

# **Atypical neural responding to hearing one's own name in adults with ASD**

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## **Author note**

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## Abstract

Diminished responding to hearing the own name is one of the earliest and strongest predictors of autism spectrum disorder (ASD). Here, we studied for the first time the neural correlates of hearing one's own name in ASD. Based on existing research, we hypothesized enhancement of late parietal positive activity specifically for the own name in neurotypicals, and this effect to be reduced in adults with ASD. Source localization analyses were conducted to estimate group differences in brain regions underlying this effect. 21 adults with ASD, and 21 age- and gender-matched neurotypicals were presented with three categories of names (own name, close other, unknown other) as task-irrelevant deviant stimuli in an auditory oddball paradigm, while EEG was recorded. As expected, a late parietal positivity was observed specifically for own names in neurotypicals, indicating enhanced attention to the own name. This preferential effect was absent in the ASD group. This group difference was associated with diminished activation in the rTPJ in adults with ASD. Further, a familiarity effect was found for the N1, with larger amplitudes for familiar names (own name and close other). However, groups did not differ for this effect. These findings provide evidence of atypical neural responding to hearing one's own name in adults with ASD, suggesting a deficit in self-other distinction, associated with rTPJ dysfunction.

*Keywords:* autism spectrum disorder; ERP; own name; TPJ

*General Scientific Summary:* Infants at risk of ASD are known to show a diminished response to hearing their own name. By investigating the neural response to hearing their own name in adults with ASD, we showed for the first time that also in adulthood, individuals with ASD show an atypical response to hearing their name.

## Introduction

Autism spectrum disorder (ASD) is a neurodevelopmental disorder characterized by difficulties in social communication and interaction, as well as by restricted, repetitive patterns of behavior, interests or activities (American Psychiatric Association, 2013), assumed to be caused by brain abnormalities during early development (Courchesne, Campbell, & Solso, 2011; Minshew & Keller, 2010).

One of the earliest and strongest predictors for developing ASD is a diminished orienting response to the own name (Werner, Dawson, Osterling, & Dinno, 2000). Typically, infants respond to hearing their own name by 5 months of age (Parise, Friederici, & Striano, 2010), but retrospective studies investigating home videos of children in their first year of life that were later diagnosed with ASD, found that these children failed to respond to their name much more often than typically developing children (Baranek, 1999; Osterling & Dawson, 1994). Prospective studies with infants at risk for ASD at the age of one (Nadig et al., 2007; Zwaigenbaum et al., 2005) also found that these children showed a strongly diminished response to hearing their own name in comparison to low-risk children. Importantly, research suggests that the diminished response to the own name in infants at risk for ASD does not simply reflect a delay in language development (Nadig et al., 2007).

One's own name is a salient stimulus that is uniquely related to oneself. Research shows that one's own name is easily detected among other stimuli and enters awareness more easily than other salient information (the 'cocktail party effect', Wood & Cowan, 1995); it has even been shown to elicit a strong and robust neural response in patients that are comatose, with locked-in syndrome, or in vegetative state (Fischer, Dailler, & Morlet, 2008; Perrin et al., 2006). Such a preferential response to one's own name is considered of importance for joint attention and social interaction (Kampe, Frith, & Frith, 2003), although responding to hearing

the own name is not a necessary condition, as has been shown in deaf children (Nowakowski, Tasker, & Schmidt, 2009). Research has shown that neurotypical infants of five months old not only detect their own name, but also use it as a social cue to guide their attention (Parise et al., 2010). Among several social cues, what makes the infant's own name special is that it is an ostensive cue that is unique to each individual infant, signaling that another person intends to attract attention and to start communication about something of relevance in the external world. The ability to detect social signals directed at the self is of critical importance to successfully socially interact with others, and share new knowledge about the external world. Hence the inability to do so during infancy may severely impact social-cognitive development (Parise et al., 2010; Werner et al., 2000).

Surprisingly, to our knowledge, the neural correlates of auditory processing of the own name in ASD have never been investigated systematically. It was therefore the aim of the current study to study these neural correlates in adults with ASD, as it would be highly informative to know whether the own name, when heard, is also processed atypically in adults with ASD. First, this would indicate that atypical processing of the own name may reflect a fundamental deficit that is still apparent in adulthood. Second, this may point to specific mechanisms underlying the lack of response to the own name in children at risk for ASD.

Two recent event-related potential (ERP) studies investigated the neural response to seeing one's own name (versus other names) in adults with ASD (Cygan, Tacikowski, Ostaszewski, Chojnicka, & Nowicka, 2014; Nowicka, Cygan, Tacikowski, Ostaszewski, & Kuś, 2016), and reported an enhancement of a late parietal positivity for the own name versus that of a close other in neurotypicals, which was not observed in the ASD group. This lack of differentiation between the own name and the name of a close other is in accord with other research suggesting deficient self-other distinction in ASD. Several researchers have argued that self-other distinction (the ability to distinguish between and control neural representations

of the self and others) is a low-level neurocognitive mechanism that lies at the base of many social-cognitive processes, and that a self-other distinction deficit may explain impairments in empathy, mentalizing, and self-referential processing in ASD (see for comprehensive discussions De Guzman, Bird, Banissy, & Catmur, 2015; Sowden & Shah, 2014; Spengler, Bird, & Brass, 2010). There is indeed ample evidence of compromised self-other distinction in ASD coming from behavioral and neuroimaging studies showing diminished differentiation between representations of self- and other-related information across several domains (De Coster, Wiersema, Deschrijver, & Brass, 2017; Deschrijver, Wiersema, & Brass, 2016; Kennedy & Courchesne, 2008; Lombardo, Chakrabarti, Bullmore, & Baron-Cohen, 2011; Pfeifer et al., 2013; Spengler et al., 2010).

