their electroencephalographic activity while solving an JA task. We focused on the

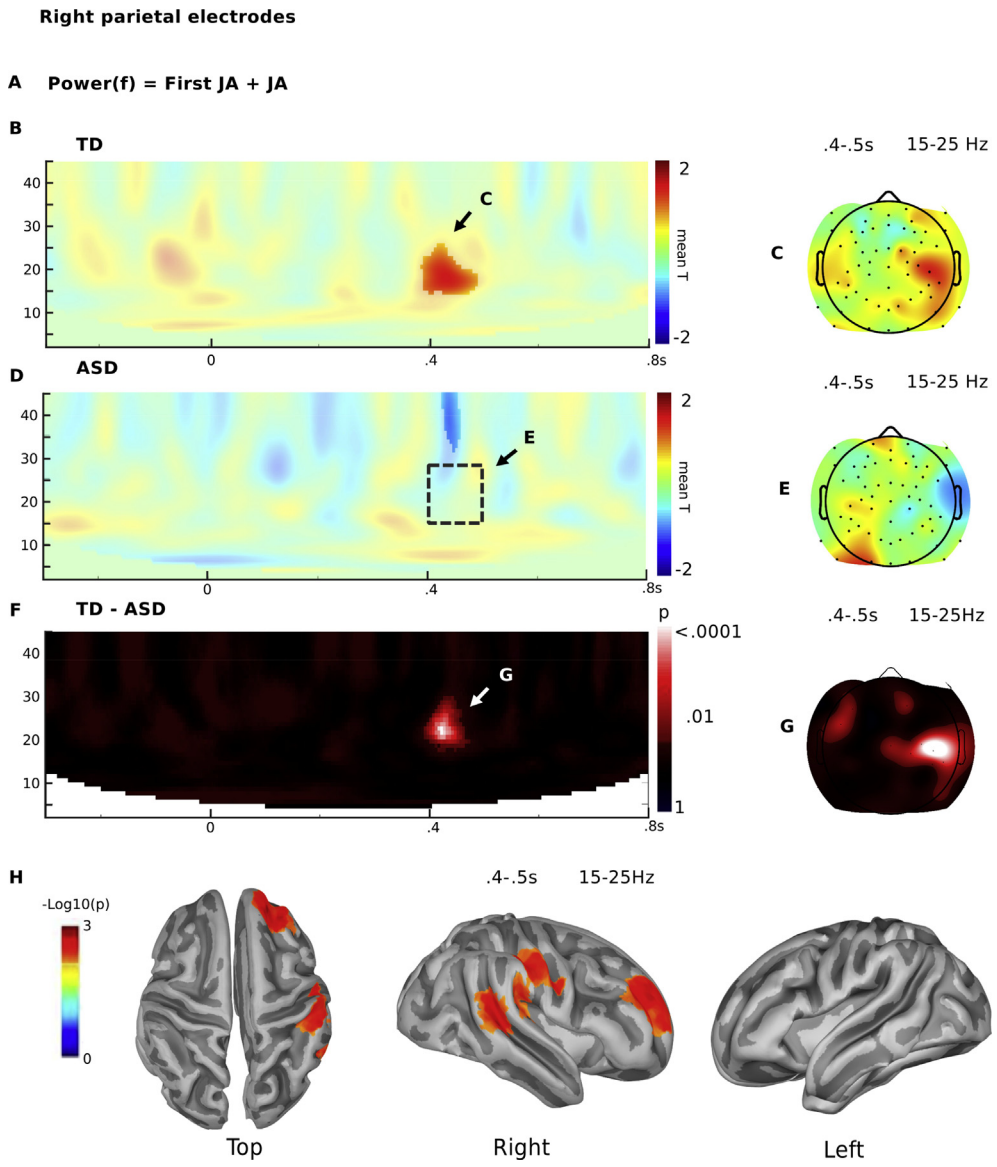

Fig. 5 – Oscillatory brain activity of the TD and ASD groups during the first JA trial of a sequence. (A) Model used to estimate the modulation of the first JA trial. (B) Time–frequency chart of the first JA regressor of TD children in right parietal electrodes. (C) Topographic distribution of the significant cluster of beta activity (15–25 Hz, .4–.55 sec) as shown in (B). (D) Time–frequency chart of the first JA regressor of the ASD group in the right parietal electrodes. The dotted-line rectangle is the region of the significant cluster in TD children. (E) Topographic distribution of the significant cluster of beta activity (15–25 Hz, .4–.55 sec) as shown in (D) using the same cluster found in TD children, as shown in (B). (F) Time–frequency chart of the *p*-values of the differences between the TD group and the ASD group in oscillatory activity of the first JA regressor. (G) Scalp distribution of *p*-values of the difference between groups as shown in (E). (H) Estimated sources of the significant cluster of beta activity differences (15–25 Hz, .4–.55 sec) between the two groups, as shown in (F and G). See also Supplementary Fig. 4.

relationship between JA and mentalization from the electrophysiological and behavioral perspectives.

The EEG results of TD children showed that initiating JA is differentially related to activity in attentional control and mentalization networks. The ERP results evidenced the activation of the attention neural network prior to sharing attention with another person. Specifically, there was a greater negativity in the right fronto-central electrodes during the JA condition. This difference corresponds to the Nc component, which has been associated with attentional control in infants and children (Courchesne, Ganz, & Norcia, 1981; Striano et al., 2006). Source analysis of the difference between JA and nJA demonstrated right-side lateralization, which is in accordance with the notion of right dominance of the attentional reorientation network (Corbetta et al., 2008). An earlier work using attentional tasks with infants showed that the sources of the Nc involves frontal areas, including the IFG (Richards et al., 2010), which is consistent with our findings. Our source analysis also

Table 2 – Robust logistic regression of explicit mentalization in both groups of children.

Robust logistic regression of mentalization	slope	s.e.	t-value	*p*-value
Intercept	−1.26	.44	−2.8	.067
Age	.47	.11	4.1	.0002
TPJ beta activity	.53	.17	3.0	.0047
IPS beta activity	−.23	.13	−1.75	.08
Diagnosis (ASD)	−.5	.12	−4.1	.0002
TPJ * Diagnosis	.02	.18	.14	.88
Freq. of isolated JA	−.2	.25	−.96	.34

