Neural correlates of own name and own face processing in neurotypical adults scoring low versus high on symptomatology of autism spectrum disorder

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Abstract

Previous event-related potential (ERP) research showed reduced self-referential processing in autism spectrum disorder (ASD). As different self-related stimuli were studied in isolation, it is unclear whether findings can be ascribed to a common underlying mechanism. Further, it is unknown whether altered self-referential processing is also evident in neurotypicals scoring high on ASD symptomatology. We compared ERPs in response to one's own name and face (versus other names/faces) between neurotypical adults scoring high versus low on ASD symptomatology. Conform previous research, the parietal P3 was enhanced, both for own name and face, indicating a self-referential effect. The N250 was only enhanced for one's own face. However, the self-referential parietal P3 effect did not correlate between the names and faces conditions, arguing against a common underlying mechanism. No group effects appeared, neither for names nor faces, suggesting that reduced self-referential processing is not a dimensional ASD feature in the neurotypical population.

Keywords: Own name, Own face, Self, Event-related potentials (ERPs), Autism Spectrum Disorder

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People with autism spectrum disorder (ASD) show deficiencies in social interaction and communication (American Psychiatric Association, 2013). To understand these deficiencies, research has predominantly focused on interpersonal processes such as mentalizing about others (i.e. the ability to attribute mental states such as believes and intentions to others). However, over the past two decades there has been an increased interest in self-related processes in ASD (Huang et al., 2017; Lombardo et al., 2007; Nijhof & Bird, 2019; Uddin, 2011). While investigating self-related processes to better our understanding of the social difficulties in ASD may seem counter-intuitive at first glance, this is not the case when we consider that, by definition, 'to interact socially' means interaction between *oneself* and others. Lombardo and Baron-Cohen (2011) for example describe how one may aid mentalizing about others by simulation, that is, putting oneself in someone else's shoes to simulate the experience of someone else within themselves. Hence, atypical self-related processing (i.e. self-referential processing) influences social interaction (Nijhof & Bird, 2019).

Self-related stimuli typically have a preferential status: they are remembered better (Symons & Johnson, 1997), recognized faster (Sui et al., 2012), and they can involuntarily capture attention (Alexopoulos et al., 2012). This self-referential effect has been reported to be weaker in ASD (Lombardo & Baron-Cohen, 2010; Uddin, 2011). When investigating self-referential processing, researchers have often used event-related potentials (ERPs) to look at neural activity, which can reveal underlying cognitive processes. This enables the study of self-related stimuli processing without relying on overt responses. ERP studies have shown that the P3, more specifically the later parietal part of the P3 complex, is strongly modulated by self-related stimuli including one's own name and face, which are arguably the most

salient self-related stimuli we encounter (Name: e.g. Folmer & Yingling, 1997; Kotlewska & Nowicka, 2015; Tacikowski et al., 2011; Tacikowski & Nowicka, 2010; Face: e.g. Sui et al., 2006; Caharel et al., 2002; Ninomiya et al., 1998; Scott et al., 2005). This parietal positive deflection occurs 300 ms or later after stimulus onset over central-parietal scalp sites and is considered to reflect the amount of attention allocated to the stimulus presented (Polich, 2007). Note that different studies that showed self-referential effects for self-related stimuli refer to this component in different ways: some use P300 (Tacikowski et al., 2011; Tacikowski & Nowicka, 2010), others Parietal Positivity (Eichenlaub et al., 2012; Nijhof et al., 2018), and again others P3b (Doradzinska et al., 2020). As the term P3b or P300 might be more fitted for classical oddball tasks, we will use the term parietal P3 in our paper.

So far, three studies have investigated the neural response to one's own name versus other names in adults with ASD (Cygan et al., 2014; Nijhof et al., 2018; Nowicka et al., 2016). Each of the studies found an enhanced parietal P3 response for one's own name versus a close-other name in the neurotypical (NT) control group while this effect was absent in the ASD group. In other words, the ASD groups did not show a self-referential effect for one's own name. The first study by (Cygan et al., 2014) employed a simple detection task targeting bottom-up attention with visual stimuli in which participants had to respond every time a name was presented. The same lab conducted a second study with visual name stimuli (Nowicka et al., 2016) that employed a speeded two-choice recognition task (familiar versus unfamiliar), targeting top-down attention. A third study conducted by Nijhof et al. (2018) looked at the neural correlates of auditory-presented names using a paradigm in which the stimuli were task-irrelevant and presented equally infrequently (as previously used in NT samples: Eichenlaub et al., 2012; Holeckova et al., 2006). This paradigm ensured that the results were not confounded by task relevant processes, instead being solely due to the inherent saliency of the names. All studies included an own name, a close-other name and an

unfamiliar/unknown name condition to control for familiarity effects. Despite the limited number of studies, the findings of these three studies suggest a diminished self-referential effect in adults with ASD. The effect was found to be independent of modality (visually or auditory) and task-relevance of the stimuli presentation. These own-name results in adults with ASD are particularly interesting as a diminished orienting response to one's own name in very young children is one of the earliest and strongest predictors of ASD (e.g. Werner et al., 2000). The diminished response to one's own name thus appears to persist into adulthood on a neural level even in participants that clearly know their own name (i.e. adults with normal to high IQ and no language difficulties).

Studies of own face processing in ASD are also scarce. To the best of our knowledge only two studies have investigated ERP correlates of own face processing in ASD. In line with own name results, Cygan et al. (2014) found enhanced parietal P3 responses to one's own face (versus a familiar face) in NT adults but no such self-referential effect for one's own face in adults with ASD. These results are consistent with those from a study by Gunji et al. (2009) which also looked at own face processing in children diagnosed with pervasive developmental disorders.

In short, these studies found that adults with ASD showed a diminished or absent enhancement of the parietal P3 amplitude in response to one's own name and face, suggesting general difficulties with self-referential processing. However, the number of studies is low, warranting replication. Furthermore, a limitation of existing work on self-referential processing in ASD is that different self-related stimuli such as one's own names and face have mainly been studied in isolation (as also noted by Nijhof & Bird, 2019). While both one's name and face refer to the concept of self, the self is an abstract multimodal concept and there is an ongoing debate about the degree of abstraction of self-representation mechanisms in the brain (Kaplan et al., 2008). Diminished self-related processing of names and faces in ASD

may reflect difficulties in a shared underlying mechanism. Alternatively, it may reflect alteration of distinct cognitive processes, as own names and faces might differ in crucial aspects from one another.

One distinction is that one's own face has been argued to refer to the neural representation of the physical self, while one's own name refers to the non-physical or psychological aspect of self (Lombardo & Baron-Cohen, 2010). This reasoning led Cygan et al. (2014) to test the hypothesis put forward by Uddin (2011) that the physical self would be less affected than the psychological self in people with ASD. As aforementioned, Cygan et al. (2014) found that both own name and face processing were atypical in ASD, casting doubts on Uddin's hypothesis. However, these findings cannot address the pertaining question of whether one common underlying mechanism caused the altered processing of self-related stimuli in ASD. A direct comparison (correlation) of the self-referential P3 effects for own name and face could shed light on this.

One's own name also differs from one's own face in another important way. While one's own face is a pure self-referential stimulus, one's own name derives its saliency not only from its self-referential nature, but also from its potential social communicative (ostensive) function. One's own name, especially when heard, typically acts as a social cue to initiate social communication, which is not the case for one's own face. In the current study, we therefore aim to directly compare (correlate) neural correlates of self-referential processing of one's own name and face with names presented auditorily.