Neuroimaging studies from our lab and others that systematically investigated self-other distinction have pointed out a distinctive role of the right temporo-parietal junction (rTPJ), sometimes accompanied by the medial prefrontal cortex (mPFC), in the process of distinguishing the self from others (Santiesteban et al., 2012; Sowden & Shah, 2014; Spengler, von Cramon, & Brass, 2009). From the beginning of our research (Brass, Derrfuss, & Von Cramon, 2005), we have related self-other distinction to the rTPJ and mPFC, by means of the imitation-inhibition task, which measures self-other distinction at the level of action planning. In follow-up studies, we showed that the rTPJ may be specifically involved in self-other distinction, and demonstrated that rTPJ activation during this low-level task overlapped with activation during mentalizing, reinforcing the hypothesis of self-other distinction being an essential mechanism underlying mentalizing (Spengler et al., 2009). Neurostimulation studies from our lab and others further showed a causal role of the rTPJ in self-other distinction (Bardi, Gheza, & Brass, 2017; Hogeveen et al., 2014; Santiesteban, Banissy, Catmur, & Bird, 2012; Sowden & Catmur, 2015). Research has shown reduced TPJ, and sometimes also mPFC activity in ASD during self-other distinction across several

domains, including mentalizing (Eddy, 2016; Kennedy & Courchesne, 2008; Murdaugh, Nadendla, & Kana, 2014; Pfeifer et al., 2013; Spengler et al., 2010), adding to accumulating evidence that the TPJ is a core region implicated in ASD (Chien, Lin, Lai, Gau, & Tseng, 2015; Fishman, Keown, Lincoln, Pineda, & Müller, 2014; Kana, Uddin, Kenet, Chugani, & Müller, 2014). Importantly, the TPJ has also been found activated in the context of hearing one's own name (Carmody & Lewis, 2006; Holeckova et al., 2008; Perrin et al., 2005). However, the evidence to date is mixed, as it was not seen in three other studies (Kampe et al., 2003; Tacikowski et al., 2011; Tacikowski, Brechmann, & Nowicka, 2013), while other regions besides the TPJ, such as mPFC and right inferior frontal gyrus (rIFG)/insula, have also been reported.

In the current ERP study, we chose to test the response to own versus other names in the auditory modality, as spoken names have a clear communicative (ostensive) function, and are more ecologically valid than written names. Besides, auditory presentation provides a better comparison to the own-name findings in infants at risk for ASD and allows for testing across the life span. We tested high-functioning adults with ASD, in order to exclude possible explanations in terms of language delays or difficulties, or not knowing one's own name. EEG studies have shown that hearing one's own name, similar to seeing one's own name, consistently shows an enhancement of a parietal positivity (PP), also referred to as P300 or P3b (Eichenlaub, Ruby, & Morlet, 2012; Folmer & Yingling, 1997; Perrin et al., 2005, 2006; Perrin, García-Larrea, Mauguière, & Bastuji, 1999; Tateuchi, Itoh, & Nakada, 2012). This parietal positive deflection, starting 300 ms or later after stimulus onset, is argued to reflect updating of stimulus representations held in working memory and the amount of top-down attention allocated to the stimulus (Debener, Makeig, Delorme, & Engel, 2005; Kok, 2001). This component is typically elicited in paradigms in which participants have to detect and respond to a target. The fact that the spontaneous neurophysiological response to the own

name is similar to detecting a target in an explicit target detection task indicates that the own name, because of its inherent salient nature, is implicitly processed as a target stimulus (Perrin et al., 1999). Importantly, other research has linked this ERP component to self-other distinction more generally (Knyazev, 2013), as well as to disrupted self-other distinction in ASD (Deschrijver et al., 2016).

We compared brain responses to hearing the participants' own name both to the name of a chosen close other, and the name of an unknown other, to be able to disentangle effects of self-other distinction from effects of familiarity or personal relevance. As in previous research, we applied an oddball paradigm, enabling the investigation of early and late (selective) attentional processes (Eichenlaub et al., 2012; Holeckova, Fischer, Giard, Delpuech, & Morlet, 2006). Within this paradigm, the names were presented as equally infrequent, task-irrelevant stimuli that did not require a response, to make sure the observed effects were due to the inherent salience of the names capturing attention, and were not confounded by potential differences in task-relevant processing. Finally, in order to control for overall level of alertness (Eichenlaub et al., 2012), participants were required to respond to an infrequent target sound.

We hypothesized an enhanced PP for one's own name versus the close other's name in neurotypicals, and this effect to be reduced or absent in adults with ASD. In keeping with the hypothesis of a deficit in self-other distinction in ASD, we expected this group difference to be accompanied by attenuated activity in the rTPJ and/or mPFC. However, based on the limited neuroimaging literature on auditory own-name processing in neurotypicals, other regions (such as, among others, the rIFG/insula) could also be expected to be implicated. Source localization analyses allowed for testing our specific hypothesis, while involvement of possible other regions could be explored as well.

Earlier ERP components that may be of relevance were explored as well: the N1, reported to show a differentiating involuntary shift of attention to familiar stimuli (Höller et al., 2011; Tateuchi et al., 2012), and the novelty P3 or P3a, reflecting the involuntary orienting of attention towards unexpected, infrequent and salient stimuli (Friedman, Cycowicz, & Gaeta, 2001; Polich, 2007).

## **Methods**

### **Participants**

A group of 24 adults (17 men) with ASD, and an age-matched group of 24 neurotypical control participants (16 men) participated in the study. All participants were right-handed and reported no hearing difficulties. ASD participants were recruited from our own research database, and through an announcement that was distributed both by the Flemish Autism Association and by Tanderuis, an organization providing in-home supervision to individuals with ASD. Control participants were recruited through online advertisements and social media, and had no reported history of psychiatric or neurological disorders. Controls were not allowed to score above the cut-off on either the Autism Spectrum Quotient (AQ) or Social Responsiveness Scale – Adult version (SRS-A) ( $\geq 32$  on the AQ, T-score  $\geq 61$  on the SRS-A). All participants gave written informed consent prior to the study and were compensated financially for their participation. The study was approved by the local faculty ethics committee of Ghent University (protocol number 2015/58bis).

All participants with ASD had received an official clinical diagnosis prior to the experiment. This diagnosis was verified with the Autism Diagnostic Observation Schedule (ADOS-2) (Lord et al., 2012), Module 4 by a trained psychologist. The revised algorithm for Module 4 of the ADOS-2 (Hus & Lord, 2014) was implemented. In our final sample,



participants scored 6.9 on the ‘Social Affect’ scale on average (cut-off = 6), and 3.9 on the ‘Restricted repetitive behaviors’ scale (cut-off = 2). Seven participants scored below the cut-off of 8 points of the ADOS total score (between 4 and 7). This is not uncommon for adults with high-functioning autism (Deschrijver, Bardi, Wiersema, & Brass, 2015; Zwickel, White, Coniston, Senju, & Frith, 2011). Because excluding these participants did not significantly alter our main findings, we chose to include all ASD participants in our analyses, in line with these previous studies.

