

UNDERSTANDING SOCIAL VERSUS NONSOCIAL INTENTION IN AUTISM
SPECTRUM DISORDER: EXPLORING THE NEURAL CORRELATES OF INTENTION
UNDERSTANDING BASED ON INTENTIONAL CONTENT

By

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ABSTRACT

UNDERSTANDING SOCIAL VERSUS NONSOCIAL INTENTION IN AUTISM SPECTRUM DISORDER: EXPLORING THE NEURAL CORRELATES OF INTENTION UNDERSTANDING BASED ON INTENTIONAL CONTENT

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Behavioral research suggests that children with Autism Spectrum Disorder (ASD) are able to understand the intentions of others when intention is communicated via an action on an object (nonsocial intention). However, the existing literature indicates that they are impaired in their ability to understand intention when it is cued using social-communicative cues (social intention). Across two separate studies, we expand upon the behavioral research conducted to date by examining the neural correlates associated with different types of intention understanding in typically developing adults, typically developing children, and children with ASD.

In Study 1, we validate a new paradigm in typically developing adults for assessing intention understanding using the late positive component (LPC), which is an event related brain potential that has been associated with the processing of intention understanding broadly defined. Study 1 results indicate that the paradigm successfully differentiates between intentional and unintentional actions for both social and nonsocial stimuli, and that the magnitude of this difference (LPC effect), for the social stimuli only, is related to social functioning in this nonclinical sample. We then utilize this new paradigm in Study 2 to compare social and nonsocial intention understanding in children with ASD and typically developing controls. Highly consistent with the extant behavioral literature, results of Study 2 indicate that children with ASD are less accurate than typically developing controls in discriminating between social intentional and social unintentional actions, but perform equally well compared to their typically

developing peers for nonsocial stimuli. In contrast to these behavioral results, no group differences were identified in LPC effect magnitude for either social or nonsocial intention understanding. However, paralleling results from Study 1, we identified a significant relationship between magnitude of the LPC social effect and level of impairment in social functioning such that as children (independent of diagnostic status) were less able to differentiate between intentional and unintentional actions at a neural level, degree of impairment in social functioning increased. As would be expected given prior research demonstrating intact nonsocial intention understanding in this population, no significant relationships were identified with the nonsocial LPC effect. Study 2 exploratory analyses indicated that social functioning alone was predictive of the magnitude of the LPC social effect. Taken together, results of these studies suggest that the LPC social intention understanding effect is not uniquely associated with ASD, but instead reflects individual differences in human social functioning, including the severe social impairments which characterize ASD.

This dissertation is dedicated to my fiancé, Adam Powers, whose proofreading skills, stimuli construction assistance, and endless support has made this all possible.

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INTRODUCTION

Deficits in higher level social cognitive skills such as joint attention and theory of mind are well-documented in children with Autism Spectrum Disorder (ASD) (Meltzoff, 2007). However, the literature is equivocal to whether children with ASD have the ability to engage in lower level social cognitive skills such as recognizing others as acting intentionally. Given that intention understanding is a foundation skill upon which more advanced social cognitive abilities develop (Tomasello, 1999; Tomasello, Carpenter, Call, Behne, & Moll, 2005), the degree to which intention understanding abilities are impaired or intact in this population has profound implications at both a practical and theoretical level (i.e., the selection of appropriate treatment targets and refinement of existing theories of social dysfunction in ASD). This study serves to fill this gap in the literature by using Event-Related Potentials (ERPs), a measurement of brain activity with millisecond temporal resolution, to investigate the extent to which children with ASD demonstrate an understanding of different types of intentional actions.

The stimuli used in this study reflect different types of intentional actions in an attempt to clarify the mixed results found in the existing ASD intention understanding literature. Even when considering only the most immature form of intention understanding (i.e., recognition of intentionality via the consistent pursuit of goals), research in ASD populations has still yielded inconsistent results. These equivocal findings may be partially explained by the varying methodology used to assess intention across studies. For example, when children with ASD are simply required to attend to actions on an object to infer another's intention, studies reliably find intact intention understanding abilities (Aldridge, Stone, Sweeney, & Bower, 2000; Carpenter, Pennington, & Rogers, 2001; Colombi et al., 2009). In contrast, when tested using paradigms that require children with ASD to attend to, and incorporate, social-communicative cues to draw

conclusions regarding intention, impairment in intention understanding is identified (D'Entremont & Yazbek, 2007).

This pattern of intact versus impaired intention understanding observed across behavioral studies of children with ASD is consistent with a cognitive neuroscience framework for intention understanding. This framework holds that there are two discrete 'types' of intention that can be separated based on their intentional content (Ciaramidaro et al., 2007). The first type of intention involves *private intentions*, which represent a private goal. Private goals are defined as those that require only a single person in order to satisfy a particular aim (Ciaramidaro et al., 2007).

Behavioral studies with children with ASD that require inference of intention based on observation of one's action on an object would thus be considered to reflect understanding of private intention. The second type of intention involves the representation of a social goal, and is thus termed *social intention* (Ciaramidaro et al., 2007). Behavioral studies with children with ASD that require inference of intention based on observation of one's social communicative behavior (such as eye gaze or facial expressions) would thus be considered to reflect understanding of social intention. Indeed, in the only study to date directly comparing private and social intention understanding in the same sample of children with ASD and typically developing controls, results indicated that the clinical population performed as well as controls on a task tapping private intention, but demonstrated impairment on a task assessing social intention understanding (Berger & Ingersoll, 2014). For the sake of face validity, we will refer to 'private' intention as 'nonsocial' intention throughout this manuscript.

Functional magnetic resonance imaging (fMRI) studies investigating nonsocial versus social intention understanding in typically developing adults support the position that intention understanding is not a unitary construct, and indicate that nonsocial and social intention

understanding are subserved by discrete neural networks (Bara et al., 2006; Bara, Ciaramidaro, Walter, & Adenzato, 2011; Ciaramidaro, Becchio, Colle, Bara, & Walter, 2014; Walter et al., 2004). However, limited work has been done to explore the neural circuitry of intention understanding in individuals with autism. More specifically, there have been no investigations to date directly comparing social and nonsocial intention in the same sample, or even studies comparing social and nonsocial intention using the same paradigm. The existing literature is thus limited in its ability to compare directly the neural correlates associated with social and nonsocial intention in ASD. Looking across the literature instead, preliminary evidence for intact nonsocial intention understanding at a neurological level comes from part of a multi-experiment study investigating the processing of goal-directed action and intention understanding in ASD by Marsh and Hamilton (2011). Participants in this study viewed basic reaching actions that could be deemed either rational or irrational based on how the action was completed given different situational contexts. Results of this study indicated that the encoding of nonsocial intentions was completed in the left anterior intraparietal sulcus (IPS) for both adults with ASD and age- and IQ-matched typically developing controls. Similar conclusions can be drawn from a separate study investigating the neural correlates of physical and intentional causality, in which individuals with ASD and age and IQ matched typically developing individuals were shown a series of comic strips depicting scenarios of either physical causal attribution (e.g., a glass falling off a table) or nonsocial intentional attribution (e.g., hanging a painting on the wall) (Kana, Libero, Hu, Deshpande, & Colburn, 2014). When comparing the processing of comic strips depicting nonsocial intention versus those depicting physical causation, both groups activated the same area (the primary center of significantly increased activation was in bilateral posterior superior temporal sulcus (pSTS) / temporal parietal junction (TPJ)).

In contrast, studies examining the neurological processes supporting social intention in those with ASD compared to typically developing controls tend to find differences in patterns of neural activation between groups. For example, Ahmed and Vander Wyk (2013) presented participants with actions that were congruent or incongruent with a previously displayed emotion. The focus here was on comparing the neurological responses involved in social intention of a group of children with ASD, a group of unaffected siblings of children with ASD, and a well-matched group of control children. Findings indicated that typically developing children used the actor's emotional expression to develop expectations for her subsequent action and had to recruit additional processing resources when her action was incongruent with the inferred prior intention (as reflected by greater activation in the pSTS for the incongruent condition compared to the congruent condition). In contrast, both unaffected siblings and children with ASD exhibited abnormal processing of social intention understanding such that they showed similar levels of activation in this area across congruent and incongruent conditions. These findings replicate previous work employing this paradigm with adults with ASD (Pelphrey, Shultz, Hudac, & Vander Wyk, 2011), and are consistent with work showing that when intentionality is cued using eye gaze, individuals with ASD fail to differentially process intentional versus unintentional stimuli at a cortical level (Pelphrey, Morris, & McCarthy, 2005). This is in contrast to typically developing controls, who display sensitivity in the pSTS to intention conveyed via gaze shifts.

Taken together, there is evidence for social intention understanding network dysfunction in ASD, with nonsocial intention understanding network functioning remaining intact. However, hemodynamic imaging methods lack the temporal resolution necessary to delineate the chronology of intention understanding processing. The time course of intention understanding

processing is particularly important to understand in ASD, as real world interactions require individuals to process and react appropriately to another's intentions at a fast pace. Event-related potentials (ERPs) are an alternative approach to measuring brain activity with millisecond temporal resolution. ERPs provide information regarding a subset of processes involved between stimulus encoding and response execution.

While a handful of studies have used ERPs to investigate the neural correlates of action understanding in typically developing adults using paradigms that may be thought to assess nonsocial intention understanding (Balconi & Caldiroli, 2011; Reid et al., 2009; Reid & Striano, 2008; Sitnikova, Kuperberg, & Holcomb, 2003), only one study to date has explicitly assessed both social and nonsocial intention understanding (Y. Wang, Huang, & Lin, 2012). Participants in this study were shown comic strips of intentional actions, with ERPs time-locked to presentation of the last scene (which depicted the fulfilled intention). Wang and colleagues (2012) identified a posterior late positive component (LPC) in the 300 milliseconds (ms) - 1000ms post stimulus time window that showed enhancement of the ERP effect for social compared to nonsocial intention. Based on these findings, the LPC appears to be a good candidate for studying intention understanding. Indeed, a number of other ERP studies examining violations of goal-directed action sequences (De Bruijn, Schubotz, & Ullsperger, 2007; Maffongelli et al., 2015; Sitnikova, Goff, & Kuperberg, 2009; Sitnikova et al., 2003), violations of goal-related object affordances (Vö & Wolfe, 2013), and intention understanding more generally (Van der Cruyssen, Van Duynslaeger, Cortoos, & Van Overwalle, 2009; Vistoli, Passerieux, Zein, et al., 2015; Y. Wang et al., 2012) have also identified a late positive component that broadly extends from approximately 300ms-1000ms depending on the task. The late positivity observed across these studies is generally posteriorally distributed and is broadly

considered to reflect the processing of salient events such as those that are novel or unexpected (Sassenhagen, Schlesewsky, & Bornkessel-schlesewsky, 2014).

Wang et al.'s (2012) study is useful in better understanding how typically developing individuals process social compared to nonsocial intention, but the paradigm is limited in its ability to assess intention understanding as a basic construct. This is because their study included only intentional actions. This limits the paradigm's utility in investigating the degree to which intention understanding is impaired versus intact in a clinical population, as demonstration of intact intention understanding requires the ability to differentiate a goal-directed action from an unintentional or accidental action (Carpenter, 2006). Given that we are interested in exploring the extent to which different types of intention understanding are impaired or intact in individuals with ASD, we require an ERP paradigm that compares both intentional and unintentional actions across different stimuli types (i.e., social and nonsocial intention understanding). Our research addresses this issue by creating a novel ERP paradigm that specifically evaluates the extent to which an individual can differentiate intentional from unintentional action, while also comparing social and nonsocial intention understanding processing. In Study 1 we validate this new paradigm using a sample of typically developing adults. In Study 2 we go on to use this task to evaluate and compare the neural correlates of intention understanding in children with ASD and typically developing controls.

STUDY 1

The primary aim of Study 1 was to validate a paradigm for assessing intention understanding in a neurotypical adult sample in preparation for examining intention understanding in children with ASD. In this study we recorded ERPs while typically developing adults viewed two sets of picture sequences with a terminal picture that was either congruent or incongruent with the intention conveyed. One set of pictures conveyed social intention and the other conveyed nonsocial intention. On the basis of previous studies that have found larger ERP effects for unexpected compared to expected actions (e.g., De Bruijn et al., 2007), we hypothesized that viewing the incongruent (unintentional) terminal picture would elicit more pronounced electrical activity than the congruent (intentional) terminal picture, indicating intention understanding. We expected this effect to be a posterior positivity occurring sometime between 300-1000ms post stimulus onset given previous work examining intention understanding using ERPs (Maffongelli et al., 2015; Sitnikova et al., 2003; Van der Cruyssen et al., 2009; Vistoli, Passerieux, Zein, et al., 2015; Y. Wang et al., 2012). Further, based on the findings of Wang and colleagues (2012), we expected that the social stimuli would evoke an enhanced late positivity compared to the nonsocial stimuli, indicating differential processing for these two type of intention. We did not expect to find any differences in response accuracy or reaction time across the two stimuli types given the relative ease of the task and that participants in this study are typically developing adults.