Furthermore, no studies so far have investigated the neural response to one's own name or face in a NT population who score high (versus low) on ASD symptomatology. Investigating associated features of ASD in the NT population may be valuable to help identify potential dimensional and categorical specifiers of ASD. The dimensional view of ASD highlights the concept of autistic traits and associated features that run throughout the

whole population, with the clinically diagnosed ASD subpopulation at the extreme end of the spectrum. Indeed, Abu-Akel et al. (2019) investigated the continuum nature of the distribution of autistic traits and found support for a complementary role of both categorical and dimensional approaches to ASD. Given that we still ultimately rely on categorical diagnoses of ASD even though the spectrum extends into the general population, dismissing the relationship between ASD and associated autistic traits/features in the general population will lead to the loss of potentially valuable findings. Research in the NT population can hence help to identify which traits/features of ASD are dimensional and which are categorical (i.e. distinguish the NT population from the ASD population).

Current study

In the current study we investigated the neural correlates of *both* own name and face processing in a sample of NTs, comparing adults scoring low versus high on ASD symptomatology. This study may help to determine whether there is a common mechanism underlying the (diminished) self-referential effect of own name and face processing. In addition, we wanted to investigate whether the reduced self-referential effect for one's own name and face is a specific, categorical feature of ASD, or a dimensional one that runs throughout the population.

Participants completed two tasks: one task included name stimuli whereas the other included face stimuli. Based on previous findings, we expected a greater parietal P3 response for one's own name and face compared to the close-other name and face in the low-scoring group, indicating a self-referential effect, and this effect to be diminished or absent in the high-scoring group. Furthermore, if the processing of one's own name and face is (in part) driven by the same mechanism, the self-referential effect for one's own name and face are expected to correlate positively with each other.

Although the parietal P3 was the main focus of the current study as this component

has most consistently been shown to reflect self-referential effects and to be sensitive to self-other distinction, we also explored earlier components commonly investigated in own name and face literature, to enrich the field. For the name stimuli we analysed the N1 (a negative waveform occurring between 80 and 120 ms after stimulus onset over fronto-central scalp sites) associated with an involuntary shift of attention. This early component has been found to be modulated by the level of stimuli familiarity, that is, the N1 has been reported to be larger for own-names than for unfamiliar names but not for close-other names (e.g. Höller et al., 2011; Nijhof et al., 2018; Tateuchi et al., 2012). Furthermore, we analysed the P2 (also sometimes referred to as the early novelty P3) associated with an involuntary shift of attention toward unexpected, infrequent and salient stimuli (Friedman et al., 2001; Polich, 2007). Nijhof et al. (2018) found this component to be larger for the close-other name than for one's own-name. For the face stimuli we analysed the N170 (a negative waveform occurring approximately 170 ms after stimulus onset over occipito-temporal scalp sides) associated with structural encoding of faces (e.g. Eimer, 2011; Estudillo, 2012). Although some studies found this early face component to be modulated by familiarity, the majority of studies did not (Estudillo, 2017). The N250 (a negative waveform occurring approximately 250 ms after stimulus onset over occipitotemporal sites) has been argued to be a more reliable index of stimuli familiarity (Tanaka et al., 2006), and is therefore also analysed.

Method

Participants

We included participants that scored low (≤ 2) or high (≥ 5) on the Autism-spectrum quotient-10, a brief sceener for ASD (AQ-10; Allison et al., 2012). These cut-off scores for the AQ-10 were based on a previous study by Nijhof et al. (2017), who showed less spontaneous mentalizing in NT adults scoring low on ASD than those who scored high. Candidates were excluded from participation if they reported any neurological or psychiatric

disorder or if they reported to have facial hair, face piercings or tattoos. They were also excluded if they wore glasses and were less attuned to recognize their face without glasses versus wearing glasses. Candidates with such distinguishing facial features were excluded to ensure that any effects on the task including faces were due to processing of the basic essential facial features (e.g. eyes, nose, mouth, face structure). We recruited candidates via online and printed advertisements distributed around various faculties of Ghent University. An online pre-screening questionnaire with which we assessed the inclusion and exclusion criteria was completed by 594 candidates, 253 of whom met the eligibility criteria and were invited to participate. Of the 253 eligible candidates, 106 candidates scored low and 147 candidates scored high on the AQ-10. Thirty candidates of each group (i.e. low-scoring group and high scoring group) agreed to participate resulting in a final sample of 60 participants. Groups did not differ in age, sex, or years of education (see Table 1). Participants were reimbursed for their time. This study was approved by the local ethics committee of the Faculty of Psychology and Educational Sciences of Ghent University (EC/2018/23).

Table 1
Participant characteristics.

	Low-scoring group *M* (*SD*)	High-scoring group *M* (*SD*)	*F*-value (*p*)
Age	21.90 (2.82)	21.50 (1.46)	0.48 (.493)
Sex (number females)	27	27	N/A
Years of education	15.67 (1.88)	15.77 (1.48)	0.05 (.820)
AQ – total	11.73 (5.70)	19.87 (8.22)	19.58 (< .001)
SRA-A – total	32.03 (17.06)	51.57 (23.47)	13.60 (.001)

note. Low-scoring group: $n = 30$, High-scoring group: $n = 30$; AQ = Autism-spectrum quotient; SRS-A = Social responsiveness scale – adults.

Measurements

Questionnaires

The full AQ (Baron-Cohen et al., 2001) and the social responsiveness scale for adults

(SRA-A; Constantino, 2002), two commonly used questionnaires in ASD research, were administered to check for the reliability of the AQ-10 scores. Across the two groups the mean scores of these questionnaires (AQ: $M = 15.80$, $SD = 8.12$; SRS-A: $M = 41.77$, $SD = 22.58$) were comparable with previous findings in neurotypical populations (AQ: Ruzich et al., 2015; SRS-A: Ingersoll et al., 2011). These mean scores per group were also in line with the findings of Nijhof et al. (2018). In further confirmation of the groups, the AQ and SRS-A scores differed significantly between groups (see Table 1), and both questionnaires significantly correlated with the AQ-10 (AQ: $r = .55$, $p < .001$, SRS-A: $r = .45$, $p < .001$) and each other ($r = .74$, $p < .001$).

Name Task

The auditory name task was identical to the one described by Nijhof et al. (2018). The task consists of five stimuli: The standard sound (66%; 198 trials), the equally infrequent deviant sounds (i.e. 30 trails each/10% each; first name only), namely, the participants' own name, close-other name, and unknown-name, and the target sound (4%; 12 trials). Participants were instructed to press the space bar as quickly and accurately as possible to the target sound. The stimuli were presented randomly, but with the restriction that a non-standard stimulus was always followed by at least one standard sound. The task consisted of two blocks of 150 trials (300 trials in total) with a short break in between. The standard stimulus was a 1000 Hz 500 ms-tone and the target stimulus was a modified square wave of 35 Hz lasting 228 ms. The lengths of the name stimuli were between 515 ms and 700 ms. Stimuli were followed by a silent inter-stimulus interval (ISI; jittered: 1075-1425 ms with steps of 25 ms and an average ISI of 1250 ms) and were presented binaurally through EEG-compatible insert earphones (ER-3C, MedCat). Triggers to mark the timestamp came through just at the onset of the stimuli presentation.