One participant in the ASD group failed to correctly respond to the targets, and for 1 participant in the control group, due to a technical error, data for half of the task were lost. Additionally, EEG data for 2 participants in the ASD group and 2 participants in the control group showed too many artifacts. Final data analysis was thus carried out on 21 participants in the ASD group (14 men), and 21 participants in the neurotypical group (14 men). See Table 1 for an overview of the demographics of the final participant sample, and Supplementary Table 1 for the data of each individual. Neither age ( $t(40) = -0.73, p = .47$ ; age range ASD 24-43, control 20-48), nor gender ratio (Pearson chi-square  $< 0.01, p > .99$ ) differed significantly between groups.

A four-subtest short-form (KAUFMAN2) of the Wechsler Adult Intelligence Scale, 3<sup>rd</sup> ed. (WAIS-III) (Grégoire & Wierzbicki, 2009), which has good predictive accuracy of total test scores in individuals with high-functioning ASD (Minschew, Turner, & Goldstein, 2005), was administered for participants that had not received a full WAIS test in the past five years (3 participants with ASD had already received this). IQ was in the normal range for both groups (ASD: 82-131, control: 87-128), and did not differ significantly between groups ( $t(40) = -0.22, p = .83$ ).

## **Questionnaires**

All participants filled out Dutch versions of the AQ and the SRS-A. The AQ (Baron-Cohen, Wheelwright, Skinner, Martin, & Clubley, 2001; Hoekstra, Bartels, Cath, & Boomsma, 2008) is a 50-item self-report screening questionnaire for adults, quantifying the degree to which one has traits associated with ASD. The SRS-A (Constantino & Gruber, 2002; Noens, De la Marche, & Scholte, 2012) is a 64-item questionnaire measuring different behavioral dimensions characteristic of ASD.

### **Auditory oddball paradigm**

Participants carried out an auditory oddball paradigm, consisting of five different auditory stimuli: the standard sound (66%, 198 trials), the target sound (4%, 12 trials) and three names presented as equally infrequent deviants: participants' own name (10%, 30 trials), the name of a close other (10%, 30 trials), and the name of another person unknown to them (10%, 30 trials). These 300 trials were presented in two blocks of 150 trials with a short break in between. All stimuli were presented binaurally through EEG-compatible insert earphones (ER-3C, MedCat), using Presentation software (version 16.5), with a silent inter-stimulus interval (ISI) that was jittered between 1075 and 1425 ms (steps of 25 ms) with an average ISI of 1250 ms. Stimulus presentation was semi-random: each non-standard stimulus was followed by at least one standard sound. Participants were instructed to respond to the target sound as fast and accurately as possible, by pressing the space bar. They were told that there would also be other auditory stimuli, but that these were task-irrelevant.

The standard stimulus was a 1000-Hz tone lasting 500 ms. The target stimulus was a modified square wave of 35 Hz (228 ms). Name stimuli were first names only, lasted between 515 and 700 ms, and were uttered by the same female voice and normalized to have the same maximum volume. The first names of close and unknown other were selected based on a short form participants filled out beforehand. For the 'close other' condition, participants were

asked to indicate the first name of a person close to them (e.g., a family member, partner or good friend). For the ‘unknown other’ condition, participants had to indicate on a list with ten Dutch first names (five male, five female) the names that they could associate with someone they knew. One of the remaining names was assigned to the ‘unknown other’ condition. All unknown names were two syllables long. On average, the own and close other names were two syllables long as well (range 1 – 3), and this did not differ between groups (own name:  $t(40) = 0.76$ ,  $p = .45$ ; close other name:  $t(40) = -0.24$ ,  $p = .81$ ).

## **Procedure**

Participants started with the execution of another, unrelated task wherein no names were presented, which will be reported elsewhere. After a break, participants performed the oddball task, while EEG activity was being recorded. Afterwards, they filled in the questionnaires. For neurotypical participants, this was followed by administration of the WAIS-III short-form test. ASD participants carried out the ADOS-2 and the WAIS-III short-form in a separate session.

## **EEG recording and analyses**

EEG activity was recorded (1024 Hz sampling rate) with an ActiveTwo EEG amp (BioSemi) from 64 active electrodes placed according to the 10-20 international system. Additional electrodes were placed above and below the participant’s left eye, at the external canthi of the eyes, and on both mastoids. Offline, data were re-referenced to the average of the left and right mastoids, and filtered with a 0.5 Hz (12 dB/oct) high-pass filter, a 30 Hz (12 dB/oct) low-pass filter, and a 50 Hz notch filter, using Brain Vision Analyzer (BVA) software (version 2.1.0, Brain Products, Gilching, Germany).

EEG was segmented in epochs of 1000 ms, time-locked to the onset of the name. A pre-stimulus baseline of 100 ms was applied. Data were corrected for ocular artifacts with the ‘Gratton & Coles’ method for ocular correction as implemented in BVA. Subsequently, segments were rejected if amplitude exceeded  $\pm 100 \mu\text{V}$ . Bad electrode channels were interpolated using a spherical spline procedure (order of splines: 4). The remaining segments were then averaged and baseline-corrected per condition, per participant. This resulted in an average of 29 own name trials, 29 close other trials, and 29 unknown other trials. There was no group difference in number of trials (own name:  $t(40) = -0.53$ ,  $p = .60$ ; close other:  $t(40) = -0.10$ ,  $p = .92$ ; unknown other:  $t(40) = -0.32$ ,  $p = .75$ ).

The ERPs showed a clear N1 followed by a positive complex (see Supplementary Figures 1 and 2). Definition and analyses of the components were based on earlier studies with a similar paradigm (Eichenlaub et al., 2012; Holeckova et al., 2006) and visual inspection of the scalp topography across conditions (see Supplementary Figure 1). See Supplementary Figure 2 for the grand average ERPs per group. An N1 with a central distribution was observed. N1 amplitude was defined as the mean activity between 130 and 210 ms post-stimulus-onset. Within the positive complex, 3 distinct components could be identified based on their unique stable topographies, in accordance with earlier research (Eichenlaub et al., 2012; Holeckova et al., 2006). First, a fronto-central deflection was apparent, which we refer to as the early novelty P3 (early nP3). The early nP3 amplitude was defined as the mean activity between 290-350 ms. This was followed by a stable topography with both frontal and parietal components, referred to as the late nP3 (mean activity between 380-440 ms). Finally, the data showed a late parietal positivity (PP), which was quantified as the mean activity between 500 and 800 ms.