To assess the potential utility of this paradigm for investigating intention understanding in ASD, we also examined the relationship between sub-clinical autistic traits in our participants and their psychophysiological response to each stimuli type. Given prior research indicating that individuals with ASD have intact nonsocial intention understanding but impaired social intention

understanding, we predicted that there would be no relationship between sub-clinical autistic traits and the magnitude of the difference between LPC amplitudes for intentional and unintentional stimuli (i.e., the LPC effect) for nonsocial intention stimuli, but that a significant association between sub-clinical autistic traits and neurological responses to social intention stimuli would exist. More specifically, we expected that as ASD symptomatology increased, the magnitude of the LPC effect for social stimuli would be attenuated. This pattern would suggest that with increasing ASD symptomatology, individuals are less likely to differentiate between intentional and unintentional socially-cued actions.

Method

Participants

Thirty-five college-aged young adults (17 female; 20.0 ± 2.3 years) participated in this investigation. An initial sample of 40 participants was recruited, with one participant being excluded due to recent brain injury resulting from concussion, and four participants failing to perform the experimental task at greater than 50.0% correct. All participants provided written informed consent in accordance with the Institutional Review Board at Michigan State University and reported being free of any neurological disorder, psychological condition, previous history of head trauma, cardiovascular disease, physical or intellectual disabilities, and indicated normal or corrected-to-normal vision.

Intention Understanding Task

This task is designed to assess intention understanding by capitalizing on prior research indicating differing patterns of cortical activation when viewing unexpected goal-directed actions compared to expected goal-directed actions (Sitnikova et al., 2009; Sitnikova, Holcomb, Kiyonaga, & Kuperberg, 2008; Sitnikova et al., 2003). As this study focused on assessing and

comparing social and nonsocial intention understanding, two types of stimuli were included. Nonsocial stimuli were adapted from previous work using ERPs to investigate intention understanding in infants (Reid, Csibra, Belsky, & Johnson, 2007). Stimuli for the social intention task drew from infant intention understanding looking-time paradigms (Phillips, Wellman, & Spelke, 2002), which have also been used with children with ASD (Vivanti et al., 2011).

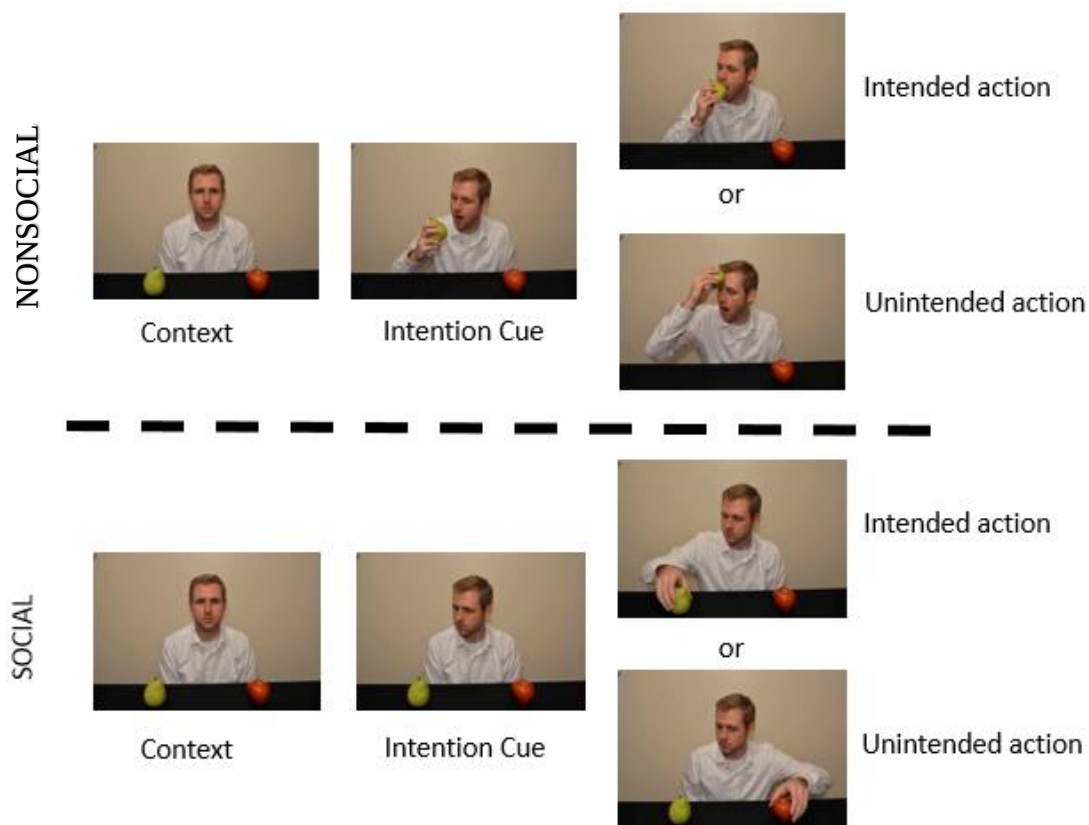
In this task, participants were shown a sequence of 3 images depicting a male actor interacting with one of two objects. Objects were food or beverages typically consumed with one hand (e.g., apple, donut, bottle of water, etc.), and did not contain any labels with words or letters. Objects rested on a black table equidistantly to the right and left of the actor. The first image was the same for both stimuli types, and provided the context of the action (actor seated at a table with two objects). The second image depicted a cue as to what the actor intended to do. For the social stimuli, the man's gaze and head was turned toward one of the objects; for the nonsocial stimuli the man lifted the object toward his mouth as if to eat or drink it. The terminal picture in each sequence depicted the actor either completing the intended action or performing an unintended action with the object. For the social stimuli, the actor grasped the object he was looking at (intended) or the object he was not looking at (unintended). For the nonsocial stimuli, the actor put the object to his mouth (intended) or elsewhere on his head (unintended). For both sets of stimuli, the intentionality of the terminal picture was dictated by the cue.

The social intention stimuli required the participants to infer the actor's intent based on visual referential looking. Visual referential looking has been conceptualized as engaging mechanisms involved in the attribution of intentions and goals to others, and implies that the person has an intention to act upon the object of reference (Calder et al., 2002). Whether or not

the terminal picture was considered intentional thus depended on the participant attributing intention to the actor after viewing the cue (visual referential looking).

Nonsocial intention stimuli required participants to infer the actor's intent based on the perceived rationality of the actions on the object. As an action is considered rational only with respect to a particular goal or intention (Buttelmann et al., 2014), whether or not the terminal picture was considered intentional depended on the participant assigning a particular goal to the actor after viewing the cue (object manipulation). See Figure 1 for examples of each stimuli type.

Figure 1. Example stimuli for each stimulus type.



Stimuli Construction and Validation. A total of 320 picture sequences were constructed using 160 different objects. These sequences were divided into two lists. Each list contained 40 nonsocial intended stimuli, 40 nonsocial unintended stimuli, 40 social intended stimuli, and 40 social unintended stimuli. The two lists contained the same picture sequence frames, but varied in whether the frame was paired with an expected or unexpected ending.

Normative ratings regarding intention in this task were obtained from a sample of 100 college-aged young adults with all participants providing written informed consent in accordance with the Institutional Review Board at Michigan State University. None of the participants from the norming study were included in the other aspects of this investigation. Participants were asked to rate each stimuli sequence with respect to the degree to which they considered the terminal picture as fulfilling the actor's intention, with 1 being "*Action was very unexpected*" and 7 being "*Action was very expected*". Participants were randomly assigned to view one of the two lists, such that for each of the social and nonsocial intention frames, half of the participants saw that frame with an intended ending and half saw it with an unintended ending. Thus, every participant saw every frame exactly one time, and across participants every social and nonsocial intention frame was presented an equal number of times with an intended or unintended ending.

Consistent with the theoretical conceptualization of the task, intended stimuli ($M = 6.39$, $SD = 2.00$) were rated as being more expected than unintended stimuli ($M = 1.65$, $SD = 3.00$), $F(1,98) = 16074.74$, $p < .001$), when examined using a repeated measures ANOVA with list as a between subjects factor and stimuli type and intentionality as within subjects factors. No differences in expectedness were observed between the ratings for nonsocial and social stimuli, nor were any interactions observed ($p > .05$). No items were excluded based on these analyses.

Autism Symptomatology

The Broad Autism Phenotype Questionnaire (BAPQ; Hurley, Losh, Parlier, Reznick, & Piven, 2007) was administered to assess the amount of autistic traits shown by an individual. The BAPQ is a 36-item self-report scale, with each item rated on a 6-point (1–6) scale, from “very rarely” to “very often.” Item responses are averaged together to form an overall score and three subscale scores (aloof, $\alpha = 0.94$; rigidity, $\alpha = 0.91$; pragmatic language, $\alpha = 0.85$; Hurley et al., 2007). Higher scores are indicative of greater expression of autistic traits. This measure has been shown to be a psychometrically sound method for assessing the broad autism phenotype in non-clinical adult populations in terms of internal consistency and criterion and incremental validity (Ingersoll, Hopwood, Wainer, & Donnellan, 2011).

Behavioral Performance Measures

Reaction time and response accuracy were evaluated in order to quantify participants’ behavioral performance. As participants were instructed to press a button only in response to intentional actions, reaction time data is only available to be assessed comparing across stimuli types. Additionally, all analyses with reaction time should be treated as exploratory given that there is a delay embedded in the task sequence between the terminal picture and when the participant is prompted to respond. This was done in order to avoid motor artifact contaminating the ERP effect of interest. In terms of response accuracy, correct responses to intentional trials were indicated by button press, correct responses to unintentional trials were quantified as lack of a button press response.

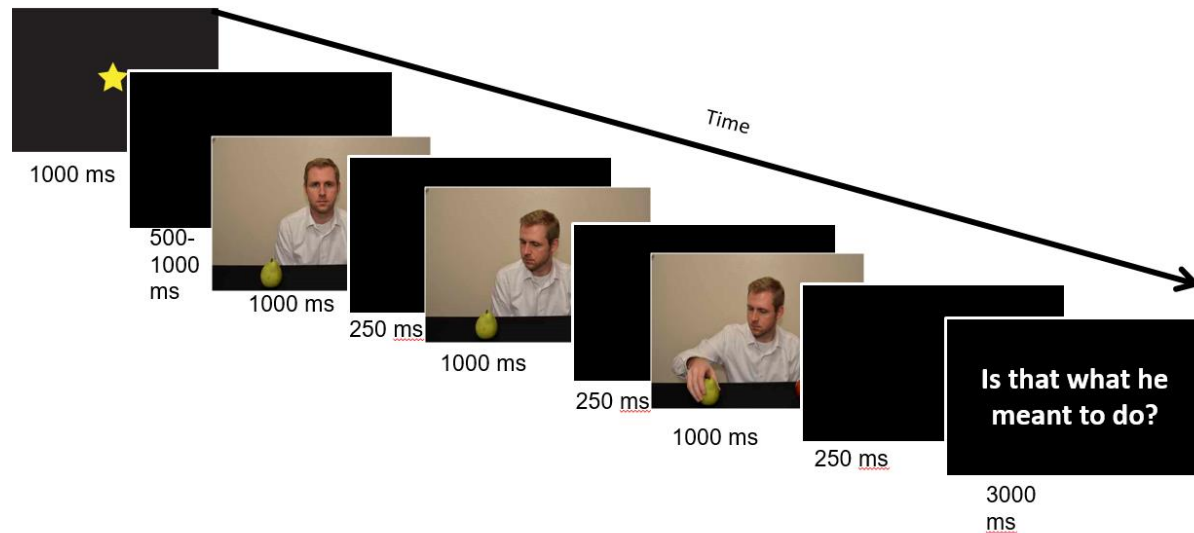
Procedure

Following provision of informed consent, participants completed the BAPQ. Participants were then fitted with a 64-channel Quik-cap (Compumedics Neuroscan, Inc.) and provided with

task instructions. Participants were given the opportunity to ask questions prior to completing 12 practice trials. The purpose of the practice trials were to familiarize participants with the pace and structure of the task only. Corrective feedback was not given, as we did not want to explicitly instruct participants in how to interpret the cues.