All name stimuli were recorded by the same female native Flemish speaker with a

friendly intonation and were then normalized to have the same maximum volume. We used Audacity (https://www.audacityteam.org/), open-source audio software, to normalize the stimuli, following Nijhof et al. (2018). Participants were asked to fill in a short form prior to the test session day inquiring about the names. For the own name stimuli, we used the name of the participant unless the participant had a more popular nickname. For the close-other name, we used a name of a person that the participant indicated to be close to of the same gender (e.g. family member or close friend). We asked participants to, if possible, indicate a close-other name that has roughly the same number of syllables as their own name. For the unknown-other name, participants indicated from a list of five Flemish names (gender specific) whether they knew someone with that name or not. We chose an unknown name that closely matched the number of syllables in the participants' own name amongst the names they selected.

Face Task

The face task consisted of four stimuli: the faces (36 trials each/30% each), namely, one's face, the close-other face and the unknown face, and the infrequent famous face that served as a target (12 trials; 10%). Participants were instructed to press the space bar as quickly and accurately as possible to the target face. On each trial a fixation cross was presented for 200 ms, followed by a face stimulus for 500 ms. The face stimulus was than replaced by a blank inter-trial interval (ITI; jittered: 1275-1625 ms with steps of 25 ms and an average ITI of 1450). The stimuli were presented randomly, but with the restriction that a target face was never presented on two consecutive trials. The tasks consisted of two blocks of 60 trials (120 trials in total) with a short break in between.

Participants were asked to send images of themselves and their gender-matched close other prior to the test session day. The faces of the unknown and famous persons were taken from the internet. For the famous person we used an image of Emma Watson and Daniel

Radcliffe for female participants and male participants respectively. Before the start of the task we asked the participants whether they knew the identity of the target face. All face images showed individuals facing forward, looking straight into the camera portraying a neutral expression, with mouth closed and without any hair covering the face. All images were cropped to an oval mask with Adobe Photoshop revealing only the basic essential facial features (e.g. eyes, nose and mouth) and thereby removing distinguishing features (e.g. hair style and color, clothing, earring). All face images were confined to an oval shape (5.73º x 8.20º) on the basis of the position of the eyes and mouth and were displayed in grey scale against a black background. Within participants, the face stimuli were matched for mean luminance and contrast using the SHINE toolbox in MATLAB (Willenbockel et al., 2010).

Procedure

Participants were seated in a faraday cage 60 cm from a 24-inch computer monitor. The tasks were presented using Presentation Software (version 18.1; Neurobehavioral Systems, Inc., Berkeley, CA, USA). Before the start of the experiment all participants signed an informed consent and completed the three questionnaires. Subsequently, participants completed the two tasks. The task with which participants started (i.e. name or face task) was counterbalanced across participants. Next, participants completed two other computer tasks. However, as these tasks are not used to answer our research question, they are not reported here any further. The whole experiment lasted around 1.5 hours.

EEG Recordings and Pre-processing

EEG was continuously recorded from 64 scalp sites using an ActiCHamp amplifier (Brain Products, Enschede, The Netherlands) and BrainVisionRecorder software (version 1.21.0304, Brain Products, Gilching, Germany). Ag/AgCI (active) electrodes were mounted in an elastic cap (ActiCAP, Munich, Germany) and positioned according to the extended 10-20 international system. All channels were amplified against Fz. Vertical electro-oculogram

(EOG) was recorded with additional bipolar AG/AgCI sintered ring electrodes placed above and below the left eye. Horizontal EOG was recorded with FT9 and FT10 electrodes embedded in the cap. The sampling rate was 1000 Hz.

Off-line analysis of the EEG signal was performed using BrainVisionAnalyzer software (version 2.1.0, Brain Products, Gilching, Germany). First, we implemented the Butterworth zero phase filters: high-pass – 0.1 Hz, 12 dB/oct; low-pass – 30 Hz, 12 dB/oct; notch filter – 50 Hz. Second, we re-referenced data to the average reference. Third, we corrected ocular artifacts using Infomax Independent Component Analysis on whole data (Mennes et al., 2010). Next, the EEG of correct trials were segmented to obtain epochs extending from 200 ms before to 1000 ms after the stimulus onset. Subsequently semi-automatic artifact rejection was applied (maximum allowed voltage step: 50 μV; minimum and maximum permitted amplitudes: ±100 μV; lowest allowed activity in intervals: 0.5 μV). Electrodes with consistently poor signal quality were however removed and then interpolated using a spherical spline procedure (order of splines: 4). We defined electrode sides as '*bad*' if artifacts rejection would have resulted in ≥ 5% loss of data. No more than four channels were interpolated within a participant. Overall, only 0.26% of the name and 0.55% of the face data (60 participants x 64 channels) were interpolated. Finally, we averaged and baseline-corrected (-200 – 0 ms) the remaining segments per condition, per participant. This resulted in an average of 29.32 (*SD* = 1.13) own-name trials, 29.23 (*SD* = 1.18) close-other-name trials, 29.15 (*SD* = 1.31) unknown-name trials, 34.40 (*SD* = 1.37) own-face trials, 34.50 (*SD* = 1.32) close-other-face trials, and 33.80 (*SD* = 2.04) unknown-face trials, per participant.

Data analysis

We used the mean of values at each time point within a certain interval to assess the ERP components. This method is less affected by possible low single-to-noise ratio than peak analysis. Yet, for the analyses of the N170 we did look at peak amplitudes and latencies as a

meta-analysis reported N170 latencies to faces to be delayed in individuals with ASD (Kang et al., 2018). The N170 peaks were detected semi-automatically, with manual adjustment for peaks misidentified by BrainVisionAnalyzer. Definition and analyses of the components were based on the visual inspection of grand-average ERPs and scalp topography across conditions and groups (collapsed localizer approach, see Figure 1 and 2 for the scalp topographies), and on earlier studies (Nijhof et al., 2018; Tacikowski et al., 2011; Tacikowski & Nowicka, 2010). Within the positive deflection around 300 ms, two distinct components could be identified with unique stable topographies for both names and faces, which we refer to as the early and late P3. We quantified the components at the following time-windows and electrode sites. For names: late P3 (450-650 ms; pooled: P3 + Pz + P4), early P3 (320-400 ms; pooled: FCz + Cz + CPz), P2 (220-310 ms; Cz), and N1 (120-180 ms; Cz). For faces: late P3 (400-500 ms; pooled PO3 + P3 + POz + Pz + PO4 + P4), early P3 (290-400 ms; pooled PO3 + POz + PO4), N250 (250-300 ms; pooled left hemisphere: TP9 + P7 + PO7, pooled right hemisphere: TP10 + P8 + PO8), N170 (most negative peak between 110-210 ms at P7 and P8). We performed mixed-design ANOVAs separately for names and faces with Group (low-scoring, high-scoring) as between subject factor, and Person (own, close-other, unknown) as within subject factor. We chose to collapse (i.e. pool) data across nearby recording sites to prevent the loss of statistical power for all the components. For the N170 and N250 analyses, we added hemisphere (left, right) as an additional within subject factor. All effects with more than one degree of freedom in the numerator were adjusted for violations of sphericity according to the Greenhouse-Geisser formula (Greenhouse & Geisser, 1959). Bonferroni correction for multiple comparisons were applied to the post-hoc analyses.

For the correlation analyses we calculated difference scores of the late P3 amplitude for names and faces (i.e. own – close-other). These difference scores were used to assess the

relationship of self-referencing and ASD symptomatology, and the relationship of self-referencing of name and face.

Results

Our main hypotheses as formulated in the introduction relate to the late (parietal) P3 for both names and faces. For completeness we also performed analyses for the early P3, and two earlier components, that is, the N1 and P2 for names, and the N170 and N250 for faces. Below, we describe the results for the name and face stimuli separately starting with the late (parietal) P3, followed by the remaining components in temporal order.