Repeated-measures ANOVAs were performed. These always included Group as a between-subjects factor, a within-subjects factor of Name (own name, close other, unknown

other), and within-subjects factors for the electrodes that were chosen for each of the components. For the PP, the factor Electrode included the P3, Pz and P4 electrodes. The N1 was analyzed with a factor Electrode (C3, Cz, C4). The early nP3 was analyzed over electrodes FC3, FCz and FC4. Finally, the late nP3, given its fronto-parietal topography, was analyzed using a lateral and a sagittal factor, over nine electrodes: F3, Fz, F4/C3, Cz, C4/P3, Pz, P4. Effects were adjusted for sphericity violations if required (Greenhouse-Geisser). Bonferroni correction was applied to post-hoc analyses on significant main effects of name or interaction effects between name and group. Effect sizes were reported, namely partial eta squared ( $\eta_p^2$ ) for the ANOVAs (0.01 = small, 0.06 = medium, 0.14 = large effect) and Cohen's d (0.2 = small, 0.5 = medium, 0.8 = large effect) for the t-tests (Cohen, 1988).

### **Source localization**

In case of a Name x Group interaction for PP, the neural sources underlying this effect were estimated with standardized low-resolution brain electromagnetic tomography (sLORETA) (for details see Pascual-Marqui, 2002). sLORETA is based on the neurophysiological assumption of coherent coactivation of neighboring cortical areas (known to have highly synchronized activity) and computes the “smoothest” of all possible activity distributions, to deal with the inverse solution problem and to limit the number of possible solutions. Because of our a-priori predictions, a Bonferroni correction was considered too stringent; the level of significance for all analyses was therefore set to  $p < .01$ , to control for false positives (see also Paul, Walentowska, Bakic, Dondaine, & Pourtois, 2016).

## Results

### Behavioral results

Accurate responses within 1,000 ms were considered correct. Accuracy was very high for both groups and did not differ between groups (ASD group: 99.2%; control group: 99.2%;  $t(40) = 0.00$ ,  $p > .99$ ,  $d < 0.01$ ). Average target RT in the ASD group was 510 ms, and 457 ms in the control group. RT did not differ significantly between groups, although there was a trend toward slower responding in the ASD group ( $t(40) = -1.99$ ,  $p = .05$ ,  $d = 0.63$ ).

### ERP data

**PP.** Grand average waveforms and topoplots are displayed in Figure 1. Analyses revealed both a significant main effect of Electrode ( $F(2, 80) = 7.68$ ,  $p < .01$ ,  $\eta_p^2 = 0.16$ ) and of Name ( $F(2, 80) = 5.73$ ,  $p < .01$ ,  $\eta_p^2 = 0.13$ ), and crucially also a significant Group by Name effect ( $F(2, 80) = 4.59$ ,  $p = .01$ ,  $\eta_p^2 = 0.10$ ). The main effect of Group was not significant ( $F(1, 40) = 0.09$ ,  $p = .77$ ,  $\eta_p^2 < 0.01$ ), nor were any of the other effects (all  $p > .63$ ). PP amplitude was larger at Pz and P4 than at P3 ( $p < .001$ ,  $d = 1.50$ , and  $p = .01$ ,  $d = 0.83$ , respectively), and for own names versus close other ( $p = .01$ ,  $d = 0.80$ ) and unknown other names ( $p < .01$ ,  $d = 0.88$ ). Post-hoc comparisons for the Group x Name interaction showed that the difference between own and both close and unknown other name was significant in the control group ( $p < .01$ ,  $d = 1.55$ , and  $p < .01$ ,  $d = 1.43$ , respectively), whereas this PP enhancement for own name was lacking in the ASD group, with no difference between the own name and close or unknown other name condition ( $p = .93$ ,  $d = 0.04$ , and  $p = .60$ ,  $d = 0.24$ , respectively). The difference between close and unknown other

was neither significant in the control group ( $p = .73$ ,  $d = 0.16$ ), nor in the ASD group ( $p = .61$ ,  $d = 0.23$ ).

**PP: Source localization.** By means of sLORETA, we estimated the underlying sources of the significant Group x Name interaction for the PP (500-800 ms). Figure 2 depicts the group differences in activation for the contrast own name - close other name. Adults with ASD showed less activation for this contrast in the right TPJ (BA 40; peak MNI coordinates:  $x = 65$ ,  $y = -40$ ,  $z = 30$ ;  $t(40) = 2.86$ ,  $p = .007$ ; red/orange). Increased activity in adults with ASD was found in the right IFG (BA 9; peak MNI coordinates:  $x = 50$ ,  $y = 10$ ,  $z = 30$ ;  $t(40) = -3.17$ ,  $p = .003$ ; blue), and adjacent voxels in the right middle frontal gyrus (BA 9; peak MNI coordinates:  $x = 50$ ,  $y = 15$ ,  $z = 30$ ;  $t(40) = -3.01$ ,  $p = .005$ ).

**PP: Link with ASD symptoms.** The difference (own – close other) in PP amplitude (averaged over P3, Pz and P4) did not significantly correlate with ADOS scores within the ASD group, or with AQ or SRS scores either within or across groups (the latter being examined across all participants with the effect of group partialled out to control for main group differences) (all  $p$ -values  $> .1$ ).

**N1.** There was a significant main effect of Name ( $F(2, 80) = 4.09$ ,  $p = .02$ ,  $\eta_p^2 = 0.09$ ), with larger N1 amplitudes for own and close other name than for the unknown name condition ( $p = .01$ ,  $d = 0.82$ , and  $p = .02$ ,  $d = 0.72$ , respectively; but note that this second comparison did not survive Bonferroni correction), indicative of a familiarity effect. N1 amplitude was not different between own name and close other ( $p = .93$ ,  $d = 0.03$ ). Groups did not differ with respect to this, as indicated by a non-significant Group x Name effect ( $F(2, 80) = 1.45$ ,  $p = .24$ ,  $\eta_p^2 = 0.04$ ) and a non-significant Group x Name x Electrode effect ( $F(4, 160) = 0.77$ ,  $p = .57$ ,  $\eta_p^2 = 0.02$ ). There was a trend toward a main effect of Group ( $F(1, 40) = 4.00$ ,  $p = .05$ ,  $\eta_p^2 = 0.09$ ), pointing toward generally smaller N1 amplitudes in the ASD group. No other effects were significant (all  $p > .11$ ).