After the practice trials, participants were presented with 8 blocks of 20 trials to avoid participant fatigue, with the order of blocks randomized across participants. Each block contained 10 social and 10 nonsocial stimuli, presented in randomized order. Each trial was preceded by a yellow star for 1000 ms, which acted as a warning that a new picture sequence was about to begin, followed by a blank screen presented for a randomly jittered duration of between 500 and 1000 ms. Trials were then presented one picture at a time in the center of the screen. Each picture appeared for 1000 ms and was followed for 250 ms by a blank screen. After an interval of 1250 ms from terminal picture onset, a visual prompt appeared on the screen indicating the onset of the response interval. This visual prompt remained on the screen until participants made a response (up to 3 seconds). Participants were instructed not make any behavioral response until the visual prompt appeared on the screen. Participants made responses during the response interval with a response button held in one hand; response hand was counterbalanced across participants. Participants were instructed to press the button only if the actor completed the intended action. No button press was required for unintentional actions. Participants did not receive corrective feedback on their performance at any time, so as to not cue participants to the experimental manipulation (i.e., to use eye gaze to determine which object the actor would pick). The next sequences of images began 2000 ms after a button press was made or the 3000 ms response interval, whichever occurred first. See Figure 2 for task sequence.

Figure 2. Trial sequence for presentation of action sequences.



Psychophysiological Recording, Data Reduction and Analysis

EEG activity was recorded from 64 electrode sites arranged in an extended montage based on the International 10-10 system (Chatrian, Lettich, & Nelson, 1985) using a Neuroscan Quik-Cap (Compumedics, Inc., Charlotte, NC). Recordings were referenced to averaged mastoids (M1, M2), with AFz serving as the ground electrode and impedance less than 10 k Ω . Electrodes were placed above and below the left orbit and on the outer canthus of both eyes to monitor electrooculographic (EOG) activity with a bipolar recording. Continuous data were digitized at a sampling rate of 1000 Hz and amplified 500 times with a DC to 70 Hz filter using a Neuroscan SynAmps RT amplifier. EEG data was then imported (Delorme & Makeig, 2004) and was filtered using a 0.05 Hz high pass IIR filter to remove slow drifts (Mognon, Jovicich, & Bruzzone, 2011). ICA decomposition was performed using the extended Infomax algorithm to extract subgaussian components using the default settings in EEGLAB. ICA components that exhibited statistically significant correlation and overlap with eye blink activity and produced a

statistically significant reduction in the eye blink artifact present within the EEG were identified as artifactual using the `icablinkmetrics` function (Pontifex, Miskovic, & Laszlo, 2017) and then removed. Stimulus-locked epochs were created for trials from -100 to $1,000$ ms around the stimulus, baseline corrected using the prestimulus period, and filtered using a low-pass IIR filter at 30 Hz (24 dB/octave). Artifact in the EEG signal was identified if an amplitude excursion of ± 75 μ V occurred. Artifact-free trials were averaged without regard for behavioral accuracy.

Visual examination of the waveforms indicated a clear late positivity. As such, the LPC was evaluated as the mean amplitude within a 50 -ms interval surrounding the largest positive-going peak within a 300 – 700 ms latency window (Pontifex, Saliba, Raine, Picchietti, & Hillman, 2013). Amplitude was measured as the difference between the mean prestimulus baseline and mean peak-interval amplitude, while peak latency was defined as the time point corresponding to the maximum peak amplitude.

Statistical Analysis

All statistical analyses were conducted with $\alpha = .05$ using the Greenhouse-Geisser statistic with subsidiary univariate analyses of variance (ANOVAs) as needed to correct for violations of sphericity and Bonferroni-corrected t tests for post hoc comparisons. Table 1 provides means for behavioral and neuroelectric measures as a function of stimuli type and intentionality.

Table 1

Study 1: Means and Standard Deviations for Behavioral and Neuroelectric Measures as a Function of Stimuli Type and Intentionality in Typically Developing Adults

	Social		Nonsocial	
	Intentional	Unintentional	Intentional	Unintentional
Response accuracy, % (SD)	93.6 (9.72)	94.1 (13.3)	91.9 (13.2)	91.8 (14.6)
Reaction time, ms (SD)	481.7 (130.0)		585.0 (156.7)	
LPC Amplitude, μ V(SD)	8.6 (4.2)	12.7 (4.1)	10.4 (4.7)	16.3 (5.2)
LPC Latency, ms (SD)	379.2 (47.3)	413.1 (70.3)	416.4 (77.3)	459.2 (90.6)

Reaction time data for intended stimuli were analyzed using a paired *t*-test comparing social vs. nonsocial stimuli types. Analysis of response accuracy was conducted using a 2 (Stimuli type: social, nonsocial) $\times$ 2 (Intentionality: intended, unintended) multivariate repeated measures ANOVA. The LPC was assessed separately for amplitude and latency using a 2 (Stimuli type: social, nonsocial) $\times$ 2 (Intentionality: intended, unintended) $\times$ 7 (Site: Fz, FCz, Cz, CPz, Pz, POz, Oz) multivariate repeated measures ANOVA. Main effects and interactions with electrode site are not commented on unless they are of theoretical significance.

To address the extent to which intention understanding abilities (i.e., the ability to differentiate between intentional and unintentional actions) relate to autism symptomatology, difference waves were calculated ($LPC_{unintentional} - LPC_{intentional} = LPC \text{ effect}$) for each stimuli type. Pearson bivariate correlations were computed between LPC effects for each stimuli type and autism symptomatology as quantified by the BAPQ. Given that the repeated measures ANOVAs revealed significant interaction-effects with electrode site (suggesting that the stimuli type- and intentionality-effect differ between electrode sites), correlational analyses were run at each individual electrode site.

Results

Behavioral Performance Measures

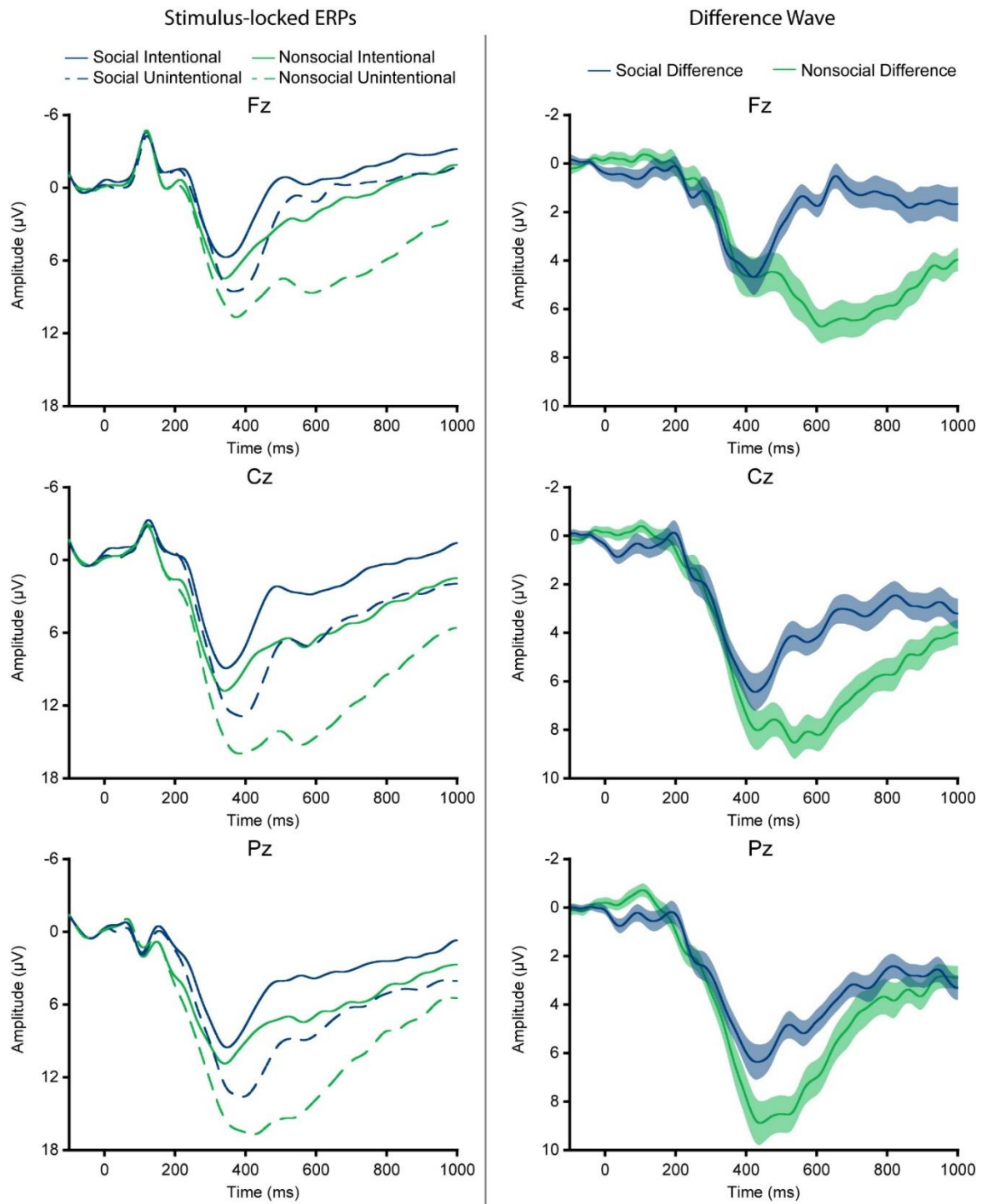
Reaction Time. Analysis of RT revealed that participants responded more quickly to social ($M = 481.7$, $SD = 130.0$) than nonsocial stimuli ($M = 585.0$, $SD = 156.7$), $t(34) = 8.9$, $p < 0.001$, $d = .72$.

Response Accuracy. Analysis revealed greater accuracy for social ($M = 93.9$, $SD = 11.2$), relative to nonsocial stimuli ($M = 91.9$, $SD = 13.6$), ($F(1,34) = 5.6$, $p < 0.05$, $\eta^2 = .14$); no differences in accuracy across intentionality conditions were identified ($F(1,34) = 0.1$, $p = .83$, $\eta^2 = .00$). No interaction between stimuli type and intentionality ($F(1,34) = 0.3$, $p = .57$, $\eta^2 = .01$) was observed.

Neuroelectric Measures

LPC Amplitude. Analysis revealed larger LPC amplitude for nonsocial ($M = 13.3$, $SD = 4.4$), relative to social ($M = 10.6$, $SD = 3.9$), stimuli ($F(1,34) = 29.6$, $p < .001$, $\eta^2 = .47$); and larger LPC amplitude for the unintentional ($M = 14.5$, $SD = 4.2$), compared to the intentional ($M = 9.4$, $SD = 4.1$), condition ($F(1,34) = 101.1$, $p < .001$, $\eta^2 = .75$). A main effect of site ($F(6,29) = 29.3$, $p < .001$, $\eta^2 = .86$) was identified, with Bonferroni corrected post-hoc tests revealing larger LPC amplitudes at posterior compared to anterior electrode sites. Refer to Figure 3 for grand average waveforms time-locked to terminal stimulus in each sequence for each stimulus and intention type.

Figure 3. The left panel provides grand average waveforms time-locked to terminal stimulus in each sequence for each stimulus and intention type from Study 1. The right panel provides grand average difference waves illustrating the difference between intended and unintended stimuli for each stimulus type from Study 1. The shaded area surrounding difference waves represents standard error.