demonstrated activity in the IPS, an area that has consistently been related to attentional processes in adults (Corbetta et al., 2008). Since our analysis window was the time preceding the JA behavior, this activity could be related to stimulus novelty or salience, which could trigger attentional reorientation (see Fig. 6 for a schematic representation). Alternatively, the activity could be preparatory to sharing attention with another person. The latter interpretation is supported by evidence in adults that has shown that right IFG and lateral parietal activity is related to initiating JA rather than to attentional reorientation per se (Caruana, Brock, et al., 2015; Redcay, Kleiner, & Saxe, 2012). Other confounding factors can play a role, such as preparatory activity to perceive another person or motor activity preparing for head motion.

Studies with adults have consistently shown that JA activates brain regions related to mentalization, such as the medial prefrontal cortex and the TPJ (Caruana, Brock, et al., 2015; Redcay et al., 2010; Schilbach et al., 2010; Williams, Waiter, Perra, Perrett, & Whiten, 2005). Because there is no evidence that establishes whether this activity is necessary for JA behavior or if it is only involved during JA in children/adults with mentalization skills, we correlated the Nc modulation between JA and nJA with the children's explicit mentalization skills (i.e., performance in the False Belief Task). The correlation was not significant for ERP modulation. This could indicate that this activity is more related to attentional processing than to mentalization processing (Chawarska et al., 2016; Jones & Klin, 2013). Nevertheless, in order to make a more precise interpretation, it would be necessary to have a measurement of the specific ERP activity during the mentalization task.

Interestingly, our ASD group demonstrated a reverse pattern of Nc modulation when compared to TD children. This modulation seemed to be driven by the nJA condition (see Supplementary Figure 3). The source of the modulation was identified as dorsoparietal and superior frontal regions (near the frontal eye fields). In adults, these areas are more active during the reorientation of attention in the nJA condition (Schilbach et al., 2010). We found that the frontal modulation in the Nc component correlates with autistic symptomatology. One possible interpretation of this modulation might be related to an alteration in the attention to social stimuli in the ASD group. Considering that the source of Nc modulation is located in the dorsal attentional network (responsible of maintaining the focus of interest), the correlation between the severity of the autistic symptoms and the Nc modulation could indicate differences in the focus of interest, i.e., focusing on an object rather than on another person. Considering that this modulation is given mainly for the nJA conditions (see above, Fig. 6 and Supplementary Figure 3), this finding could indicate that children with more severe symptomatology keep their interest on objects that they do not have the will to share with the experimenter. This interpretation is in accordance with evidence that shows that children with ASD pay less attention to social stimuli than TD children (Chawarska et al., 2016; Jones & Klin, 2013).