**Early nP3.** There was a significant main effect of Electrode ( $F(2, 80) = 37.80, p < .001, \eta_p^2 = 0.49$ ), due to larger amplitudes at FCz than FC3 and FC4 ( $p < .001$  for both). Further, the interaction effect Group by Name was significant ( $F(2, 80) = 3.88, p = .03, \eta_p^2 = 0.09$ ). No other effects were significant (all  $p > .08$ ). Post-hoc comparisons per group revealed no significant differences between either own or close other name and unknown other ( $p = .15, d = 0.67$ , and  $p = .13, d = 0.71$ , respectively) or between own and close other ( $p = .28, d = 0.50$ ) in the control group. In the ASD group, the difference between own or close other name and unknown other was also not significant ( $p = .23, d = 0.56$ , and  $p = .27, d = 0.51$ , respectively). There was, however, a significant difference between own and close other name ( $p = .01, d = 1.28$ ), with amplitudes being greater for the close other name.

**Late nP3.** Analyses revealed only a significant main effect of Laterality ( $F(2, 80) = 26.32, p < .001, \eta_p^2 = 0.40$ ), with largest amplitudes at the midline ( $p < .001$ ). Neither the main effect of Name was significant ( $F(2, 80) = 2.28, p = .11, \eta_p^2 = 0.05$ ), nor were any of the other main or interaction effects (all  $p > .17$ ).



## Discussion

We investigated for the first time neural responding to hearing one's own name versus other names in adults with ASD. We found an increase in PP amplitudes specifically for the own name in neurotypicals, which was absent in adults with ASD. This effect was related to attenuated rTPJ activation in ASD. At the N1, a familiarity effect was present, which did not differ significantly between groups. We will now discuss our findings in more detail.

In line with previous findings (Eichenlaub et al., 2012; Folmer & Yingling, 1997; Perrin et al., 2005, 2006, 1999; Tateuchi et al., 2012), ERPs showed a late positive parietal deflection, referred to as PP (Eichenlaub et al., 2012), being specifically enhanced for the own name. Crucially, this preferential effect for the own name was only found in the control group, whereas it was completely absent in the ASD group. Since hearing one's own name is considered highly significant as a social cue and personally relevant in all contexts, it is not surprising that the PP was found to be larger for own names in the control group. It is therefore even more striking that adults with ASD, who clearly know their own name, did not show this effect.

Source localization analyses showed that the group difference in own-name processing at the level of the PP had its origin in an increase in activity in the rTPJ for the control group, which was attenuated in the ASD group. Increased rTPJ activity in response to hearing one's own name has been reported previously using fMRI and PET (Carmody & Lewis, 2006; Holeckova et al., 2008; Perrin et al., 2005). The finding of diminished rTPJ activity for hearing one's own name in the ASD group is in accord with our hypothesis of a deficit in self-other distinction, and corresponds to results of earlier studies showing reduced TPJ activity in ASD during tasks requiring self-other distinction (Lombardo et al., 2011; Spengler et al., 2010) and mentalizing (Eddy, 2016; Murdaugh et al., 2014; Pantelis, Byrge, Tyszka, Adolphs,

& Kennedy, 2015). It further adds to the accumulating evidence that the rTPJ is a core region implicated in ASD (Chien et al., 2015; Fishman et al., 2014; Kana et al., 2014).

In contrast, the difference between own and close other name showed more activity in the rIFG for the ASD group. It is obvious that adults with ASD can distinguish their own name from other names, however the results indicate that hearing the own name in ASD does not elicit activation in an area typically associated with a low-level neurocognitive mechanism of self-other distinction (rTPJ), but rather elicits activation in an area typically associated with processing of external salient stimuli (rIFG). There is ample evidence that the rIFG is involved in bottom-up redirecting of attention to external (social as well as non-social) salient stimuli (Corbetta & Shulman, 2002; Hampshire, Chamberlain, Monti, Duncan, & Owen, 2010). Adults with ASD may show increased rIFG activity for their own name, as they have learnt that their own name is an important salient stimulus (more salient than other names), and this may reflect a compensatory mechanism for the affected self-other distinction mechanism at the level of the rTPJ. Of course, this is speculative and hence further research is warranted. No other areas were implicated, adding to the scarce and inconsistent findings on the neural basis of auditory own-name processing to date. This warrants further systematic research on the neural basis of auditory own-name processing, applying different paradigms. Source localization of EEG data evidently comes with limitations, and merits replication of our finding using methods with better spatial resolution, such as fMRI.

Some other findings regarding the earlier ERP components that we explored are worth mentioning. In the N1 time window, a familiarity effect was observed, with greater amplitudes for hearing the own or close other's name than for the unknown other, which was however not different between groups. To the best of our knowledge, this evidence is the first to demonstrate the presence of a familiarity effect on auditory name processing at the N1 level but is in accord with research showing larger N1 amplitudes in response to familiar sounds

(Kirmse, Jacobsen, & Schröger, 2009) and faces (Caharel et al., 2002; Marzi & Viggiano, 2007) as compared to unfamiliar stimuli. N1 amplitudes tended to be overall smaller in the ASD group, as reflected by a marginally significant group effect. Smaller amplitudes of early negative ERPs, including the mismatch negativity, in response to auditory speech and non-speech stimuli have been reported previously in children with ASD (Dunn, Gomes, & Gravel, 2008; Kuhl, Coffey-Corina, Padden, & Dawson, 2005; Lepistö et al., 2006). This could be taken to suggest that bottom-up regulation of auditory sensory input is less effective in individuals with ASD (Bomba & Pang, 2004; Haesen, Boets, & Wagemans, 2011). It is important to note however that this marginal group difference for the N1 was found independent of name condition and that additional explorative correlational and covariance analyses did not reveal an association between the N1 group effect and our main finding for the PP. Still, it would be an interesting line of future research to investigate the relationship between basic auditory processing and own-name processing deficits in ASD.

The oddball paradigm we used also allowed the investigation of orienting processes, as reflected in the nP3, often found enlarged for salient stimuli that more easily capture attention (Debener et al., 2005; Friedman et al., 2001; Nieuwenhuis, De Geus, & Aston-Jones, 2011). Previous findings for this component with regard to name processing are, however, scarce and inconclusive. Tateuchi et al. (2012) found larger nP3 amplitudes for own versus close other's names, while Eichenlaub et al. (2012) did not observe such an increase. In the current study, we also did not find enhanced nP3 amplitudes for the own name in neurotypicals or adults with ASD. We did however find an unexpected interaction effect between name and group at the early nP3, with larger amplitudes for close other than for own names in the ASD group only. This finding suggests atypical stronger orienting to familiar names than own names in ASD, but should be interpreted with caution as it was not hypothesized, and warrants further investigation.