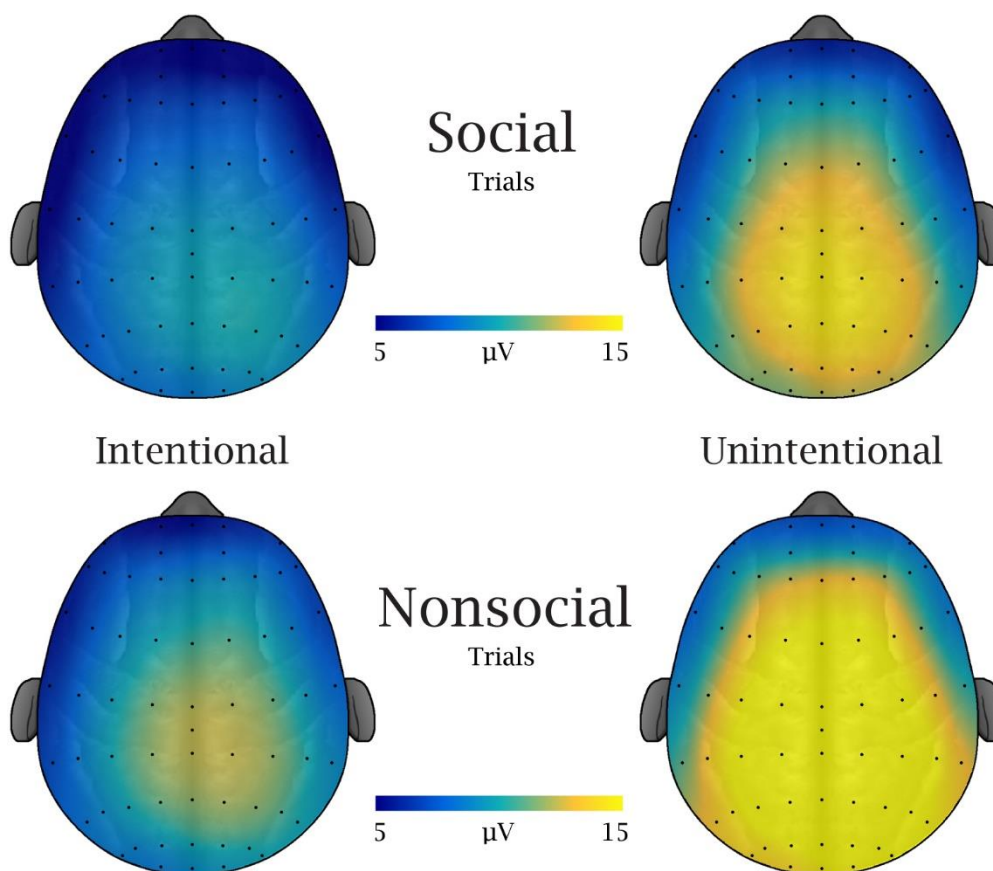


An interaction of stimuli type $\times$ intentionality ($F(1,34) = 5.9, p < .05, \eta^2 = .15$) was also observed. To examine this interaction in more detail, simple main effects of intentionality using local error terms were computed separately for each type of stimuli. For nonsocial stimuli, there was a significant main effect of the intentionality condition ($F(1,34) = 62.8, p < .001, \eta^2 = .65$). For social stimuli, there was also a significant main effect of the intentionality condition ($F(1,34) = 81.8, p < .001, \eta^2 = .71$). These analyses indicated that the LPC amplitude for the intentional condition (Nonsocial: $M = 10.4, SD = 4.7$; Social: $M = 8.6, SD = 4.2$) was smaller relative to the unintentional condition (Nonsocial: $M = 16.3, SD = 5.2$; Social: $M = 12.7, SD = 4.1$) for both nonsocial ($d = 1.20$) and social ($d = 0.99$) stimuli, but that the difference between conditions was greater for nonsocial stimuli.

An interaction of stimuli type $\times$ site ($F(6,29) = 3.3, p < .05, \eta^2 = .41$) was also observed. Post hoc comparisons were performed by examining simple main effects of stimulus type at each site. All seven sites demonstrated a statistically significant main effect (all p 's $< .01$), such that the LPC amplitude was greater for nonsocial compared to social stimuli across the scalp. Examination of effect sizes associated with each of the main effects revealed that the effect of stimuli type (i.e., nonsocial versus social) was largest over parietal electrode sites (CPz; nonsocial $M = 15.4, SD = 4.9$, social $M = 12.1, SD = 4.5, d = 0.73$, Pz: nonsocial $M = 14.9, SD = 5.2$, social $M = 12.1, SD = 4.9, d = 0.56$; POz: nonsocial $M = 13.7, SD = 5.0$, social $M = 11.3, SD = 4.7, d = 0.48$). Simple main effects were also used to follow up a significant intentionality $\times$ site ($F(6,204) = 9.7, p < .001, \eta^2 = .22$) interaction. Again, all seven sites demonstrated a statistically significant main effect (all p 's $< .001$), such that the LPC amplitude was greater for unintentional compared to intentional terminal pictures. Examination of effect sizes associated with each of the main effects revealed that the effect of intentionality (i.e., intentional versus

unintentional conditions) was also maximal over parietal electrode sites (CPz; nonsocial $M = 10.8$, $SD = 4.7$, social $M = 16.7$, $SD = 4.6$, $d = 1.25$, Pz: nonsocial $M = 10.5$, $SD = 4.6$, social $M = 16.5$, $SD = 5.5$, $d = 1.18$; POz: nonsocial $M = 9.6$, $SD = 4.3$, social $M = 15.2$, $SD = 5.6$). Refer to Figure 4 for topographic maps of LPC amplitude by stimuli type and intentionality.

Figure 4. Illustration of topographic differences in LPC amplitude as a function of stimuli and intention type in typically developing adults participating in Study 1. The LPC represents the 300-700ms epoch in this nonclinical population.



LPC Latency. Paralleling reaction time findings, this analysis revealed longer LPC latency for nonsocial ($M = 437.8$, $SD = 66.2$), relative to social stimuli ($M = 396.2$, $SD = 44.9$), ($F(1,34) = 15.7$, $p < .001$, $\eta^2 = .32$); and longer LPC latency for unintentional ($M = 436.1$, $SD = 70.4$), compared to intentional stimuli ($M = 397.8$, $SD = 43.7$), ($F(1,34) = 10.7$, $p < .01$, $\eta^2 = .24$). No interaction of stimuli type $\times$ intentionality ($F(1,34) = .2$, $p = .67$, $\eta^2 = .01$) was observed.

Relationship Between LPC Effect and Autism Symptomatology

Correlational analyses are based on peak amplitude and latency of difference waves in order to reflect each participant's ability to differentiate intentional from unintentional stimuli. See Table 2 for correlational analyses and Figure 3 for difference waves. No significant relationships were observed for any BAPQ subscale or overall scale and nonsocial LPC effect amplitude. In contrast, the amplitude of the LPC effect for social stimuli was significantly related to the Aloof BAPQ subscale at all electrode sites except Oz, such that as the magnitude of the LPC effect decreased (i.e., difference between intentionality conditions becomes smaller), BAPQ Aloof Personality subscale scores increased. Amplitude of the LPC effect for social stimuli was also significantly related to Overall BAPQ scores at FCz, Pz, and POz, although this appears driven by particularly strong relationships between the LPC effect and the Aloof subscale at these sites. No significant relationships were observed for any BAPQ subscale or overall scale and nonsocial or social LPC effect latencies.

Table 2

Study 1: Bivariate Correlations Between BAPQ Scores and LPC Effect by Stimuli Type and Electrode Site for Typically Developing Adults

	Overall		Aloof		Pragmatic Language		Rigidity	
	Amplitude	Latency	Amplitude	Latency	Amplitude	Latency	Amplitude	Latency
Fz								
Nonsocial Stimuli	-.14	.07	-.03	.11	-.26	-.06	-.08	.09
Social Stimuli	-.33	.04	-.48**	.12	-.19	-.12	-.03	.08
FCz								
Nonsocial Stimuli	-.27	.14	-.21	.17	-.33	-.03	-.10	.18
Social Stimuli	-.43**	.27	-.61**	.28	-.27	.01	-.06	.29
Cz								
Nonsocial Stimuli	-.20	.08	-.25	.12	-.24	-.03	-.11	.10
Social Stimuli	-.32	.19	-.46**	.14	-.22	.12	-.03	.19
CPz								
Nonsocial Stimuli	-.27	.07	-.33	.1	-.27	.05	-.16	.02
Social Stimuli	-.30	.05	-.43**	-.01	-.22	.05	-.02	.09
Pz								
Nonsocial Stimuli	-.24	-.08	-.20	.06	-.15	-.17	-.20	-.09
Social Stimuli	-.35*	-.05	-.40*	.01	-.27	-.02	-.13	-.07
POz								
Nonsocial Stimuli	-.26	-.04	-.25	-.01	-.25	-.19	-.22	.09
Social Stimuli	-.38	-.17	-.37*	-.21	-.25	-.16	-.25	.04
Oz								
Nonsocial Stimuli	-.29	-.05	-.23	-.03	-.28	-.16	-.12	.07
Social Stimuli	-.22	-.14	-.16	-.19	-.19	-.10	-.18	.00

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

Discussion

The primary aim of the present study was to validate a paradigm for assessing intention understanding that could be used to measure intention understanding in individuals with ASD. As expected, both types of stimuli effectively evoked differential neurological activity across intentional and unintentional conditions. Specifically, we found that unintentional stimuli evoked a larger LPC than intentional stimuli. This effect was observed at roughly 400ms and appeared to be largely posteriorally distributed. The ability of this paradigm to differentiate between intentional and unintentional action indicates that it would be appropriate for assessing the extent to which intention understanding is intact in a given population. In addition, we found that neural activation differed between the social and the nonsocial stimuli, with a greater LPC observed for nonsocial compared to social stimuli independent of condition. This pattern is the reverse of what was hypothesized based upon the findings of Wang and colleagues (2012), and will be discussed further in the general discussion section. We also identified a difference in the processing associated with each stimuli type such that, contrary to our hypotheses predicting no differences, typically developing participants responded faster and had greater accuracy when viewing social stimuli. This is in line with LPC latency results, which indicate shorter latencies for social stimuli compared to nonsocial stimuli. Taken together, this pattern suggests that the processing of social intention understanding may be simpler or more automatic than the processing of nonsocial intention understanding in this nonclinical population.

Findings of the current study are consistent with previous work suggesting a dissociation between social and nonsocial intention understanding and ASD symptomatology (Berger & Ingersoll, 2014). The identified relationship between subclinical autism symptomatology and the magnitude of the difference between intentional and unintentional stimuli in the social condition

indicates that as autism symptomatology increases, the ability to differentiate between intentional and unintentional action, when it is cued using social-communicative behaviors, decreases. It is unsurprising that this relationship was identified only for the Aloof subscale of the BAPQ, as this subscale is most highly related to social reciprocity and social understanding. The other two subscales reflect unusual patterns of language and behaviors, which would not necessarily be expected to relate to intention understanding. These findings strongly suggest that this paradigm is appropriate for assessing intention understanding in individuals with a clinical diagnosis of ASD moving forward.

STUDY 2

Study 2 extends upon the results of Study 1 by using the newly validated paradigm to assess, for the first time, the event-related brain potentials associated with social and nonsocial intention understanding in a cross-sectional sample of preadolescent children with ASD and typically developing children. Given prior behavioral research indicating that individuals with ASD have intact nonsocial intention understanding but impaired social intention understanding, together with the results of Study 1 identifying a relationship between subclinical ASD symptomatology and LPC amplitude for social stimuli only, we predicted no differences between typically developing controls and children with ASD in LPC amplitude for either intentional or unintentional trials for nonsocial stimuli. In contrast, we hypothesized that children with ASD would manifest a reduced LPC effect (i.e., a smaller difference between intentional and unintentional trials) for social stimuli. Taken together, this pattern of findings would suggest that, compared to their typically developing peers, children with ASD are less able to differentiate intentionality when cued using social-communicative behaviors, but that their nonsocial intention understanding abilities are intact. We expected similar findings regarding behavioral performance, such that children with ASD would be slower and less accurate than controls in responding to social stimuli but not to nonsocial stimuli. Given that Study 1 assessed autism symptomatology dimensionally rather than across diagnostic categories, we also included a dimensional measure of autism symptomatology in the current study. We expect to replicate Study 1 findings that regardless of diagnostic status, increased levels of autism symptomatology are related to a smaller LPC effect.

Method

Participants

Participants were 21 children with a community diagnosis of ASD (19 male) and 22 typically developing (TD) children (17 male) between the ages of 8-12 years. Participants were recruited by advertisements to local agencies serving children with autism, community events and listservs, and local schools. Participants were matched groupwise on age, gender, pubertal status, and estimated cognitive abilities (see Table 3 for participant characteristics). No significant differences were observed between groups on any of these demographic variables (p 's > 0.1). All participants had normal or corrected to normal vision. Participants were excluded if there was a known genetic disorder, seizures, significant sensory or motor impairment, serious head injury, or use of anticonvulsant medications. Additional exclusionary criteria for typically developing children included any history of developmental delay or a first degree relative with ASD. One typically developing child was excluded from analyses due to failure to provide sufficient artifact free data.

Table 3

Study 2: Participant Characteristics

	Typically Developing Children (n = 21)	Children with ASD (n = 21)	<i>t</i>
Number of males	16	19	1.54 ^a
Age in years (SD)	10.44 (1.30)	10.97 (1.09)	-1.43
WASI Full Scale IQ (SD)	105.43 (15.06)	96.81 (16.16)	1.79
Tanner Staging Pubertal Status (SD)	1.65 (.63)	1.81 (.75)	-.74

^a Number of males compared across groups using Pearson Chi-Square

ASD diagnoses were confirmed using the Autism Diagnostic Observation Schedule—Second Edition (ADOS-2; Lord et al., 2012) and DSM-5 criteria. All ASD children scored above the cut-off on the ADOS-2 for either Autism or Autism Spectrum Disorder. Typically developing children were screened for developmental concerns using the Social Communication Questionnaire (SCQ; Rutter, Bailey, & Lord, 2003), with all participants scoring below the cut-off of 15 ($M = 4.5$, $SD = 3.3$). Four typically developing children and four children with ASD reported diagnoses of Attention Deficit Hyperactivity Disorder (ADHD); these children were not required to discontinue stimulant medication use prior to study participation¹.