Names

Late P3

The late P3 analysis yielded a main effect of Person, $F(2, 116) = 27.85$, $p < .001$, $\eta p^2 = .32$. The late P3 amplitude was larger for own names ($M = 1.85$, $SD = 1.44$), than for close-other names ($M = 1.14$, $SD = 1.25$; $p = .001$, $d = 0.52$), and unknown names ($M = 0.61$, $SD = 1.15$; $p < .001$, $d = 0.95$). The late P3 amplitude was also larger for close-other names than for unknown names ($p = .002$, $d = 0.44$). There was no main effect of Group, $F(1, 58) = 0.07$, $p = .793$, $\eta p^2 = .00$. Importantly, and unlike hypothesized, there was no Person x Group interaction effect, $F(2, 116) = 0.64$, $p = .527$, $\eta p^2 = .01$. The absence of the expected Person x Group effect was not due to the inclusion of unfamiliar names since omitting this condition from the analysis did not change the results. See Figure 1 for grand average waveforms and topographies of the late P3 for names.

N1

The N1 analysis yielded a main effect of Person, $F(2, 116) = 3.31$, $p = .040$, $\eta_p^2 = .05$, driven by a smaller N1 amplitude for own names ($M = -2.11$, $SD = 1.74$) than unknown names ($M = -2.63$, $SD = 2.06$; $p = .036$, $d = 0.27$). No differences between own names and close-

other names (M = -2.18, SD = 1.92), or close-other names and unknown names were revealed (p = 1.000, d = 0.04; p = .126, d = 0.22, respectively). There was no main effect of Group, $F(1, 58) = .03$, p = .867, η_p^2 = .00, or Person x Group interaction, $F(2, 116) = 1.59$, p = .209, η_p^2 = .27.

P2

The P2 analysis yielded a main effect of Person, $F(2, 116) = 5.80$, p = .004, η_p^2 = .09, driven by a smaller P2 amplitude for own names (M = 3.20, SD = 2.70) than for unknown names (M = 4.23, SD = 2.64; p = .002, d = 0.39). No difference between own names and close-other names (M = 3.61, SD = 2.50), or close-other names and unknown names were revealed (p = .634, d = 0.16; p = .132, d = 0.24, respectively). There was no main effect of Group, $F(1, 58) = 0.00$, p = .962, η_p^2 = .00, or Person x Group interaction effect, $F(2, 116)$ = 1.12, p = .330, η_p^2 = .02.

Early P3

The early P3 analysis yielded a main effect of Person, $F(1.82, 116) = 4.70$, p = .013, η_p^2 = .08, driven by a larger early P3 amplitude for own names (M = 3.25, SD = 2.37) than for unknown names (M = 2.48, SD = 2.17; p = .002, d = 0.34). No differences between own names and close-other names (M = 2.92, SD = 2.73), or close-other names and unknown names were revealed (p = .735, d = 0.13; p = .251, d = 0.17, respectively). There was no main effect of Group, $F(1, 58) = 0.12$, p = .730, η_p^2 = .00, or Person x Group interaction effect, $F(1.82, 116) = 1.25$, p = .288, η_p^2 = .02.

Conclusion

In sum, an enhanced late P3 amplitude was found for one's own name relative to the close-other name, indicating a self-reference effect. However, no group difference was found for this effect. Furthermore, none of the earlier components showed a self-reference effect

(i.e. no significant difference between own name and close-other name) or group differences. See Supplementary Table 1 for a summary of the ANOVA results.

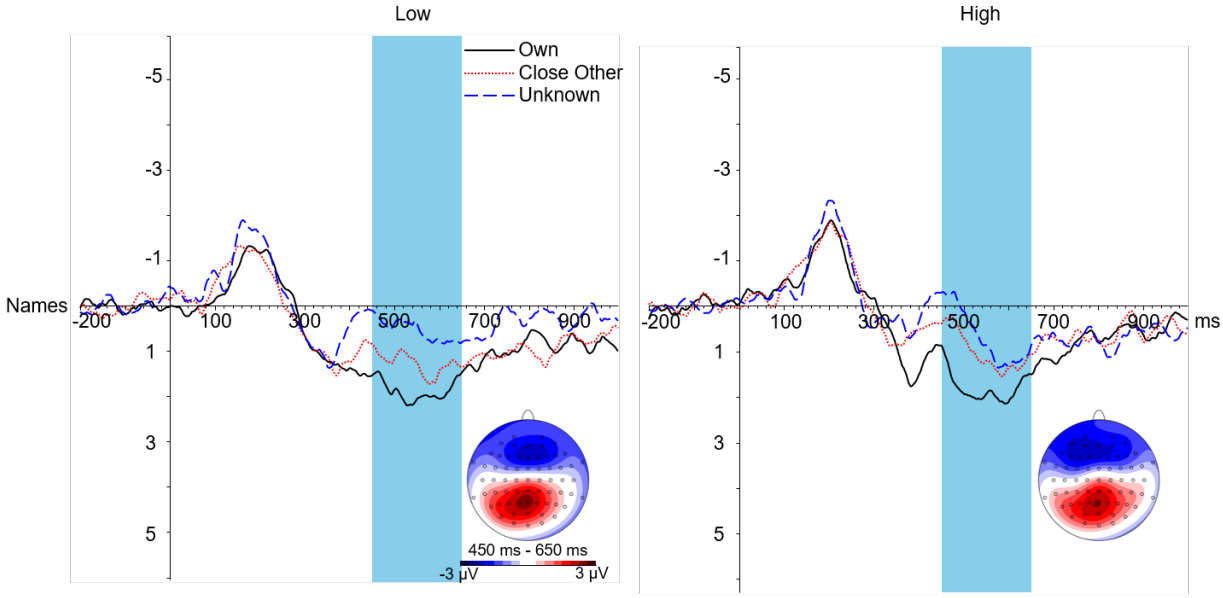

Fig 1 Grand average waveforms per name condition included in the late P3 (pooled: P3 + Pz + P4) analysis per group, as well as the topographies across conditions (450-650ms). Left: low-scoring group; right: high-scoring group. See Supplementary Fig 1 for the grand average waveforms and the topography of the N1, P2, and early P3.

Faces

Late P3

The Late P3 analysis yielded a main effect of Person, $F(2, 116) = 4.47$, $p = .013$, $\eta_p^2 = .07$, driven by a larger late P3 amplitude for own faces ($M = 4.45$, $SD = 2.10$) than for close-other faces ($M = 3.80$, $SD = 1.99$; $p = .012$, $d = 0.31$). No differences between own faces and unknown names ($M = 4.04$; $SD = 2.14$) or close-other faces and unknown faces were revealed ($p = .216$, $d = 0.19$; $p = .845$, $d = 0.07$, respectively). There was no main effect of Group, $F(1, 58) = .245$, $p = .622$, $\eta_p^2 = .00$. Importantly, and unlike hypothesized, there was no Person x

Group interaction effect, $F(2, 116) = .68$, $p = .509$, $\eta_p^2 = .01$. The absence of the expected Person x Group effect was not due to the inclusion of unfamiliar faces since omitting this condition from the analysis did not change the results. See Figure 2 for grand average waveforms and topographies of the late P3 for faces.