The analysis of oscillatory activity revealed another feature related to social cognitive networks, namely increased beta power in TD children engaged in JA as compared to ASD children. One of the sources of the beta power was the right TPJ, which is suggested to be part of the mentalization network (Saxe, Whitfield-Gabrieli, Scholz, & Pelphrey, 2009). This area shows consistent activation during JA tasks in adults (Caruana, de Lissa, & McArthur, 2015; Redcay et al., 2010; Schilbach et al., 2010; Williams et al., 2005), as well as during development from childhood to adolescence (Oberwelland et al., 2016). This network is consistently more active in JA as compared to nJA behaviors (Williams et al., 2005). A recent study of structural connectivity in children found that the development of mentalization skills correlates with changes in the structure of white matter in the precuneus, temporoparietal and frontal regions, and an increase in structural connectivity between the temporoparietal and frontal regions (Wiesmann et al., 2017). Accordingly, in our study beta modulation was only present in children that correctly solved the False Belief Task. Evidence indicates that before the age of 2, children can get some inference related to others' intentions and perspective in an automatic way, without awareness or

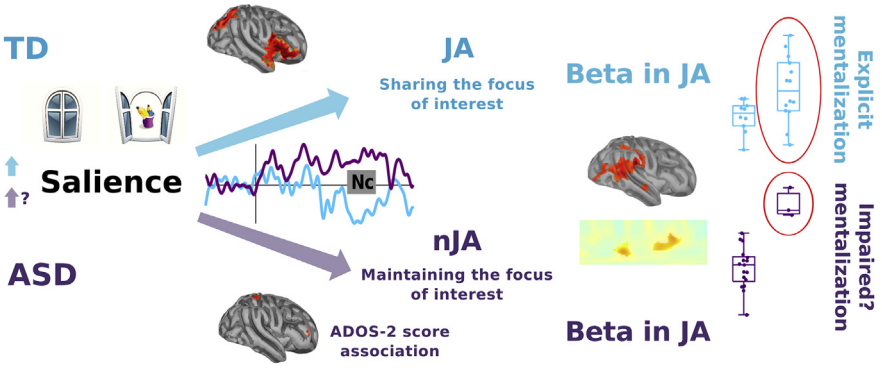

Fig. 6 – Schematic representation of the results interpretations.

control, a process called implicit mentalization (Moll & Kadipasaoglu, 2013; Van Overwalle & Vandekerckhove, 2013). In spite of this fact, changes in connectivity between temporal and frontal regions seem to be related solely to explicit mentalization (Wiesmann et al., 2017). Our findings support this line of evidence, showing that functional modulation is present once children have developed explicit mentalization skills. Studies with adults have not demonstrated modulation in the TPJ during implicit mentalization tasks (Schneider, Slaughter, Becker, & Dux, 2014). Children without explicit mentalization skills did not display beta modulation during JA trials, although they presented a similar number of JA trials. Consequently, in children that have already developed explicit mentalization skills, self-initiated JA would involve additional social processing, while in younger children without explicit mentalization abilities, JA may arise through simpler lower level processes (Corkum & Moore, 1998; Gredebäck, Fikke, & Melinder, 2010; Martin & Santos, 2016; Woodward, 2003). In other words, while temporoparietal activity may not be crucial for engaging in JA, it may be recruited once children can perceive the perspectives and intentions or preferences of others (Billeke, 2016; Suzuki, Adachi, Dunne, Bossaerts, & O'Doherty, 2015). Beta modulation in the temporoparietal region may represent the functional maturation of the mentalization network associated with explicit mentalization skills.