All participants provided written assent and their legal guardians provided written informed consent in accordance with the Michigan State University Institutional Review Board.

Intention Understanding Task

The task for Study 2 was identical to that established in Study 1.

Autism Symptomatology

The Social Responsiveness Scale-Second Edition (SRS-2) is a continuous, quantitative measure of social ability that generates an index of deficiency in social reciprocity, rather than providing an “all-or-nothing” characterization about the presence of symptoms or a given disorder (Constantino & Gruber, 2012). It was originally created (Constantino & Gruber, 2005) for use in studies examining the genetics of ASD, which required measurement of social deficits in families of children with ASD who may exhibit milder ASD phenotypes that are not clinically impairing. Thus, the SRS-2 is able to assess social impairments in groups of individuals both with and without ASD. The total norming sample for the SRS-2 was approximately 1,900 individuals nationally representative with regard to gender, ethnicity, education, and geographic

¹ Analyses were run both with and without ADHD participants. Results did not change meaningfully when these participants were excluded, thus only analyses with the full sample are reported here.

region; separated by rater type as well as age and gender of individual rated. Overall, the scale shows strong internal consistency (Constantino et al., 2003, 2007; Constantino & Todd, 2003), test-retest reliability (Constantino et al., 2003; Constantino & Todd, 2003), and inter-rater reliability (Pine, Luby, Abbacchi, & Constantino, 2006).

The SRS-2 is a parent report questionnaire with 65 questions on a 4-point Likert scale. Higher scores indicate more autistic traits; T-scores ≥ 65 (i.e., 1.5 SDs $\geq$ the population mean of 50) suggest clinically significant autistic traits. It yields a total score and five domain subscale scores (Social Awareness, Social Communication, Social Cognition, Social Motivation, and Restricted Interests and Repetitive Behaviors) expressed as T-scores, as well as two higher order indices that correspond to the two symptom domains of ASD (social communication and interaction, and restricted interests and repetitive behavior). Examples of items from each scale include: “Is aware of what others are thinking and feeling” (Social Awareness); “Takes things too literally and doesn’t get the real meaning of conversation” (Social Communication); “Doesn’t understand how events are related to one another (cause and effect) the way other children his or her age does” (Social Cognition); “Avoids people who want to be emotionally close to him or her” (Social Motivation); and “Has an unusually narrow range of interests” (Restricted Interests and Repetitive Behaviors). Given findings from Study 1 that LPC social effect magnitude was more related to specific symptoms of ASD than overall ASD symptomatology, our analyses for Study 2 will use the five domain subscale scores to better examine the specificity of the relationship between ASD symptoms and LPC effect magnitude.

Behavioral Performance Measures

Reaction time and response accuracy were evaluated in order to compare functional performance across groups and assess the degree to which measures of neural activity and

behavior align. As participants were instructed to press a button only in response to intentional actions, reaction time data is only available to be assessed comparing across stimuli types.

Additionally, as noted in Study 1, all analyses with reaction time should be treated as exploratory given that there is a delay embedded in the task sequence between the terminal picture and when the participant is prompted to respond. Again, this was done in order to avoid motor artifact contaminating the ERP effect of interest. In terms of response accuracy, correct responses to intentional trials were indicated by button press, correct responses to unintentional trials were quantified as lack of a button press response.

Procedure

Children with ASD completed activities over the course of two separate visits. The first visit (Day 1) included provision of informed consent and assent, administration of the ADOS-2, and the opportunity to try on the cap and become familiar with the laboratory. On the second visit (Day 2), following provision of informed consent and assent, participants were administered the WASI while parents completed questionnaires. From this point forward, procedures regarding capping, instructions, practice trials, and task sequencing were identical to those detailed in Study 1. Typically developing children completed activities in a single visit, which included provision of informed consent and assent plus those activities from ASD participant's Day 2. All participants were reinforced for on-task behavior with tokens that were exchanged at the end of study for small prizes. Participants were also compensated \$10/hour for their time. Participation in the study required approximate 4 hours of time for children with ASD, and 2.5 hours of time for typically developing controls.

Psychophysiological Recording, Data Reduction and Analysis

Procedures for EEG recording and processing were identical to those described in Study 1. Visual examination of the waveforms again indicated a clear late positivity. As such, paralleling Study 1, the LPC was evaluated as the mean amplitude within a 50-ms interval surrounding the largest positive-going peak within a 300–700 ms latency window (Pontifex et al., 2013). Amplitude was again measured as the difference between the mean prestimulus baseline and mean peak-interval amplitude, while peak latency was defined as the time point corresponding to the maximum peak amplitude.

Statistical Analysis

All statistical analyses were conducted with $\alpha = .05$ using the Greenhouse-Geisser statistic with subsidiary univariate analyses of variance (ANOVAs) as needed to correct for violations of sphericity and Bonferroni-corrected *t* tests for post hoc comparisons. Table 4 provides means for behavioral and neuroelectric measures as a function of stimuli type and intentionality. Refer to Table 5 for a summary of ANOVA results for key outcome variables.

Table 4

Study 2: Means and Standard Deviations for Behavioral and Neuroelectric Measures Collapsing Across Electrode Site as a Function of Stimuli Type and Intentionality in Typically Developing Children and Children with ASD

	Typically Developing Children				Children with ASD			
	Social		Nonsocial		Social		Nonsocial	
	Intentional	Unintentional	Intentional	Unintentional	Intentional	Unintentional	Intentional	Unintentional
Response accuracy, % (SD)	94.62 (8.34)	89.66 (24.70)	96.25 (6.37)	91.45 (25.52)	86.06 (15.28)	70.71 (28.23)	93.41 (9.00)	94.78 (10.41)
Reaction time, ms (SD)	762.49 (213.32)		626.46 (176.93)		832.26 (307.66)		701.05 (327.13)	
LPC Amplitude, μ V(SD)	12.59 (7.17)	15.63 (9.27)	13.33 (9.47)	21.81 (10.02)	11.19 (7.17)	14.04 (9.27)	12.62 (9.47)	19.43 (10.02)
LPC Latency, ms (SD)	373.71 (56.27)	393.29 (81.79)	413.10 (95.30)	430.78 (109.19)	392.54 (56.27)	390.63 (81.79)	406.20 (95.30)	430.65 (109.19)

Table 5

Study 2: ANOVA Results for key outcome variables

Effect	Response Accuracy F ($\eta^2\rho$)	Reaction Time F ($\eta^2\rho$)	LPC Amplitude F ($\eta^2\rho$)	LPC Latency F ($\eta^2\rho$)
Group	2.41 (.06)	.86 (.02)	.86 (.02)	.03 (.00)
Stimuli Type	26.12 (.40)***	31.23 (.44)***	23.35 (.37)***	13.32 (.25)**
Intentionality	4.67 (.10)*		68.01 (.63)***	3.65 (.08)
Site			12.40 (.68)***	3.26 (.08)*
Group x Stimuli Type	16.88 (.30)***	.01 (.00)	.00 (.00)	.42 (.01)
Group x Intentionality	.15 (.00)		.52 (.01)	.22 (.01)
Group x Site			1.23 (.17)	.99 (.02)
Stimuli Type x Intentionality	12.08 (.23)**		20.49 (.34)***	.64 (.02)
Stimuli Type x Site			.86 (.13)	4.96 (.11)**
Intentionality x Site			4.21 (.42)**	5.15 (.11)**
Group x Stimuli Type x Intentionality	11.63 (.23)**		.50 (.01)	.86 (.02)
Group x Stimuli Type x Site			1.52 (.21)	.97 (.02)
Group x Intentionality x Site			.43 (.07)	5.52 (.01)
Stimuli Type x Intentionality x Site			2.58 (.31)*	.59 (.02)
Group x Stimuli Type x Intentionality x Site			.90 (.13)	.43 (.01)

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

Statistical Analyses Behavioral Performance Measures. Reaction time was entered into a mixed model, repeated-measures ANOVAs with group (TD and ASD) as between-subject factor and stimuli type (social, nonsocial) as within-subject factors. Intentionality was not evaluated in terms of reaction time as participants only responded to intended trials. Response accuracy was assessed using a separate mixed model, repeated measures ANOVA with group

(TD and ASD) as our between-subject factor, and stimuli type (social, nonsocial) and intentionality (intentional, unintentional) as within-subject factors.

Statistical Analyses ERP Measures. The LPC was assessed separately for amplitude and latency using a mixed model, repeated measures ANOVA with group (TD, ASD) as between-subject factor and stimuli type (social, nonsocial), intentionality (intended, unintended) and electrode site (Fz, FCz, Cz, CPz, Pz, POz, Oz) as within subject factors. To address the research question regarding the extent to which children with ASD and typically developing children differ in their ability to differentiate intentional from unintentional actions by stimuli type, difference waves were calculated ($LPC_{unintentional} - LPC_{intentional}$) and assessed using a repeated measures ANOVA with group (TD, ASD) as between-subject factor and stimuli type (social, nonsocial) and electrode site (Fz, FCz, Cz, CPz, Pz, POz, Oz) as within subject factors. Based on Study 1's findings that autism symptomatology is dimensionally related to the LPC effect, bivariate correlations were computed between SRS subscale scores and LPC difference wave amplitudes. Given that the repeated measures ANOVAs revealed significant interaction-effects with electrode site (suggesting that the stimuli type- and intentionality-effect differ between electrode sites), correlational analyses were run for each individual electrode site. As Study 1 failed to identify a significant relationship between LPC effect latency and autism symptomatology, we chose not to evaluate that relationship here in order to minimize the number of correlational analyses being performed. Spearman's rho correlations were used to examine the relationship between LPC effect and autistic traits due to non-normal distribution of SRS data.

Results

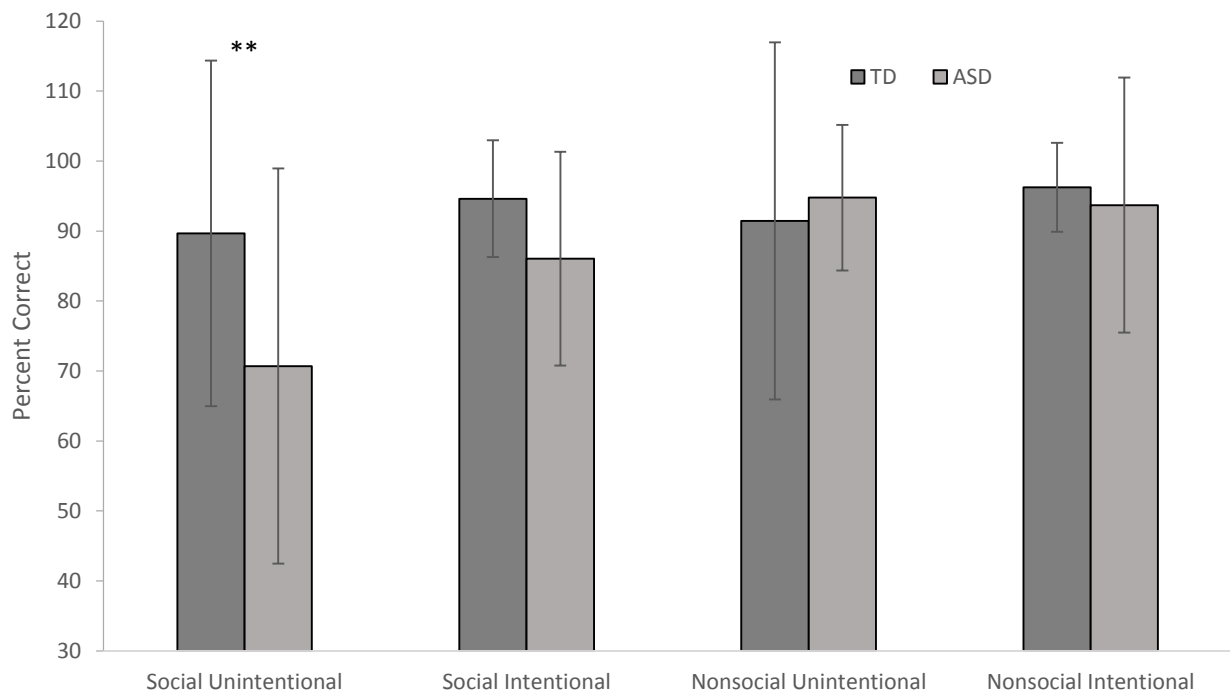
Behavioral Performance Measures

Reaction Time. Analysis of reaction time revealed a main effect of stimuli type [$F(1,40)=31.23, p<.001, \eta^2_p = 0.44$], with significantly faster reaction times for the nonsocial stimuli ($M = 663.75, SD = 264.73$) compared with social stimuli ($M = 797.38, SD = 263.00$). We did not find a main effect of group [$F(1,40)=.86, p=.36, \eta^2_p = 0.02$], or a significant stimuli type x group interaction [$F(1,40)=0.01, p=0.92, \eta^2_p = 0.00$]. Taken together, these results suggest that reaction time did differ between stimuli types but was not differentially affected by levels of autistic traits. We are unable to analyze reaction time effects for intentionality as participants only responded to intentional stimuli.