N170

The N170 peak amplitude analysis yielded a main effect of Hemisphere, $F(1, 58) = 21.83$, $p < .001$, $\eta p^2 = .27$, revealing that in general faces elicited more activity in the right hemisphere ($M = -5.17$, $SD = 3.42$) than in the left hemisphere ($M = -3.42$, $SD = 3.25$). There were no significant main effects of Person or Group, and no significant interaction effects, all $ps > .05$

The N170 peak latency analysis yielded a main effect of Person, $F(2, 116) = 6.82$, $p = .002$, $\eta p^2 = .11$, driven by a shorter latency for unknown faces ($M = 162.67$, $SD = 9.84$) than for close-other faces ($M = ,166.44$, $SD = 10.95$; $p = .011$, $d = 0.36$) and own faces ($M = 166.42$, $SD = 11.57$; $p = .005$, $d = 0.35$). No difference was found between own faces and close-other faces ($p = 1.000$, $d = 0.00$). The main effect of Hemisphere was not significant and so were the interaction effects, all $ps > .05$.

N250

The N250 analysis yielded a main effect of Person, $F(2, 116) = 7.69$, $p = .001$, $\eta_p^2 = .14$, driven by a larger amplitude for own faces ($M = 1.50$, $SD = 2.67$) than for close-other faces ($M = 2.19$, $SD = 2.71$; $p = .003$, $d = 0.26$) and unknown faces ($M = 2.33$, $SD = 2.86$; $p = .003$, $d = 0.30$). There was no difference between close-other faces and unknown faces ($p = 1.000$, $d = 0.05$). Furthermore, there was a main effect of Hemisphere, $F(1, 58) = 25.08$, $p < .001$, $\eta_p^2 = .30$, as generally faces elicited larger N250 amplitudes in the left hemisphere ($M = 1.29$, $SD = 2.75$) than in the right hemisphere ($M = 2.73$, $SD = 2.82$). There was no significant main effect of Group, $F(1, 58) = 1.56$, $p = .217$, $\eta_p^2 = .03$, and there were no significant

interaction effects, *p*s > .05.

Early P3

The early P3 analysis yielded a main effect of Person, $F(2, 116) = 5.89$, $p = .003$, $\eta_p^2 = .10$, driven by a larger amplitude for own faces ($M = 6.38$, $SD = 3.05$) than for unknown faces ($M = 5.73$, $SD = 3.12$; $p = .008$, $d = 0.21$). No differences between own faces and close-other faces ($M = 5.91$, $SD = 3.11$) or close-other faces and unknown faces were found ($p = .066$, $d = 0.15$; $p = .008$, $d = 0.06$, respectively). There was no main effect of Group, $F(1, 58) = .02$, $p = .884$, $\eta_p^2 = .00$, or Person x Group interaction effect, $F(2, 116) = 0.39$, p = .535, $\eta_p^2 = .01$.

Conclusion

In sum, similar to the names results, an enhanced late P3 amplitude was found for one's own face relative to the close-other face, indicating a self-reference effect. However, no group difference was found for this effect. The N250 also showed a self-referential effect with larger amplitudes for one's own face than the close-other face, but again groups did not differ for this effect. None of the other components showed a self-reference effect or group differences. See Supplementary Table 2 for a summary of the ANOVA results.

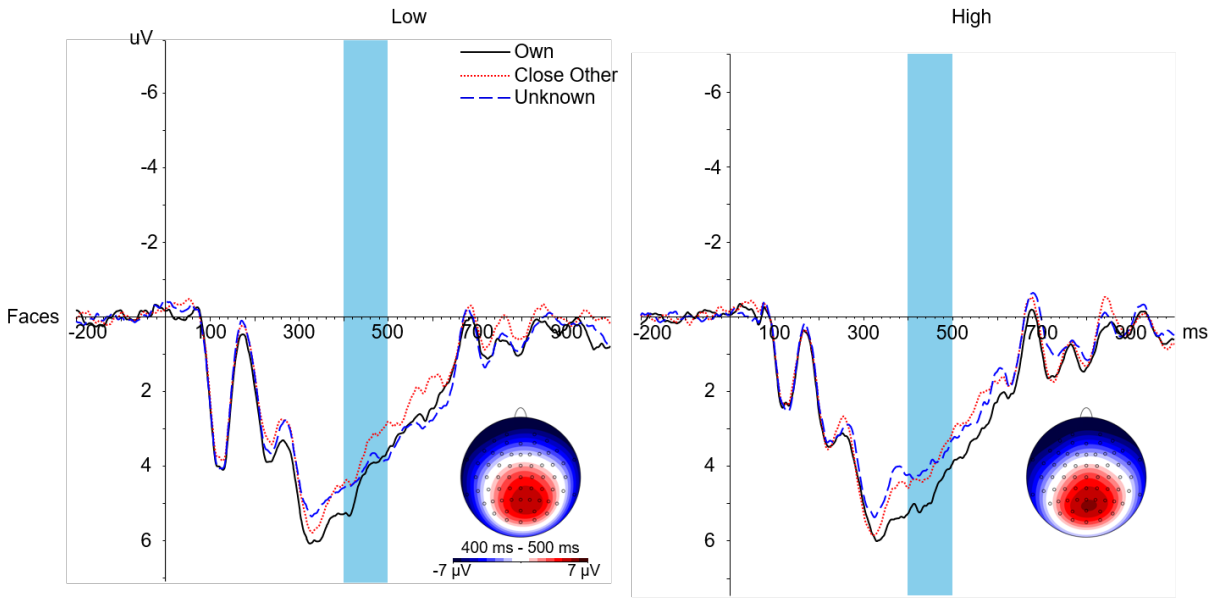

Fig 2 Grand average waveforms per face condition included in the late P3 (pooled: PO3 + P3 + POz + Pz + PO4 + P4) analysis per group, as well as the topographies (400-500ms) across conditions. Left: low-scoring group; right: high-scoring group. See Supplementary Fig 2 for the grand average waveforms and the topography of the early N170, N250 and early P3.

Correlation analyses

The late P3 difference scores (own – close-other) for names and faces (both pooled over included electrodes) did not significantly correlate with each other across groups, $r = .03$, $p = .832$, 95% CI [-0.23, 0.28] (see Figure 3)[1]. Analysing the two groups separately did not affect the results. The late P3 amplitudes for names and faces also did not significantly correlate with the AQ or SRS-A scores across groups, all *p*s > .05.

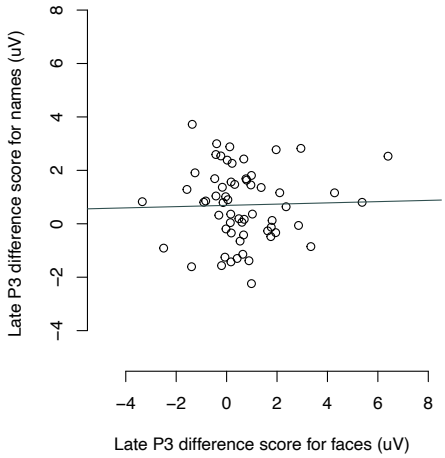

Fig 3 Scatterplot with regression line representing the association between the late P3 difference scores for names (y-axis) and faces (x-axis)

Discussion

This study examined the neural response to hearing one's own name and seeing one's own face (versus others' names and faces) in the same sample of adults scoring low versus

[1] For comparison reasons, we also performed the analysis, correlating the own name and own face amplitudes - an approach used by Tacikowski and Nowicka (2010). This did not change the results as again no significant correlation was found (r = .02, p = .867, 95% CI [-0.23, 0.28].

high on ASD symptomatology. In a relatively large sample of 60 participants, we found a self-referential effect for own names and faces, replicating previous studies. That is, we found the parietal P3 amplitude (in the current study referred to as late P3) to be enhanced for one's own name and face relative to the close-other name/face, indicating that the parietal P3 can be used as a valid index of self-referential processing. However, no relationship between the two self-reference effects (names and faces) was found. Furthermore, contrary to our prediction, we did not observe group differences for self-referential processing, neither for names nor faces.