Beta oscillatory activity may be related to top-down control processing (Bosman et al., 2012; Buschman, Denovellis, Diogo, Bullock, & Miller, 2012; Buschman & Miller, 2007; Richter, Thompson, Bosman, & Fries, 2017). Studies with monkeys have revealed that gamma coherence is greater during bottom-up visual searches, while beta coherence between the lateral intraparietal area and the frontal cortex is greater during top-down visual searches (Buschman & Miller, 2007). Likewise, beta oscillations in the parietal regions influence visual areas during directed attention (Richter et al., 2017). Considering these findings, the increase in beta power that TD children demonstrated could indicate a top-down process. Studies in human adults have also demonstrated temporoparietal modulation in alpha/beta oscillations during social interactions. Thus, JA and sharing attention on an object with another person throughout declarative pointing modulates alpha/beta activity in temporoparietal electrodes (Brunetti et al., 2014; Lachat, Hugueville, Lemaréchal, Conty, & George, 2012). Moreover, alpha synchronization between central and temporal regions correlates with Reading the Mind in the Eyes Test in typically developing adolescent but not in adolescents with ASD (Jaime et al., 2016). Alpha/beta modulation in the right TPJ in adults correlates with anticipation of the other person's behavior during social interaction (Billeke et al., 2013, 2015; Melloni et al., 2016). This seems to be specific to anticipating another person's behavior, rather than to compute responses (Billeke et al., 2015, 2014; Billeke, Soto-Icaza, Aspé-Sánchez, Villarroel, & Rodríguez-Sickert, 2017). Interestingly, beta activity in TD children is also located in the IPS, although this activity correlates negatively with explicit mentalization skills. A possible explanation of this is that as children develop explicit mentalization abilities, less recruitment of the attentional network is required to share attention with other people. This is in accordance with neuroimaging evidence that shows an increase over time in the specificity of TPJ activity related to social processing (Oberwelland et al., 2016; Redcay et al., 2010; Saxe et al., 2009). Thus, beta oscillation could be related to top-down social processing rather than to bottom-up reorienting attentional processing. With top-down processing we refer to a controlled processing, probably accessible to awareness and introspection, that are features of explicitly mentalization. Interestingly, we did not find alpha modulations in our groups. Studies in infants reveal that alpha desynchronization (i.e., a decrease in the event-related induced power) is related to attentional processing (Xie, Mallin, & Richards, 2017). Although some studies in adults show alpha and beta modulation in TPJ during social interaction (Billeke et al., 2013), it seems that beta modulation is more specific than alpha modulation for social related to attentional processing (Billeke et al., 2015; Melloni et al., 2016; Park et al., 2018). More studies are necessary to demonstrate a dissociation between alpha and beta in social processing.

TPJ beta activity in both groups correlated explicitly with achieving mentalization. As a group, the children with ASD did not demonstrate beta modulation. However, the ROI analysis of the right TPJ showed that the ASD children that correctly solved the False Belief Task displayed a beta modulation comparable to that of TD children with explicit mentalization skills (see Fig. 6 and Supplementary Figure 5). Our partial correlation analysis of the ASD group suggests that the ERP modulation correlates with autistic symptoms, independent of the relationship between beta modulations and mentalization. Although only a small number of children with ASD in our sample correctly solved the False Belief Task, this suggests a dissociation between attentional processing and explicit mentalization in persons with ASD. Indeed, high-functioning ASD adults show explicit mentalization skills, although they do not demonstrate implicit mentalization skills (Senju, Southgate, White, & Frith, 2009). Thus, beta temporoparietal oscillations could be used as a marker to study the relationship between implicit and explicit mentalization skills in ASD and TD children.

There is evidence that the imbalance between excitatory and inhibitory neural activity is related to ASD pathogenesis (Gao & Penzes, 2015). Specifically, the inhibitory interneuron system is closely related to rhythmogenesis, which is necessary to generate synchronic cortical network activity during cognitive processing (Wang, 2010). Oscillatory EEG activity may reflect neural synchronization/desynchronization of cerebral networks (Billeke, Ossandon, et al., 2017; Wang, 2010). Studies have suggested the existence of GABAergic interneuron impairment in individuals with ASD (Gao & Penzes, 2015; Yizhar et al., 2011). The oscillatory alterations observed in ASD children during our cognitive task can be interpreted as weak coordination of neural populations (Larrain-Valenzuela et al., 2017). Beta oscillations play a crucial role in long-distance neuronal coordination (Donner & Siegel, 2011; Spitzer & Haegens, 2017). The TPJ belongs to brain networks that display a non-canonical network structure, unlike sensory-motor networks (Buckner & Krienen, 2013). This non-canonical brain architecture means that regions possess connections that do not conform a sequence of feedforward and feedback relationships, but rather tend to be reciprocally connected with common targets and inputs distributed