Response Accuracy. The omnibus analysis for response accuracy evidenced main effects of stimuli type [$F(1,40)=26.12, p<.001, \eta^2_p = 0.40$] and intentionality [$F(1,40)=4.67, p<.05, \eta^2_p = 0.10$], and 2-way interactions of stimuli type x group [$F(1,40)=16.88, p<.001, \eta^2_p = 0.30$] and stimuli type x intentionality [$F(1,40)=12.08, p<.01, \eta^2_p = 0.23$]. However, these analyses were superseded by 3-way interactions of stimuli type x intentionality x group [$F(1,40)=11.63, p<.001, \eta^2_p = 0.23$]. We did not identify a main effect of group [$F(1,40)=2.41, p=.13, \eta^2_p = 0.06$], or a group x intentionality interaction [$F(1,40) = 0.15, p = 0.70, \eta^2_p = 0.00$]. Decomposition of the stimuli type x intentionality x group interaction examined stimuli type x intentionality for each group. The subsidiary ANOVA for typically developing children yielded no significant main effects or interactions (p 's > 0.07). In contrast, the subsidiary ANOVA for children with ASD yielded a stimuli type effect [$F(1,20) = 22.76, p<.001, \eta^2_p = 0.53$] and a significant stimuli type x intentionality interaction [$F(1,20) = 13.10, p<.01, \eta^2_p = 0.40$]. Examination of simple main effects indicated that children with ASD were significantly more accurate in responding to

nonsocial stimuli ($M = 94.10$, $SD = 8.20$) than social stimuli ($M = 78.39$, $SD = 19.01$). Following up the significant stimuli type $\times$ intentionality interaction, post hoc t tests indicated greater accuracy for intentional stimuli ($M = 86.06$, $SD = 15.28$) relative to unintentional stimuli ($M = 70.71$, $SD = 28.24$) for social trials only, $t(20) = 3.59$, $p < 0.01$. Taken together, these results indicate that typically developing children and children with ASD do not differ in their response accuracy for nonsocial stimuli, but that children with ASD perform significantly worse than controls when discriminating intentional from unintentional social stimuli. This is largely driven by children with ASD's poorer performance on social stimuli unintentional trials. Accuracy performance is summarized in Figure 5.

Figure 5. Response accuracy (%) for Study 2's ERP task by group as a function of stimuli type and intentionality.



** $p < 0.01$

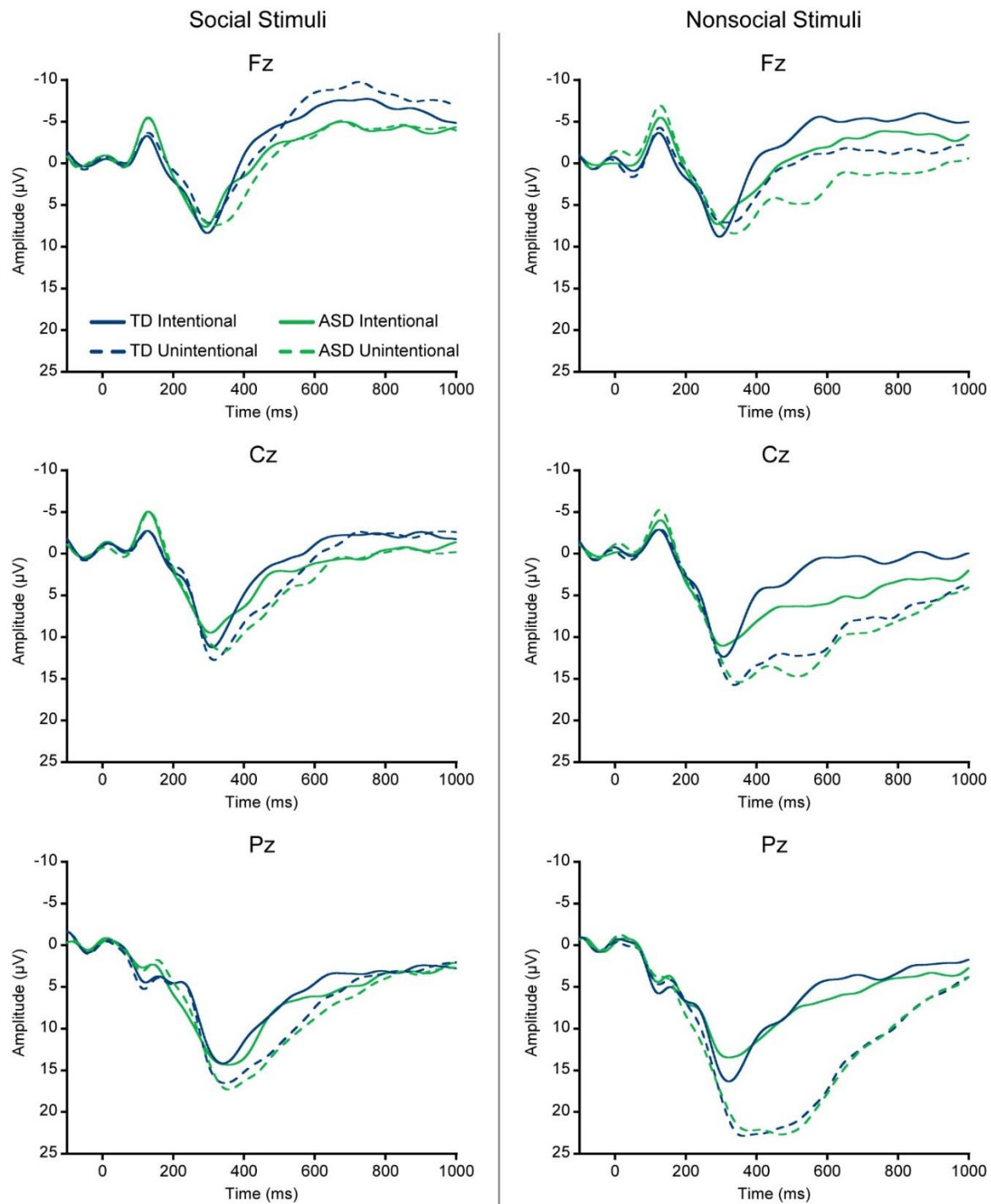
Neuroelectric Measures

LPC Amplitude. The omnibus analysis evidenced main effects of stimuli type [$F(1,40)=23.35, p<.001, \eta^2_p = 0.37$], intentionality [$F(1,40)=68.01, p<.001, \eta^2_p = 0.63$], and site [$F(2.05,82.02)=41.00, p<.001, \eta^2_p = 0.51$], and 2-way interactions of stimuli type x intentionality [$F(1,40)=20.48, p<.001, \eta^2_p = 0.34$] and intentionality x site [$F(2.62,104.75)=6.62, p<.01, \eta^2_p = 0.14$]. However, these analyses were superseded by 3-way interactions of stimuli type x intentionality x site [$F(2.73,109.20)=3.31, p<.05, \eta^2_p = 0.08$]. There were no main effects or interactions involving group (all p 's >0.36). Decomposition of the stimuli type x intentionality x site interaction examined stimuli type x intentionality at each electrode site. The subsidiary ANOVA for Fz yielded a stimuli type effect [$F(1,41)=8.41, p<.01, \eta^2_p = 0.17$] with increased amplitude for nonsocial ($M = 8.43, SD = 7.93$) relative to social ($M = 5.97, SD = 7.46$) stimuli, and an intentionality effect [$F(1,41)=10.22, p<.01, \eta^2_p = 0.20$] with increased amplitude for unintentional ($M = 8.56, SD = 8.06$) relative to intentional trials ($M = 5.83, SD = 7.36$). Electrode site FCz revealed effects of stimuli type [$F(1,41)=15.97, p<.001, \eta^2_p = 0.28$] and intentionality [$F(1,41)=21.02, p<.001, \eta^2_p = 0.34$], which were superseded by a stimuli type x intentionality interaction [$F(1,41)=6.13, p<.05, \eta^2_p = 0.13$]. Post hoc t tests indicated larger amplitude for nonsocial stimuli relative to social stimuli for unintentional trials, $t(41) = 5.15, p < 0.001$.

Electrode site Cz also yielded effects of stimuli type [$F(1,41)=16.15, p<.001, \eta^2_p = 0.28$] and intentionality [$F(1,41)=26.60, p<.001, \eta^2_p = 0.39$], which were superseded by a stimuli type x intentionality interaction [$F(1,41)=8.87, p<.01, \eta^2_p = 0.18$]. Post hoc t tests indicated larger amplitude for nonsocial stimuli relative to social stimuli for unintentional trials, $t(41) = 5.75, p < 0.001$. Similarly, electrode site CPz yielded effects of stimuli type [$F(1,41)=17.65, p<.001, \eta^2_p = 0.30$] and intentionality [$F(1,41)=47.10, p<.001, \eta^2_p = 0.54$], which were superseded by a stimuli

type x intentionality interaction [$F(1,41)=11.34, p<.01, \eta^2_p = 0.22$]. Post hoc t tests indicated larger amplitude for nonsocial stimuli relative to social stimuli for unintentional trials, $t(41) = 6.14, p < 0.001$). Analyses at electrode site Pz again yielded effects of stimuli type [$F(1,41)=20.38, p<.001, \eta^2_p = 0.33$] and intentionality [$F(1,41)=102.52, p<.001, \eta^2_p = 0.71$], which were superseded by a stimuli type x intentionality interaction [$F(1,41)=34.48, p<.01, \eta^2_p = 0.46$]. Post hoc t tests indicated larger amplitude for nonsocial stimuli relative to social stimuli for unintentional trials, $t(1,41) = 7.36, p < 0.001$). The subsidiary ANOVA for electrode site POz also yielded effects of stimuli type [$F(1,41)=10.53, p<.001, \eta^2_p = 0.20$] and intentionality [$F(1,41)=81.02, p<.001, \eta^2_p = 0.66$], which were superseded by a stimuli type x intentionality interaction [$F(1,41)=8.77, p<.01, \eta^2_p = 0.18$]. Post hoc t tests indicated larger amplitude for nonsocial stimuli relative to social stimuli for unintentional trials, $t(1,41) = 3.48, p < 0.001$). Finally, electrode site Oz also revealed effects of stimuli type [$F(1,41)=27.24, p<.001, \eta^2_p = 0.40$] and intentionality [$F(1,41)=65.87, p<.001, \eta^2_p = 0.62$], which were superseded by a stimuli type x intentionality interaction [$F(1,41)=25.31, p<.001, \eta^2_p = 0.38$]. Post hoc t tests indicated larger amplitude for nonsocial stimuli relative to social stimuli for unintentional trials, $t(1,41) = 7.13, p < 0.001$). Refer to Figure 6 for grand average waveforms time-locked to terminal stimulus in each sequence for each stimuli and intention type by group.