The main aim of the current study was to directly compare self-referential processing of face and name stimuli within one and the same sample. While the parietal P3 showed a robust self-referential effect for both one's own name and face, the results showed a striking absence of association between the parietal P3 self-referential effects for names and faces, casting serious doubts on whether they can be explained by one common mechanism. Only one other study directly compared ERPs for different self-related stimuli in one and the same sample. In that study, a correlational analysis was performed to test the association between own name and face processing (Tacikowski & Nowicka, 2010). That study did report a significant correlation between the parietal P3 amplitudes elicited by one's own name and face in a sample of NTs. However, it has to be noted that their approach hampers correct interpretation of the correlation as it may simply reflect general amplitude differences between participants, i.e., those with inherently larger ERP amplitudes (due to inter-individual variation in skull thickness, conductivity, etc.) showing them in both tasks. To ensure that the correlation reflects an association between specific self-referential effects, we conducted analyses with self-referential indexes (difference amplitude between own and close-other stimuli) and found an almost fully absent correlation between the self-referential effect of own name and face processing. Note that the absence of a correlation was not due to our

correlational approach, as using the approach of Tacikowski and Nowcika (2010) did not change the result. Intuitively, our results may seem somewhat surprising, and we acknowledge that this finding should not be overinterpreted and that more research is needed before firm conclusions can be made. However, our finding is in line with the doubt that has been raised about the implicit assumption that one common underlying mechanism contributes to different self-referential effects. The mixed findings reported in the self-processing and ASD literature has led to the question whether different aspects of self-processing rely on one shared underlying mechanism or actually rely on distinct ones, and whether ASD is associated with general difficulties in self-referential processing or only with certain specific aspects of self-referential processing (Nijhof & Bird, 2019). Indeed, an overview of findings by Nijhof & Bird (2019) points out that some of the studies report altered whereas others reported intact self-processing in ASD depending on the type of self-processing task administered, indicative of distinct mechanisms (and impairments) underlying the self-referential effects found for some self-related stimuli (See Nijhof & Bird, 2019).

A second aim of the current study was to determine whether a reduced self-referential effect for one's own name and face is a specific, categorical feature of ASD, or a dimensional one that runs throughout the population. The late P3 effect we found across groups is in line with plentiful previous studies in NT and control samples and the effect seems to be a robust marker for self-referential processing (Name: e.g. Kotlewska & Nowicka, 2015; Face: e.g. Sui et al., 2006). However, we did not find support for the hypothesis that NT adults who score relatively high on ASD symptomatology would show a diminished self-referential effect for one's own name and face. This finding is intriguing given that the P3 self-referential effect for one's own name and face has repeatedly been reported to be diminished or even absent in adults with ASD (Cygan et al., 2014; Nijhof et al., 2018; Nowicka et al., 2016). Therefore, the results of the current study putatively suggest that the reduced self-referential effect for these

stimuli may be a specific, categorical feature of ASD – not a dimensional one. In other words, the reduced self-referential effect is only evident in individuals with a diagnosis of ASD and not in those with elevated ASD symptomatology in the general population. The current study is however the first that investigated self-referential processing in a NT sample scoring low versus high on ASD symptomatology. Hence, before firm conclusions can be made regarding the self-referential effect and the categorical vs. dimensional relationship with ASD, further investigation is warranted. Ideally, future studies should apply a broader dimensional approach, including NT adults scoring low and high on ASD symptomatology, and adults with a formal diagnosis of ASD. In fact, to make reliable conclusions there is also a need for replication attempts in adults with ASD. Up till now there is only one ERP study that investigated own face processing in adults with ASD (Cygan et al., 2014). For own name processing, there is the study by Nijhof and colleagues (2018), who applied the same paradigm as we did but in adults with ASD, and a second very recent study (published in the final stages of writing this paper) that included a sample of adolescents and young adults with ASD (Schwartz et al., 2020). Both Cygan et al. (2014) and Nijhof et al. (2018) report reduced self-referential processing in adults with ASD as reflected in reduced parietal P3 amplitudes. In contrast, no group differences were reported for the positive parietal deflection to own names in the study by Schwartz et al. (2020). Note though that, in contrast to numerous studies, they also did not find a main name effect and that they further did not include a familiar other name, hampering interpretation of the findings.

In addition to the findings with regard to our main hypotheses, some ERP results of our exploratory analyses are noteworthy. However, note that no group differences were found for these components either. Like the late P3, the early P3 amplitude seemed to be sensitive to the presentation of self-related stimuli, as we found it to be larger for own names and faces compared to unknown names and faces. However, the early P3 component did not

discriminate between the processing of own names and faces and that of close-others. This finding is in line with what the early P3 is thought to reflect, namely orienting processes as this component is often found to be enlarged for salient stimuli (in this case one's own name and face) that captures attention easily (Debener et al., 2005; Friedman et al., 2001; Nieuwenhuis et al., 2011). Interestingly, similar to the late (parietal) P3, the N250 amplitude for faces also showed a self-referential effect in our study, with an enhanced amplitude for one's own face relative to the close-other face. Although this component was thought to be an index of familiarity and not self-referential processing based on a study of Tanaka et al. (2006), our results are in line with a recent study by Alzueta et al. (2019). Alzueta et al. (2019) propose that the N250 reflects a familiarity effect in which one's own face is the most familiar face possible as they found both a self-referential effect as well as a familiarity effect in their study (own > close-other > unknown). The current study, however, only found differences between one's own face and the close-other face (and not between close-other and unknown faces) indicative of a specific self-referential effect. The N170 amplitude for faces was not modulated by the type of face in accordance with previous studies (Alzueta et al., 2019; Eimer, 2011; Estudillo, 2017). The latency analysis did reveal that the latency of the N170 seems to be modulated by the familiarity of faces, with shorter latencies for unknown faces than for close-other and own faces. This is in line with a study by Webb et al. (2010) that reported shorter latencies for unknown faces than for familiar faces. Lastly, we did not find delayed N170 latencies to faces in our sample of NT individuals who score relatively high on ASD symptomatology as was previously found in an ASD sample (Kang et al., 2018). For the name stimuli, we found a larger P2 amplitude for the unknown name relative to the own name, which was unexpected as visual name processing studies have reported the reversed effect (Fan et al., 2013; Tacikowski et al., 2014). Future studies are warranted to see whether these effects are modality dependent. Our N1 findings are partially in contrast with

the finding of Nijhof et al. (2018). Similarly, they found no group difference for the N1. However, they reported a familiarity effect instead of a novelty effect. Instead, we found a larger amplitude for hearing an unknown name relative to one's own name. These contrasting findings warrants further exploration.

The findings of the current study are subject to at least three limitations. First, one could argue that the difference in AQ score between groups might not have been large enough to detect group differences. However, the groups did significantly differ on the AQ, and previous research applying a similar selection method did find group differences on a spontaneous mentalizing task (Nijhof et al., 2017). Second, although the two groups in our study were matched on gender, it is worth mentioning that both groups mostly consisted out of females. Third, while the striking lack of association between parietal P3 self-referential effects for names and faces in our study argues against a common underlying mechanism, we cannot rule out that a diminished response for one's own name and face in adults with ASD as found in previous research might be related to a common mechanism. Only a future study investigating the relationship between own name and face processing in a clinical ASD sample may give more insight into this question.