throughout the brain (Buckner & Krienen, 2013). This network architecture seems to be designed to participate in integrative computations related to internal and social processes (Margulies et al., 2016). Consequently, TPJ beta activity can be understood as a marker of an efficient computational process that leads to perspective-taking and mentalization processing during social interactions.

Finally, the results with TD children showed that beta power in the temporoparietal area was mainly modulated by the first JA trial in a sequence of consecutive JA trials. The decrease in beta power in the subsequent JA trials is similar to repetition suppression that has been described in fMRI studies (Barron, Garvert, & Behrens, 2016). Several neuroimaging studies have depicted this phenomenon as relying on the suppression of the neural response to a stimulus that is repeatedly presented. Considering that this suppression is understood as an adaptation, the decrease in the beta band suggests a similar adaptation during social interaction. Repetition suppression of oscillatory activity, as measured by EEG, has also been reported in other tasks (Hohlefeld, Nikulin, & Curio, 2011; Kongthong, Minami, & Nakauchi, 2014).

There are limitations to this study that should be considered when interpreting the findings. A broader battery of mentalization and cognitive ability tests would improve differentiating explicit mentalization abilities among participants. We did not have a precise measurement of the moment of the initiation of the JA behaviors. Thus, it is possible that in some trials the electrophysiological activity could coincide and not precede the very beginning of the JA behavior. This may preclude the accurate separation between social related and movement preparatory related activity. We did not find statistical differences between groups in our behavioral measure, which was number of JA behaviors. This fact could be due to a low statistical power due to the small sample size, but it could also be due to a low sensitivity of our behavioral measure. In this context, a more detailed description of the social interaction evoked by JA behavior, for example the time that the interaction lasted or the number of interactions that happened during one JA trial, could probably differentiate better between TD and children with ASD, as well as between children with and without mentalization. Considering the small number of children with ASD that correctly solved the False Belief Task, future research should include larger samples of children with ASD and TD children. Additionally, some subjects had less artefact–free trials than others. That generated differences in the noise-to-signal ratio. These results would have to be replicate with a greater sample size to tackle these limitations. Another fact to take into account for the interpretation of both ERP and TF results (i.e., Nc modulation and beta modulation rather than alpha modulation) is that children brain networks are different from the adult functional networks (Eggebrecht et al., 2017; Homae et al., 2010; Mills, Lalonde, Clasen, Giedd, & Blakemore, 2012). Several processes such as myelination, synaptic elimination, increase in skull thickness and fontanel closing, can influence both the amplitude and latency of the cerebral oscillation across different ages (Goldman, Shapiro, & Nelson, 2004; Luyster et al., 2014; Parker & Nelson, 2005).

In summary, our study shows a possible neuronal mechanism underlying the development of JA to explicit mentalization. Modulation in attentional processing registered by the Nc ERP is related to JA behavior, and is altered in children with ASD, reflecting their autistic symptomatology. The development of explicit mentalization correlates with beta oscillatory activity in the temporoparietal region both in children with ASD and in children with typical development. This modulation seems to be dissociated from attentional processing and autistic symptomatology. Understanding the neural mechanisms involved in typical and atypical social development can enable clinicians and therapists to direct their efforts to early intervention and diagnosis, which can improve social functioning. Earlier and more effective interventions can help families in dealing with the needs of their children and to enhance children's strengths and abilities.

Competing interests

The authors report no biomedical financial interest or potential conflicts of interest.

Author contributions

P.S–I., P.B. designed the experiment; P.B. programmed the experiment; P.S–I., P.B., conducted the experiments; P.S–I., L.V. conducted subject evaluations; P.B., P.S–I. analyzed the data; P.S–I., P.B., F.A. interpreted and discussed the results; P.S–I., P.B., F.A. wrote the manuscript.

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Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.cortex.2018.12.018.

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