Figure 6. The left panel provides grand average waveforms time-locked to terminal stimulus in each sequence at Fz, Cz, and Pz for social intentional and unintentional stimuli for typically developing children and children with ASD from Study 2. The right panel provides grand average waveforms time-locked to terminal stimulus in each sequence at Fz, Cz, and Pz for nonsocial intentional and unintentional stimuli for typically developing children and children with ASD from Study 2

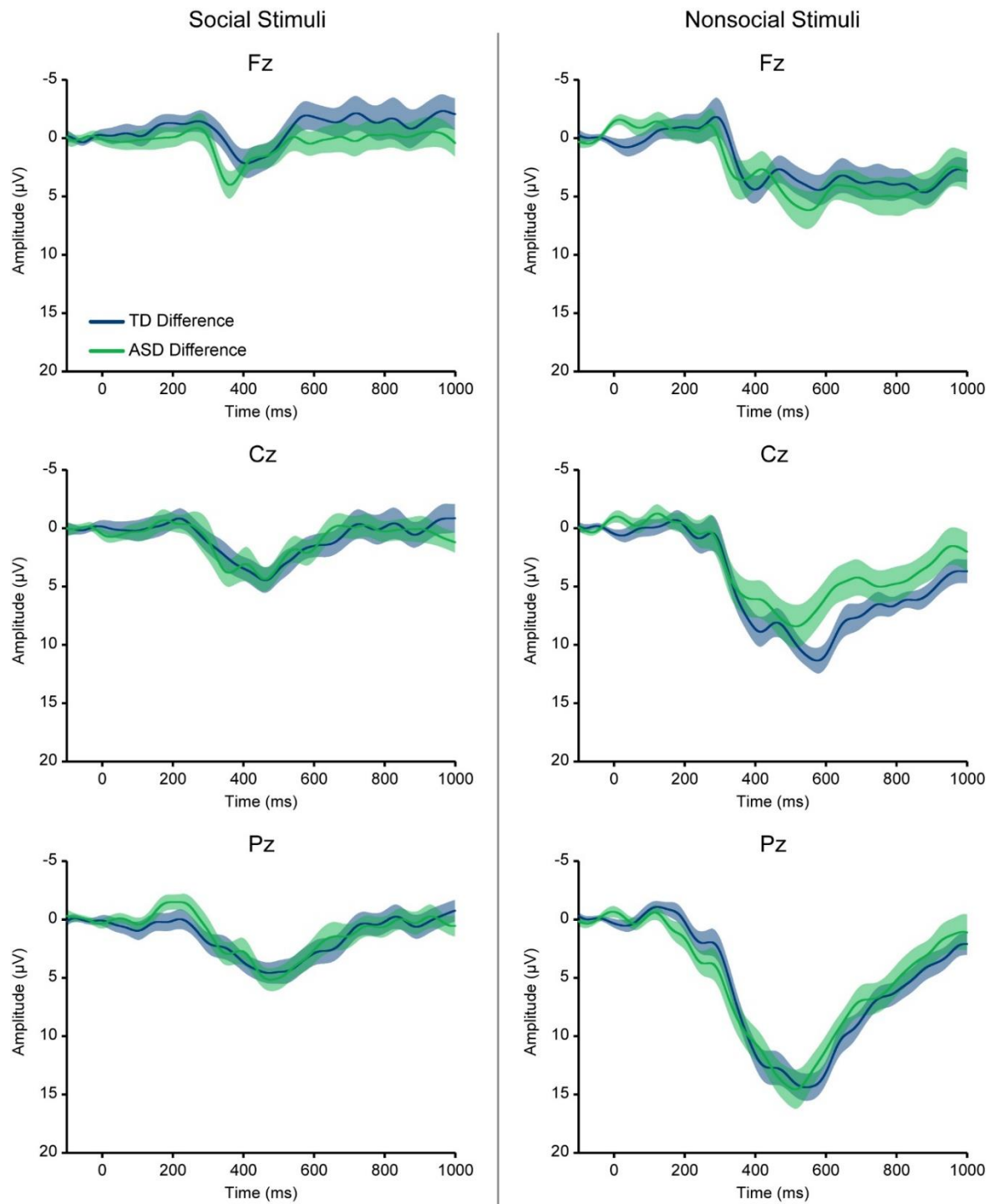


LPC Latency. The omnibus analysis evidenced main effects of stimuli type [$F(1,40)=13.32, p<.01, \eta^2_p = 0.25$] and site [$F(3.49,139.56)= 3.26, p<.05, \eta^2_p = 0.08$], which were superseded by 2-way interactions of stimuli type x site [$F(4.07,162.90)=4.96, p<.01, \eta^2_p = 0.11$] and intentionality x site [$F(4.02,160.73) = 5.15, p<.01, \eta^2_p = 0.11$]. There was no main effect of intentionality ($p = 0.06$), and there were no main effects or interactions involving group (all p 's >0.36). Decomposition of the stimuli type x site interaction used Bonferroni corrected t tests to compare social and nonsocial stimuli at each electrode site. Results indicated significantly longer LPC latencies for nonsocial stimuli at FCz ($M = 417.67, SD = 54.86$), Cz ($M = 435.55, SD = 95.23$), CPz ($M = 459.36, SD = 94.22$), and Pz ($M = 420.85, SD = 77.66$) relative to social stimuli (FCz: $M = 377.96, SD = 53.43$; Cz: $M = 376.63, SD = 60.76$; CPz: $M = 395.16, SD = 67.31$; Pz: $M = 392.01, SD = 55.00$).

Decomposition of the intentionality x site interaction used Bonferroni corrected t tests to compare intentional and unintentional trials at each electrode site. Results indicated significantly longer LPC latencies for unintentional trials at CPz ($M = 440.01, SD = 80.22$), Pz ($M = 422.23, SD = 73.51$), POz ($M = 414.04, SD = 64.49$), and Oz ($M = 408.16, SD = 69.58$) relative to intentional trials (CPz: $M = 414.50, SD = 76.97$; Pz: $M = 390.63, SD = 54.89$; POz: $M = 383.02, SD = 52.79$; Oz: $M = 378.02, SD = 41.80$).

Difference Waves. The difference waves, formed by subtracting intentional from unintentional ERPs are plotted in Figure 7. For both stimuli types, the most prominent feature was a large positivity beginning approximately 300 ms after stimulus onset with a duration of several hundreds of milliseconds. This represents the LPC effect.

Figure 7. The left panel depicts grand average difference waves at Fz, Cz, and Pz illustrating the difference between intended and unintended stimuli for social stimuli for typically developing children and children with ASD from Study 2. The right panel depicts grand average difference waves at Fz, Cz, and Pz illustrating the difference between intended and unintended stimuli for nonsocial stimuli for typically developing children and children with ASD from Study 2. The shaded area surrounding difference waves represents standard error.



The average amplitude of the LPC effect across all electrodes was larger for nonsocial than social stimuli for both groups [main effect of stimuli type: $F(1,40) = 46.87, p < 0.001$; Nonsocial: $M = 13.25, SD = 4.55$; Social: $M = 7.03, SD = 3.42$]. A main effect of electrode site ($F(1.80,71.90) = 7.39, p < 0.01$) indicated that the LPC effect was maximal over parietal electrode sites for both groups (see Table 6 and Figure 8). The interaction between stimuli type and electrode site was non-significant after correcting for non-sphericity ($p = 0.05$). No main effects or interactions involving group were identified (all p 's > 0.05).

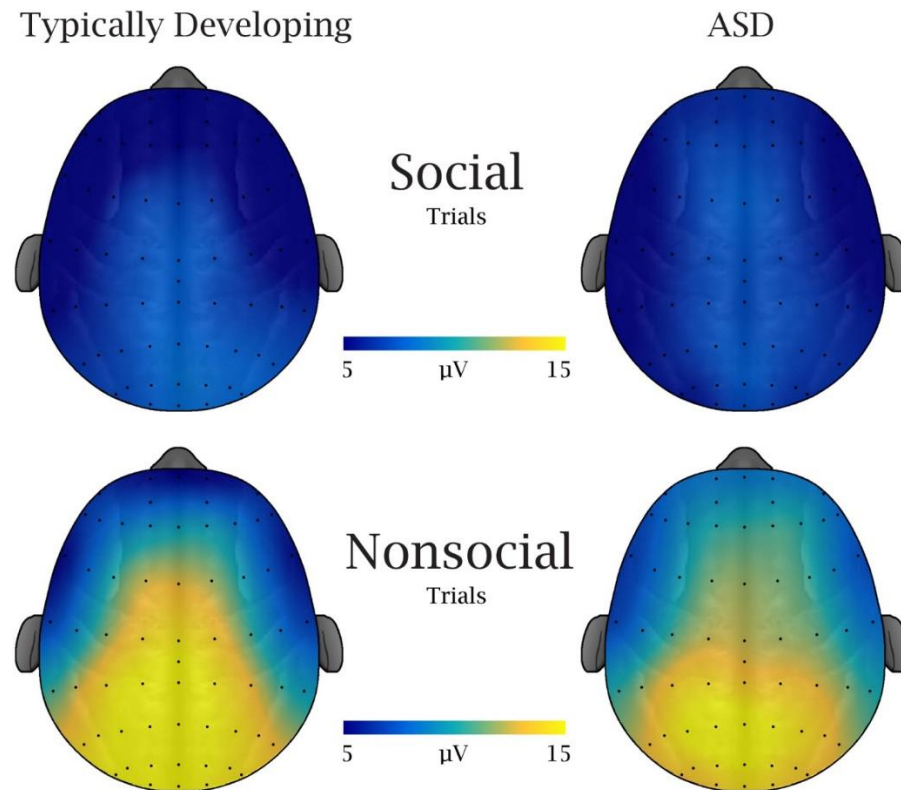
Table 6

Study 2: LPC Effect Mean Amplitude by Electrode Site Collapsing Across Groups

Electrode Site	Mean Amplitude (SD)
Fz	7.71 (4.28) ^a
FCz	9.16 (4.15) ^b
Cz	9.94 (4.03) ^c
CPz	10.38 (3.72) ^d
Pz	11.76 (4.00) ^e
POz	12.11 (5.15) ^f
Oz	9.92 (4.61) ^g

Note. a is significantly different from b-f, $p < 0.01$. e and f are significantly different from g, $p < 0.05$.

Figure 8. Topographic maps of the LPC effect: peak amplitude difference between ERPs of the intention condition subtracted from the unintentional condition separated for typically developing children and children with ASD in Study 2. The LPC represents the 300–700ms post-stimulus epoch for both groups of children.



Relationship Between LPC Effect and Autism Symptomatology

Bivariate correlations indicated no significant relationships between the magnitude of the nonsocial LPC effect and autistic symptomatology at any electrode site. The magnitude of the social LPC effect was significantly negatively associated with Social Awareness at Cz, CPz, and POz, and with Social Cognition at CPz (see Table 7). These patterns indicate that as level of impairment in social functioning increase, one's ability to differentiate between intentional and unintentional social actions decreases. No significant relationships between social LPC effect were identified for the subscales social communication, social motivation, or restricted and repetitive behaviors.

Table 7

Study 2: Bivariate Correlations Between SRS Subscale Scores and LPC Effect by Stimuli Type and Electrode Site Collapsing Across Groups

	Social Awareness	Social Cognition	Social Communication	Social Motivation	Restricted Interests and Repetitive Behaviors
Fz					
Nonsocial Stimuli	.10	.05	.05	.08	.01
Social Stimuli	-.07	-.03	-.05	.09	-.08
FCz					
Nonsocial Stimuli	.01	-.08	-.02	.02	-.05
Social Stimuli	-.15	-.09	-.09	.06	-.10
Cz					
Nonsocial Stimuli	-.10	-.11	-.06	.04	-.04
Social Stimuli	-.40**	-.28	-.28	-.10	-.24
CPz					
Nonsocial Stimuli	-.13	-.22	-.14	-.05	-.18
Social Stimuli	-.42**	-.33*	-.29	-.14	-.21
Pz					
Nonsocial Stimuli	.12	.02	.09	.17	.12
Social Stimuli	-.26	-.28	-.21	-.21	-.20
POz					
Nonsocial Stimuli	.02	.01	.06	.16	.12
Social Stimuli	-.31*	-.29	-.21	-.18	-.22
Oz					
Nonsocial Stimuli	.02	-.05	.02	.05	.08
Social Stimuli	-.24	-.16	-.15	-.18	-.10

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

To better understand the specificity of the relationship between social functioning and LPC effect magnitude, we conducted additional exploratory analyses to determine the degree to which social awareness versus response accuracy is predictive of the LPC effect at Cz, CPz, and POz electrode sites. Three multiple linear regressions (Cz, CPz, POz) were calculated to predict

LPC effect magnitude based on social awareness and response accuracy. A single measure of response accuracy (d') was used in these analyses, which was calculated by subtracting the standardized values of incorrect responses to unintentional stimuli (i.e., false alarms) from correct responses to intentional stimuli (i.e., hits). Overall, significant regression equations were found at Cz and CPz, such that social awareness (but not response accuracy) predicted LPC effect magnitude at these sites. The regression equation at POz was not significant. Regression results are summarized in Table 8.

Table 8

Study 2: Summary of Multiple Regression Analyses for Variables Predicting LPC Effect Magnitude Collapsing Across all Study 2 Participants (N=42)

Variable	Cz			CPz			POz		
	<i>B</i>	SE <i>B</i>	β	<i>B</i>	SE <i>B</i>	β	<i>B</i>	SE <i>B</i>	β
Response Accuracy	.16	.37	.07	-.01	.35	-.00	-1.01	.75	-.22
Social Awareness	-.11	.05	-.36*	-.11	.05	-.37*	-.37	.20	-.62
R^2		.15			.14			.10	
<i>F</i>		3.41*			3.14*			1.39	

* $p < .05$

Discussion

Findings from Study 2 were consistent with previous results suggesting a dissociation between social and nonsocial intention understanding in children with ASD (Berger & Ingersoll, 2014). Specifically, we demonstrated that children with ASD experience significant difficulty at a behavioral level identifying actions as intentional versus unintentional when cued using social communicative behaviors (i.e., eye gaze with head turn), but perform as well as typical controls when intentionality is cued via object manipulation. However, contrary to our hypotheses, we did not find that children with ASD were slower at making social intention understanding decisions compared to their typically developing peers. Speculatively, this may be because the embedded

delay in the task between terminal picture and response window masked any potential differences in reaction time that may have been revealed had respondents been free to respond immediately upon processing the stimuli.