To summarize, our study found the late (parietal) P3 to be a robust marker of self-referential processing for both one's own name and face, even when controlling for familiarity. Next to the late (parietal) P3, we also found a self-referential effect for the N250 for one's own face. Future research might explore this earlier component further. No relationship was found between self-referential processing of one's own name and face, tentatively suggesting that these effects may not be explained by one and the same underlying mechanism. Lastly, we did not observe group differences for the self-referential effect of both names and faces in our sample of NT individuals scoring low and high on ASD symptomatology. Our findings may suggest that the reduced self-referential effect found in

ASD samples, is a categorical feature of ASD, and not a dimensional one that runs throughout the whole population. However further research is definitely warranted before firm conclusions can be made.

Declaration

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Credit Author Statement

Danna Oomen: Validation, Formal analysis, Investigation, Data curation, Writing – original draft preparation, Writing – review and editing, Visualization; **Rachida El Kaddouri:** Conceptualization, Methodology, Writing – review and editing; **Marcel Brass:** Conceptualization, Methodology, Supervision, writing – review and editing; **Jan R. Wiersema**: Conceptualization, Methodology, Supervision, writing – review and editing

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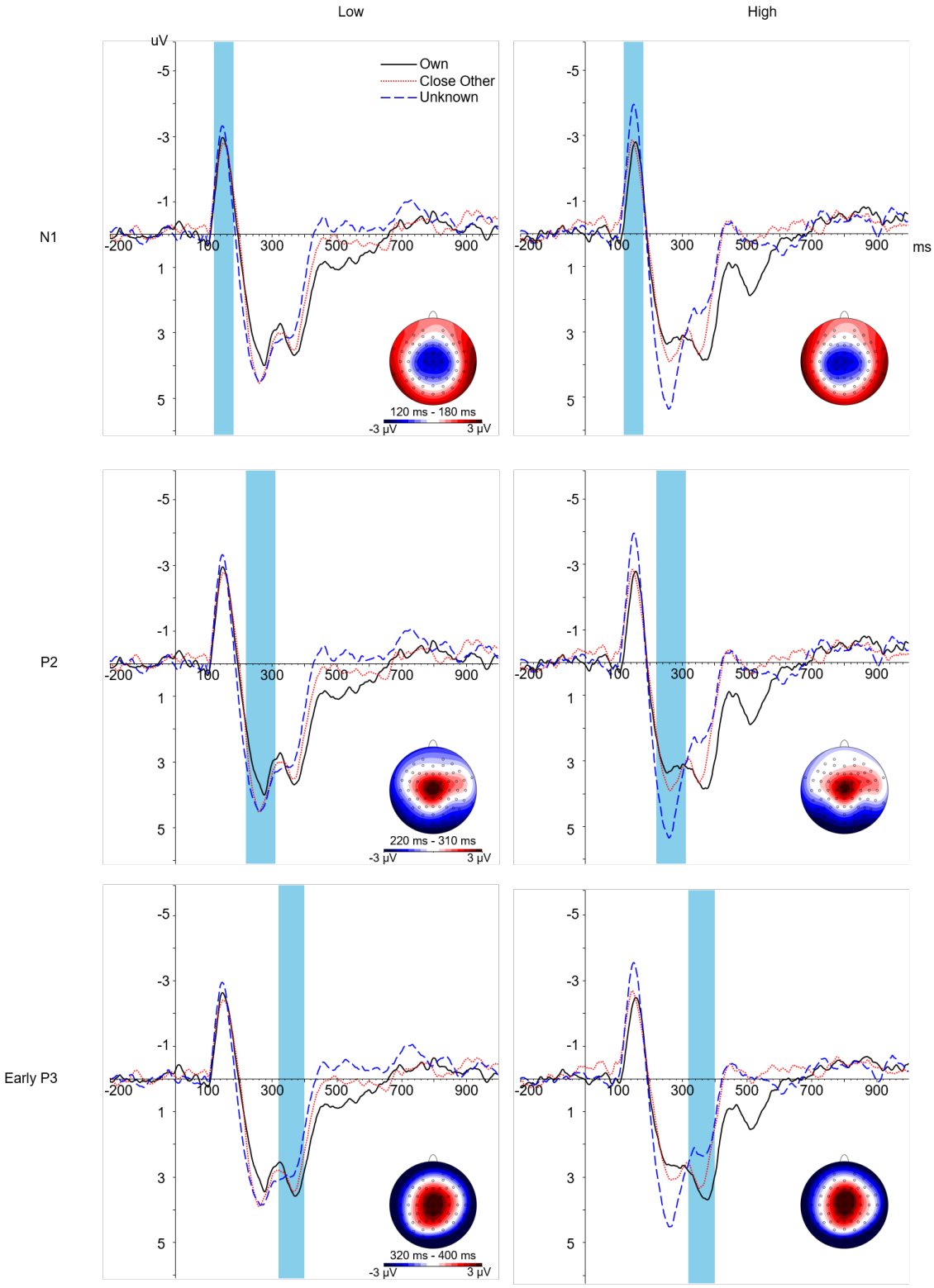
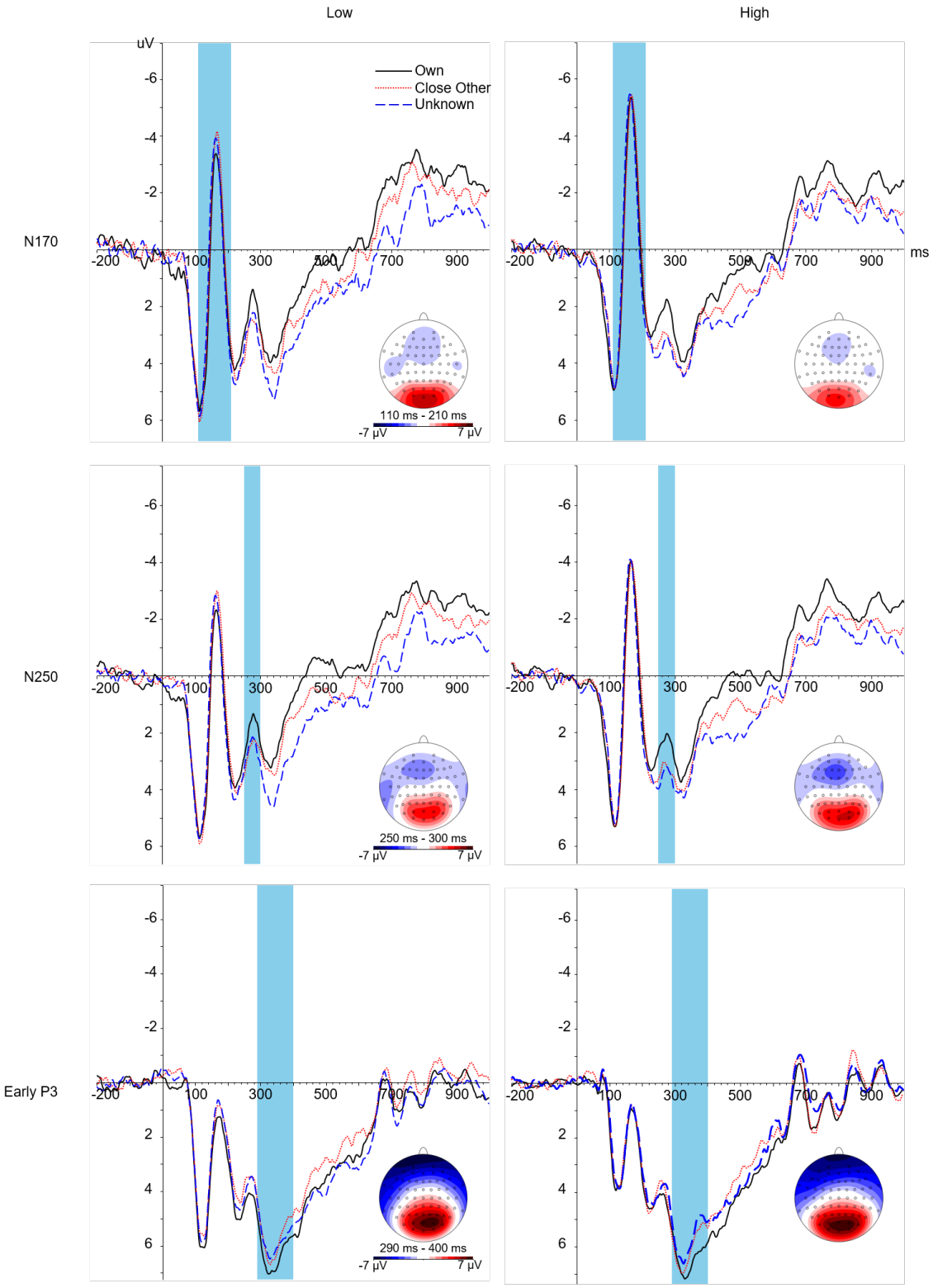
Supplementary figures