While we cannot compare our findings with other studies that have evaluated ERPs elicited by hearing the own name in ASD because our study is the first to investigate this phenomenon in ASD, it is worth comparing our results to the findings of two recent studies investigating ERPs in response to seeing the own name in ASD (Cygan et al., 2014; Nowicka et al., 2016). These studies reported, similar to what we found, enlarged amplitudes of a late parietal positivity (P300) for the own name versus other names in controls, and a lack of this modulation in individuals with ASD. Importantly, all three studies showed significant group differences for the crucial comparison between own name and close-other name, which is the purest index of self-other distinction as it controls for familiarity or personal relevance. There is however also a difference in findings: in contrast to Cygan et al. (2014) and Nowicka et al. (2016), we did not find an effect of familiarity at the PP in ASD. In their study, adults with ASD showed larger P3 amplitudes for familiar than for unfamiliar names, while in our study none of the name conditions differed significantly from each other in the ASD group. This may be due to differences in task characteristics. We used auditory stimuli, and presented names as task-irrelevant stimuli to ensure that effects were due to the inherent salience of the names capturing attention, not to be confounded by potential differences in task-relevant processing. In contrast, in their studies names were visually presented and task-relevant (i.e., required a response). Furthermore, although indicated as unfamiliar by the participant, we cannot exclude the possibility that participants were still somewhat familiar with the name presented to them in the ‘unknown other’ condition, as they might have come across this name on television or elsewhere. Future research is needed to further investigate potential reasons for this difference between studies.

Still, the results of both the visual name processing studies and our study show that the own name is a highly self-relevant stimulus irrespective of modality, and may indicate that ASD is associated with altered self-referential processing and a self-other distinction deficit

across modalities. This suggestion is strengthened by a recent finding from our lab, in which self-other distinction in ASD was studied in the tactile modality (Deschrijver et al., 2016), and showed a diminished effect in ASD at the P3 level as well. Future research is warranted on self-other distinction in ASD, using different self-related stimuli and across different sensory modalities.

The current study had some limitations. Firstly, seven adults in our ASD group did not score above the cut-off of the ADOS-2. Importantly however, excluding these participants from our analyses did not change our main findings regarding the PP. Secondly, one could argue that the adults with ASD may not have been engaged in the task. However, names were presented as task-irrelevant stimuli and adults with and without ASD were equally highly accurate in detection of the target sounds, suggesting no overall differences in task engagement. Thirdly, we did not match close names or unknown other names for gender, but it seems highly unlikely that this should have had an effect on any of our main findings. Finally, as deficits in ASD are not limited to own-name processing but entail impairments in processing of other social stimuli and basic auditory processing as well, future research is warranted to further investigate the link between these impairments in ASD.

Altogether, the current study provides evidence for altered neural processing in response to hearing one's own name in high-functioning adults with ASD, thus extending the findings of Cygan et al. (2014) and Nowicka et al. (2016) to the auditory domain. Our results contribute to recent theories arguing that social cognition problems in ASD may be caused by a specific deficit in self-other distinction (De Guzman et al., 2015; Sowden & Shah, 2014), in which the rTPJ seems to play an important role.

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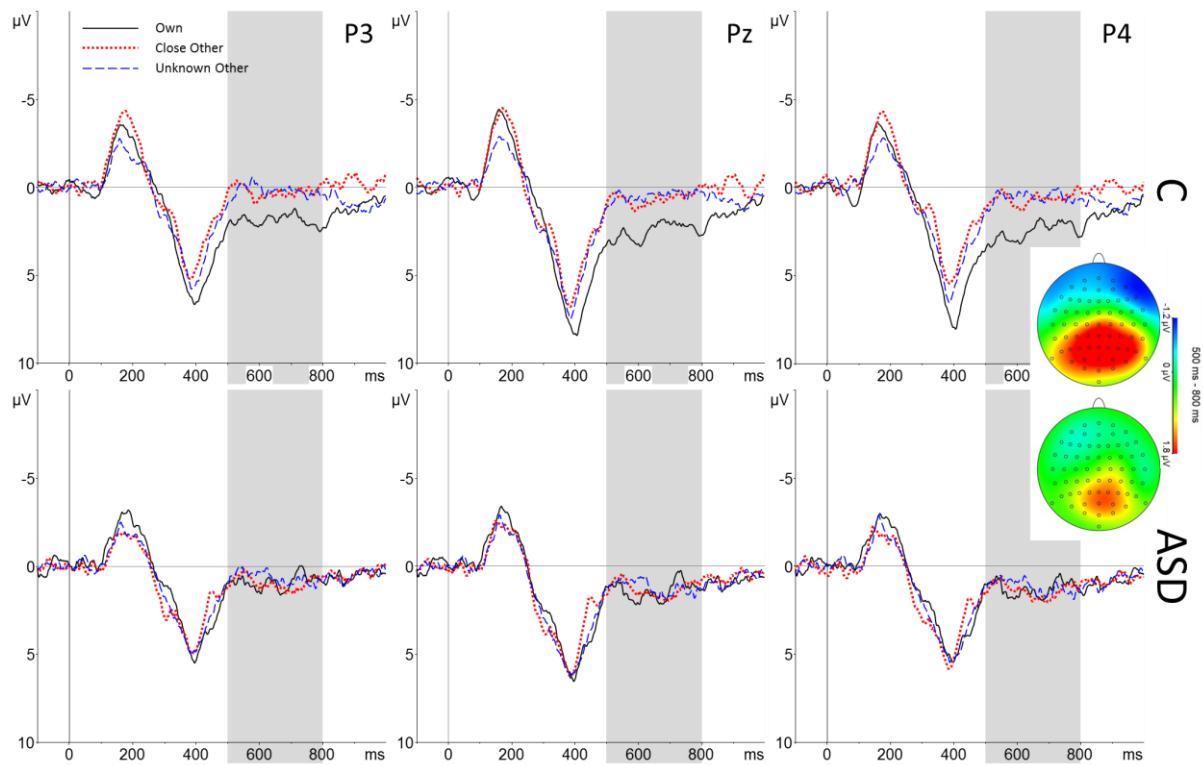
Table 1

*Participants' characteristics (M = mean, SD = standard deviation) for both groups*