Interestingly, the group differences in response accuracy were not reflected at a neurological level such that no differences in the magnitude or latency of the LPC effect were observed between groups. Independent of diagnostic classification, all experimental conditions led to a large bilateral posterior positive component ranging from 300 to 700 ms (at least) and peaking approximately at 400 ms. Despite lack of group differences in psychophysiology, we identified a relationship between the magnitude of the LPC effect and social functioning abilities across participants such that children with lower levels of social functioning were less able to differentiate between social intentional and unintentional stimuli at a neurological level regardless of diagnostic status. This is consistent with the results of Study 1, which identified a relationship between the magnitude of the LPC effect and the socially oriented subscale of the BAPQ in typically developing adults.

GENERAL DISCUSSION

Overall, our results add to the growing body of work associating a late positivity with intention understanding (De Bruijn et al., 2007; Sitnikova et al., 2009, 2008; Van der Cruyssen et al., 2009; Vistoli, Passerieux, Zein, et al., 2015; Vistoli, Brunet-gouet, Baup-bobin, & Passerieux, 2011; Vö & Wolfe, 2013; Y. Wang et al., 2012) and support Sitnikova and colleagues' (2008) position that a late positivity is uniquely associated with the integration of goal-related action information. Both the chronometry (300–700 ms) and the topography (posterior electrodes) of the observed effect in typically developing adults, typically developing children, and children with ASD are in accordance with previous ERP and MEG results showing that attribution of intentions to others in typically developing adults elicits strong neural activations in the right posterior Superior Temporal Sulcus (pSTS), Intraparietal Lobule (IPL), and Temporal Parietal Junction (TPJ) during the 200- to 600-ms time window (Vistoli et al., 2011).

For all participants across both studies, the type of stimuli and degree of intentionality depicted in the sequence modulated LPC amplitude: an enhanced positivity was observed in the unintentional compared to intentional conditions, and for nonsocial compared to social stimuli. Similarly, longer peak amplitude latencies were observed for nonsocial compared to social stimuli. While an enhanced response to unintentional compared to intentional stimuli is in line with other work showing recruitment of additional neurological resources for processing unanticipated events (e.g., Amoruso et al., 2013), the observed larger effect for nonsocial compared to social stimuli differs from one other study assessing the neural correlates of intention understanding and is the reverse of what we had hypothesized in Study 1. In their work comparing social and nonsocial intention, Wang and colleagues found that social intention

evoked a larger LPC compared to nonsocial intention (Y. Wang et al., 2012). We expect that the reason our results diverge from those of Wang and colleagues (2012) is because their social stimuli were significantly more complex. In their study, social intention was depicted by two figures interacting with one another. This required the participant to assess intentionality by integrating information across both figures, compared to our stimuli in which intention was conveyed directly to the participant via very basic social communicative cues. Our ERP findings of greater neurological activity associated with processing nonsocial compared to social intention better reflects the ontogeny of intention understanding described in the developmental literature such that basic social intention understanding emerges prior to nonsocial intention understanding, and research indicating that there is a gradient of difficulty in intention understanding from physical causality to social intention to nonsocial intention (Leslie, 1982; Spelke, Breinlinger, Macomber, & Jacobson, 1992; Wellman, Cross, & Waston, 2001).

Based on behavioral studies identifying a dissociation between understanding social and nonsocial intention in children with ASD, in Study 2 we had hypothesized that children with ASD would differentiate between intentional and unintentional stimuli at a neurological level to a lesser degree than typically developing controls, and that no differences in ERP effects would be observed between groups for nonsocial stimuli. Our ERP results failed to support this hypothesis. For *both* social and nonsocial stimuli, no group differences were observed in terms of either peak amplitude or latency of the LPC components for intentional or unintentional conditions, nor were group differences observed when examining the LPC effect. This unexpected finding regarding lack of group differences suggests that the LPC more likely maps onto specific processes reflecting individual symptoms or symptom clusters rather than diagnostic categories. Indeed, a diagnosis of ASD requires the presence of impairment in both

social reciprocity and the presence of restricted interests and/or repetitive behaviors. While there is substantial research linking the LPC to intention understanding broadly defined (De Bruijn et al., 2007; Sitnikova et al., 2009, 2008; Van der Cruyssen et al., 2009; Vistoli, Passerieux, Zein, et al., 2015; Vistoli et al., 2011; Vö & Wolfe, 2013; Y. Wang et al., 2012), we could identify no research associating this effect with restricted interests or repetitive behaviors. Given the widely acknowledged heterogeneity of the ASD population, it is thus unsurprising that we failed to identify an effect at the group level.

Additional support for our position that the LPC reflects symptom specific processes comes from our correlational findings. Results from Study 1 utilizing a population of typically developing adults with subclinical autism symptomatology found that the magnitude of the LPC effect for social stimuli was significantly related to the subscale of the Broad Autism Phenotype Questionnaire tapping social functioning, but not the subscales tapping pragmatic language or behavioral rigidity. Building upon these findings, we found that when collapsing across groups in Study 2 to assess autism symptomatology dimensionally, the magnitude of the social LPC effect was inversely related to level of social functioning at centroparietal sites. No relationships were identified between magnitude of the social LPC effect and level of communication or restricted interests / repetitive behaviors, suggesting that the LPC effect is specifically related to social functioning and not other symptoms of ASD. Taken together, these results suggest that regardless of diagnostic status, individuals with lower levels of social awareness are less able to differentiate between intentional and unintentional social stimuli.

While the ERP results from Study 1 and Study 2 appear to reflect symptomatology dimensionally, the observed behavioral data from Study 2 do map onto diagnostic categories. We found that as expected, children with ASD and typically developing children were equally

accurate in determining the intentionality of nonsocial stimuli, but children with ASD performed significantly worse than controls on social stimuli trials. In comparison to the clear lack of group differences reflected in the LPC, behavioral accuracy data seemed to reflect a lack of social intention understanding in the ASD group. The response accuracy data aligns with qualitative remarks made by many of the participants with ASD during the social practice trials such as “How am I supposed to know which one he will pick?”, “He’ll pick the cookie because cookies are good”, or “He’ll pick the soda because soda is for grown-ups”. This is wholly consistent with the broader intention understanding literature suggesting that children with ASD are functionally impaired on behavioral tasks of social, but not nonsocial, intention understanding. The dissociation between ERP findings and behavioral accuracy data suggests that the two measures are likely reflective of (or influenced by) somewhat different processes that unfold over time. This is supported by our multiple regression analyses, which indicated that the LPC effect was predicted by social functioning level, but not accuracy. Speculatively, based on the data discussed herein we hypothesize that the LPC is associated with a specific neurological process (related to social functioning) while response accuracy likely reflects the summation of a number of different cognitive operations. Future studies combining ERP and behavioral measures are necessary to further illuminate their relationship, and underscore the importance of examining constructs across multiple levels of analysis. Nevertheless, the fact that children with and without ASD and adults without ASD showed the same relationship between LPC effect magnitude and social functioning in our studies gives credence to the idea that children with ASD may benefit from treatment targeting social intention understanding. Impairments in social intention understanding processing may not be the underlying cause of ASD, but social intention understanding circuitry could be a promising therapeutic target nonetheless given that the

understanding of goal directed action has been hypothesized to act as a foundation skill for development of higher level social cognitive skills such as joint attention and theory of mind.

Our findings that the LPC social effect is not uniquely associated with ASD, but instead is related to individual differences in human social functioning (including the severe social impairments which characterize ASD) has implications for the literature attempting to identify biomarkers or endophenotypes unique to ASD. While a number of promising biomarkers for ASD have been identified including mitochondrial function, oxidative stress, and immune stress, (see Goldani, Downs, Widjaja, Lawton, & Hendren, 2014 for a review), no single biomarker to date has sufficient evidence to support routine clinical use. Indeed, attempts at using EEG to detect abnormalities unique to ASD have met with limited success (Griffin & Westbury, 2011). Study 2's observed lack of group differences in the context of significant relationships when measuring individual symptoms suggests that neurological biomarkers may be better identified by examining symptom clusters that cut across diagnostic categories. Our results suggest that the magnitude of the LPC social effect is related to social functioning in both children and adults, and may have utility as a biomarker for social functioning broadly defined. Given that the paradigm taps the earliest emerging form of intention understanding (documented in typically developing children 6-9 months of age; (H. L. Marsh, Stavropoulos, Nienhuis, & Legerstee, 2010), this task could be used to assess the extent to which infants may be at risk for difficulties in social functioning, and subsequently refer children for early intervention. This is consistent with the Research Domain Criteria (RDoC), which aim to define basic dimensions of functioning cutting across disorders as traditionally defined. Additional work would be necessary to establish the validity of this paradigm with infants, and to define the critical value of the LPC social effect associated with "impaired" social functioning.

Results of Study 1 and Study 2 also inform existing theories of intention understanding development. Tomasello and colleagues (2005) have argued that goal-directed intention understanding emerges as a function of cognitive understanding of goal-directed action. Their theory holds that the understanding of goal-directed action is separate from the social motivation to share psychological states, and together they form the foundation for joint attention. Our Study 2 group level analyses seemingly support this position, such that both groups demonstrated an equal understanding of intention at a neural level independent of how it was assessed (i.e., no differences in LPC effect magnitude across groups for either social or nonsocial stimuli), but that children with ASD were functionally impaired in their ability to demonstrate this understanding for social stimuli (potentially related to motivation to share psychological states). However, independent of disorder status, across Study 1 and Study 2 we found that social functioning predicted one's ability to differentiate between intentional and unintentional social actions. Thus, rather than representing separate, additive entities as suggested by Tomasello et al. (2005), our results suggest that cognitive understanding of goal-directed actions and social functioning overlap and work in tandem to allow an individual to fully understand goal-directed intention.

It is important to note that the present findings have several limitations. First of all, our paradigm does not tap understanding of all aspects of attributing intentions to actions. Rather, results reported here are consistent with developmental theories of intention understanding indicating that the earliest emerging form of intention understanding is understanding the pursuit of goals via goal-directed action (Leslie, 1994; Tomasello et al., 2005). As the extant literature has thus far been equivocal regarding the extent to which children with ASD have intact or impaired intention understanding, we chose to focus on this most basic form of intention understanding rather than higher levels of intention understanding that are more concerned with

rationality and propositional states (Leslie, 1994; Tomasello et al., 2005). Indeed, the high degree of consistency of the LPC (both in terms of chronicity and topography) across child and adult samples suggests that we are tapping a construct that emerges early in development. Our findings are also limited by our choice of stimuli, such that stimuli depicted only one type of social intention (eye gaze to indicate intention to select an object) and one type of nonsocial intention (lifting an object up to consume it). While this was done in order to promote consistency across trials and facilitate matching across conditions, it somewhat limits the generalizability of our results. However, in light of other research finding similar ERP results with a wide variety of stimuli including execution errors in everyday actions (De Bruijn et al., 2007), goal inferences from paragraph descriptions of actions (Van der Cruyssen et al., 2009), and comic strip scenes depicting a wide variety of action (Vistoli, Passerieux, Zein, et al., 2015), it appears that the LPC response to intentional action is quite robust. This is consistent with our finding that social functioning is predictive of the magnitude of the LPC effect, such that we would expect the effect to be evoked across a wide variety of tasks as long as the paradigm was tapping processes related to social functioning. However, it remains for future research to test this directly and assess whether the same connection between social intention understanding when cued using eye gaze and ASD symptomatology would extend to other types of social intention understanding.

It is also important to note that participants across both studies received instructions asking them to reflect whether or not the actor “did what he meant to do”. These instructions thus explicitly cued participants to reflect on the intention underlying the actor’s action. Given that there is mixed research investigating the extent to which instructions on mentalizing tasks modify (A. T. Wang, Lee, Sigman, & Dapretto, 2006) or do not modify (Iacoboni et al., 2005; Vistoli, Passerieux, El Zein, et al., 2015) results, future research should assess the extent to

which passive viewing of these stimuli elicits the same ERP results. This would allow researchers to assess the extent to which detection of intention occurs by default in ASD, or if it relies on explicit activation of the mentalizing system. Similarly, it would be interesting to modify instructions to explicitly direct children with ASD's attention to the social-communicative cues and assess the extent to which behavioral accuracy changes. These types of manipulations would facilitate an enhanced understanding of the conditions under which children with autism display intact or impaired intention understanding across neurological and behavioral levels.

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