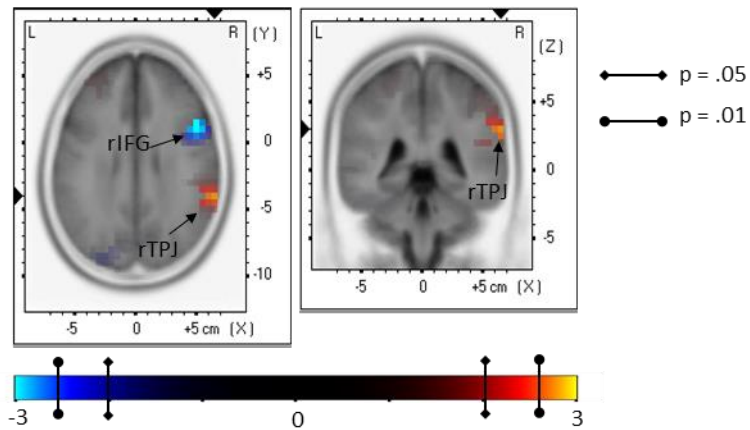
	ASD, N = 21	Neurotypicals, N = 21
	M (SD)	M (SD)
Age	33.0 (6.5)	31.3 (8.3)
IQ score	109.3 (13.4)	108.5 (10.6)
AQ score*	37.7 (7.0)	15.5 (5.0)
SRS-A T-score*	80.1 (10.1)	49.3 (6.3)

*Note.* IQ = score on the Wechsler Adult Intelligence Scale IV. AQ = Autism Spectrum Quotient. SRS-A = Social Responsiveness Scale, Adult Version.

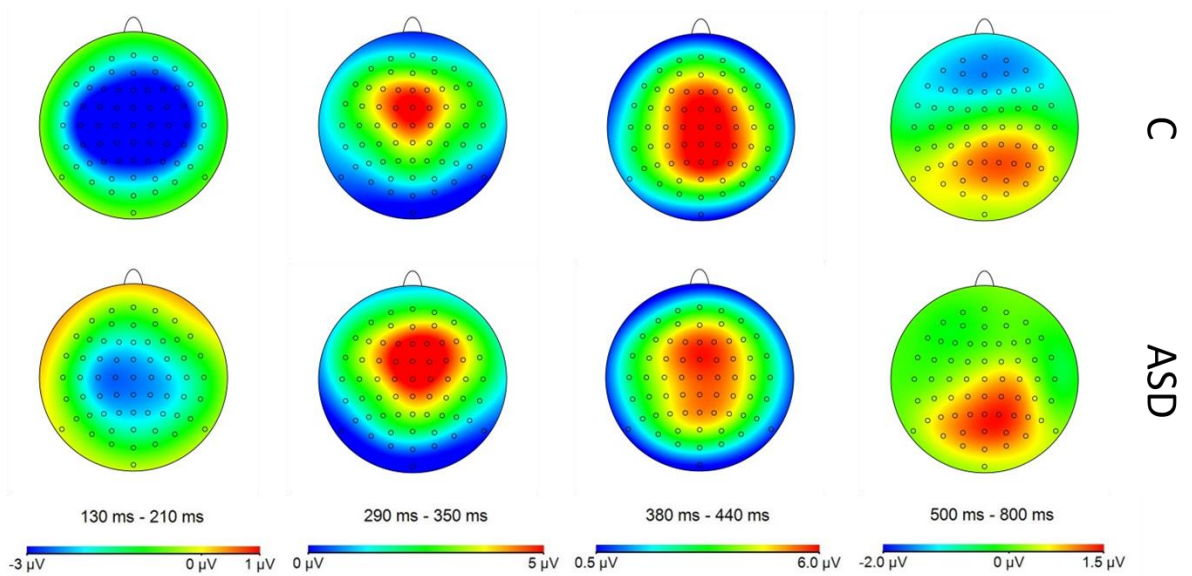
\*: groups differ from each other at  $p < .001$



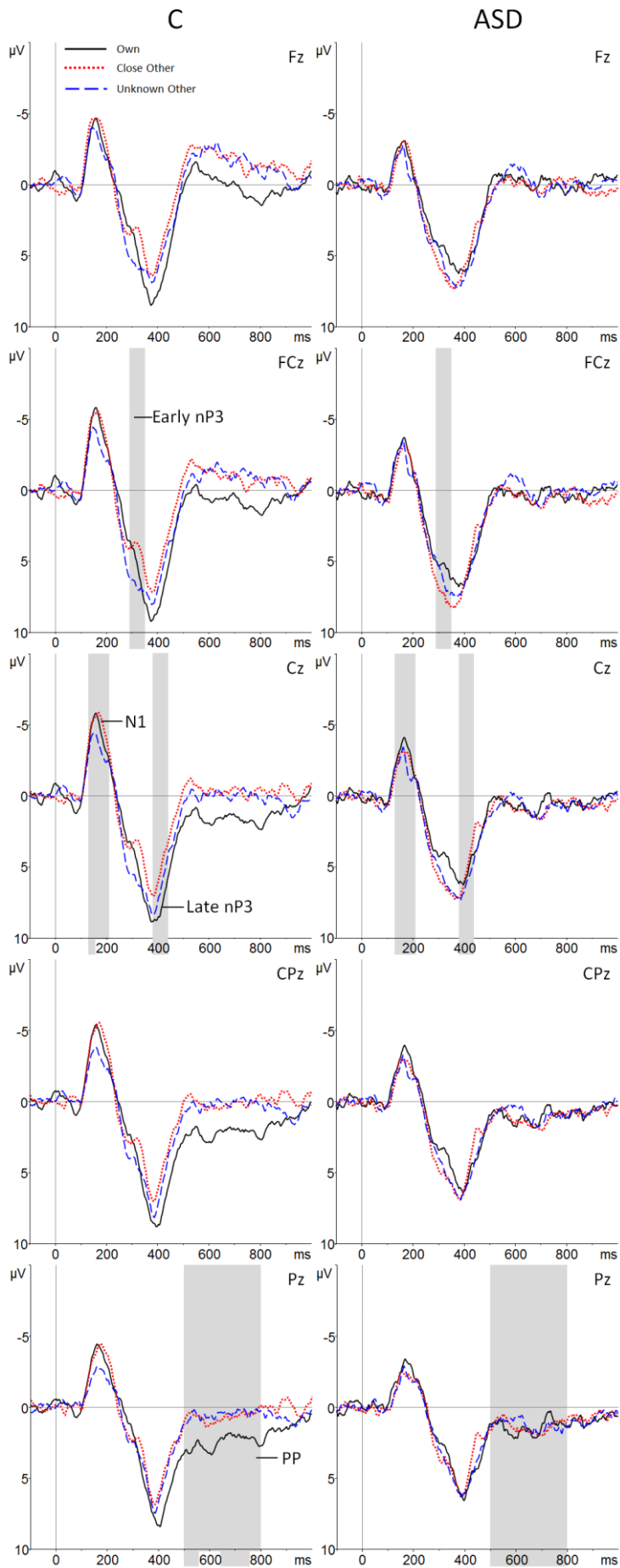
**Figure 1.** Grand average waveforms for the three electrodes (P3, Pz and P4) included in the PP analysis, as well as the topography for the PP time window (500 – 800 ms) for the own name specifically. Top: control group. Bottom: ASD group. See the online article for the color version of this figure.



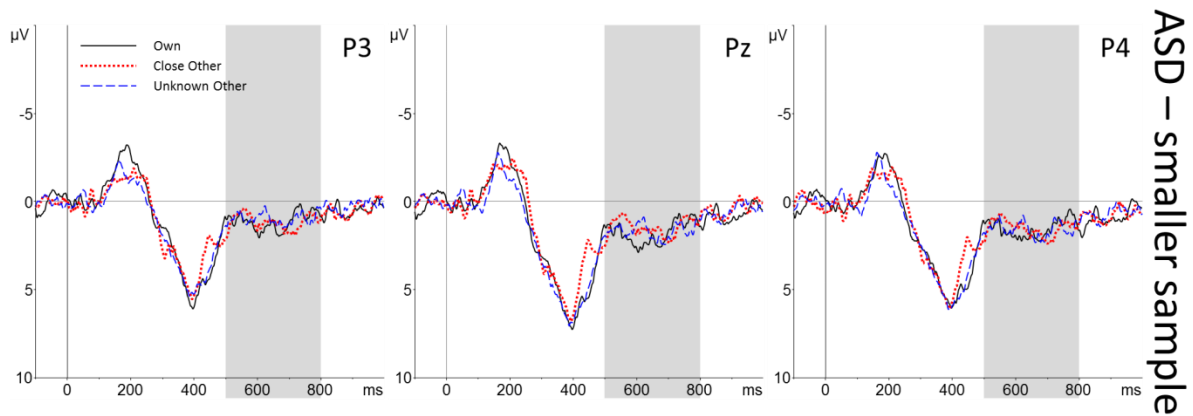
**Figure 2.** Results of the sLORETA source localization analysis of the interaction between Name (Own > Close Other) and Group (Controls > ASD) for the parietal positivity (500 – 800 ms), indicating the two main sources: right temporo-parietal junction (rTPJ) and right inferior frontal gyrus (rIFG). Red/yellow: Controls > ASD. Blue: ASD > Controls. See the online article for the color version of this figure.



*Supplementary Figure 1.* Scalp topography, displayed for all three name conditions averaged together. Upper row: control group (C), lower row: ASD group. From left to right: N1: 130 – 210 ms. Early nP3: 290 – 350 ms. Late nP3: 380 – 440 ms. Parietal positivity (PP): 500 – 800 ms. See the online article for the color version of this figure.



*Supplementary Figure 2.* Grand average waveforms for own (black), close other (red) and unknown other (blue) names, displayed for the five midline electrodes Fz, FCz, Cz, CPz and Pz. The four relevant ERP components are indicated in light-grey. Left: neurotypical control group, right: ASD group. See the online article for the color version of this figure.



*Supplementary Figure 3.* Grand average waveforms for the three electrodes (P3, Pz and P4) included in the PP analysis, for the ASD participants that had an ADOS score above the cut-off ( $N = 15$ ). Analyses revealed both a significant main effect of Electrode ( $F(2, 68) = 7.24, p < .01, \eta_p^2 = 0.18$ ) and of Name ( $F(2, 68) = 4.24, p = .02, \eta_p^2 = 0.12$ ), and crucially also a significant Group by Name effect ( $F(2, 68) = 3.97, p = .02, \eta_p^2 = 0.11$ ). The main effect of Group was not significant ( $F(1, 34) = 0.46, p = .50, \eta_p^2 = 0.01$ ). PP amplitude was larger at Pz and P4 than at P3 ( $p < .001, d = 1.57$ , and  $p = .02, d = 0.81$ , respectively), and for own names versus close other ( $p = .01, d = 0.97$ ) and unknown other names ( $p = .01, d = 0.88$ ). Post-hoc comparisons for the Group  $\times$  Name interaction showed that the difference between own and both close and unknown other name was significant in the control group ( $p < .01, d = 1.55$ , and  $p < .01, d = 1.43$ , respectively), whereas this PP enhancement for own name was lacking in the ASD group, with no difference between the own name and close or unknown other name condition ( $p = .94, d = 0.04$ , and  $p = .95, d = 0.03$ , respectively). The difference between close and unknown other was neither significant in the control group ( $p = .73, d = 0.16$ ), nor in the ASD group ( $p = .99, d < 0.01$ ).

Supplementary Table 1

*Individual demographics and questionnaire data per participant*

Ppt	Age	Gender	IQ	AQ	SRS-A	ADOS
ASD1	32	F	85	25	64	15
ASD2	40	F	124	46	94	19
ASD3	24	F	92	42	88	8
ASD4	41	M	103	46	101	14
ASD5	43	M	116	39	81	14
ASD6	24	M	115	34	78	10
ASD7	35	F	120	40	84	6
ASD8	24	M	123	32	74	3
ASD9	33	M	82	38	86	9
ASD10	34	M	131	41	70	12
ASD11	28	F	122	28	72	5
ASD12	33	F	128	45	88	10
ASD13	31	M	113	34	72	5
ASD14	24	M	104	40	72	21
ASD15	43	M	105	48	96	12
ASD16	31	M	97	21	66	10
ASD17	29	M	105	42	74	7
ASD18	40	M	112	35	81	10
ASD19	27	M	112	37	77	5
ASD20	36	F	99	37	74	17
ASD21	41	M	108	42	89	4
C1	31	F	128	10	48	
C2	31	M	105	16	60	
C3	25	F	116	26	53	
C4	26	M	115	14	51	
C5	26	F	110	14	53	
C6	20	F	119	19	45	
C7	36	F	90	19	59	
C8	43	M	112	11	44	
C9	28	M	100	7	50	
C10	25	F	126	9	35	
C11	45	M	102	16	54	
C12	32	M	87	15	51	
C13	48	F	109	12	42	
C14	26	M	95	16	51	
C15	26	M	107	17	47	
C16	25	M	110	23	57	
C17	25	M	100	19	51	
C18	25	M	105	12	47	
C19	30	M	117	11	38	
C20	47	M	109	24	52	
C21	38	M	117	16	48	



*Note.* IQ = score on the Wechsler Adult Intelligence Scale IV. AQ = score on the Autism Spectrum Quotient. SRS-A = T-score on the Social Responsiveness Scale for Adults. ADOS = score on the Autism Diagnostic Observation Schedule 2, Module 4.