Supplementary Fig 1. Grand average waveforms per name condition included in the early P3 (pooled: Fcz, Cz, Cpz), P2 (Cz), and N1 (Cz) analysis per group, as well as the topographies. Left: low-scoring group; right: high-scoring group; Top: early P3; Middle: P2; Bottom: N1.



Supplementary Fig 2 Grand average waveforms per faces condition included in the early P3 (pooled: PO3 + POz + PO4), N250 (pooled right hemisphere: TP10, P8, PO8), N170 (right hemisphere: P8) analysis per group, as well as the topographies. Left: low-scoring group; right: high-scoring group; Top: Early P3; Middle: N250; Bottom: N170.

Supplementary Table 1
Summary of ANOVA results for names.

Component	Window, electrode(s)	Design	Effects	Result	Differences*
N1	120-180 ms, Cz	Group x Person	Person	$F(2, 116) = 3.31$, $p = .040$, $\eta p^2 = .05$	own = close, $p = 1.000$, $d = 0.04$ own < unknown, $p = .036$, $d = 0.27$ close = unknown, $p = .126$, $d = 0.22$
			Group	$F(1, 58) = .03$, $p = .867$, $\eta p^2 = .00$	
			Person x Group	$F(2, 116) = 1.59$, $p = .209$, $\eta p^2 = .27$	
P2	220-310 ms, Cz	Group x Person	Person	$F(2, 116) = 5.80$, $p = .004$, $\eta p^2 = .09$	own = close, $p = .634$, $d = 0.16$ own > unknown, $p = .002$, $d = 0.39$ close = unknown, $p = .132$, $d = 0.24$
			Group	$F(1, 58) = 0.00$, $p = .962$, $\eta p^2 = .00$	
			Person x Group	$F(2, 116) = 1.12$, $p = .330$, $\eta p^2 = .02$	
Early P3	320-400 ms, pooled: FCz + Cz + CPz	Group x Person	Person	$F(1.82, 116) = 4.70$, $p = .013$, $\eta p^2 = .08$	own = close, $p = .735$, $d = 0.13$ own > unknown, $p = .002$, $d = 0.34$ close = unknown, $p = .251$, $d = 0.17$
			Group	$F(1, 58) = 0.12$, $p = .730$, $\eta p^2 = .00$	
			Person x Group	$F(1.82, 116) = 1.25$, $p = .288$, $\eta p^2 = .02$	
Late P3	450-650 ms, pooled: P3 + Pz + P4	Group x Person	Person	$F(2, 116) = 27.85$, $p < .001$, $\eta p^2 = .32$	own > close, $p = .001$, $d = 0.52$ own > unknown, $p < .001$, $d = 0.95$ close > unknown, $p = .002$, $d = 0.44$
			Group	$F(1, 58) = 0.07$, $p = .793$, $\eta p^2 = .00$	
			Person x Group	$F(2, 116) = 0.64$, $p = .527$, $\eta p^2 = .01$	

Note. Group = (low-scoring, high-scoring); Person = (own, close-other, unknown); * Bonferroni correction for multiple comparisons applied.

Supplementary Table 2
Summary of ANOVA results for faces.

Component	Window, electrode(s)	Design	Effects	Result	Differences*
N170 amplitude	Peak analysis 110-210 ms, left: P7 right: P8	Group x Person x Hemisphere	Person	$F(1, 116) = 0.91$, $p = .404$, $\eta p^2 = .02$	
			Group	$F(1, 58) = 0.81$, $p = .372$, $\eta p^2 = .01$	
			Hemisphere	$F(1, 58) = 21.83$, $p < .001$, $\eta p^2 = .27$	Right > left
			Person x Group	$F(2, 116) = 0.35$, $p = .703$, $\eta p^2 = .01$	
N170 Latency	Peak analysis 110-210 ms, left: P7 right: P8	Group x Person x Hemisphere	Person	$F(2, 116) = 6.82$, $p = .002$, $\eta p^2 = .11$	own = close, $p = 1.000$, $d = 0.00$ own > unknown, $p = .005$, $d = 0.35$ close > unknown, $p = .011$, $d = 0.36$
			Group	$F(1, 58) = 0.00$, $p = .973$, $\eta p^2 = .00$	
			Hemisphere	$F(1, 58) = 0.04$, $p = .853$, $\eta p^2 = .00$	
			Person x Group	$F(2, 116) = 0.22$, $p = .725$, $\eta p^2 = .01$	
N250	250-300 ms, pooled left: TP9 + P7 + PO7, pooled right: TP10 + P8 + PO8	Group x Person x Hemisphere	Person	$F(2, 116) = 7.69$, $p = .001$, $\eta p^2 = .14$	own > close, $p = .003$, $d = 0.26$ own > unknown, $p = .003$, $d = 0.30$ close = unknown, $p = 1.000$, $d = 0.05$
			Group	$F(1, 58) = 1.56$, $p = .217$, $\eta p^2 = .03$	
			Hemisphere	$F(1, 58) = 25.08$, $p < .001$, $\eta p^2 = .30$	left > right
			Person x Group	$F(2, 116) = 0.14$, $p = .974$, $\eta p^2 = .00$	
Early P3	290-400 ms, pooled PO3 + POz + PO4	Group x Person	Person	$F(2, 116) = 5.89$, $p = .003$, $\eta p^2 = .10$	own = close, $p = .066$, $d = 0.15$ own > unknown, $p = .008$, $d = 0.21$ close = unknown, $p = .008$, $d = 0.06$
			Group	$F(1, 58) = .02$, $p = .884$, $\eta p^2 = .00$	
			Person x Group	$F(2, 116) = 0.39$, $p = .535$, $\eta p^2 = .01$	
Late P3	400-500 ms, pooled PO3 + P3 + POz + Pz + PO4 + P4	Group x Person	Person	$F(2, 116) = 4.47$, $p = .013$, $\eta p^2 = .07$	own > close, $p = .012$, $d = 0.31$ own = unknown, $p = .216$, $d = 0.19$ close = unknown, $p = .845$, $d = 0.07$
			Group	$F(1, 58) = .245$, $p = .622$, $\eta p^2 = .00$	
			Person x Group	$F(2, 116) = .68$, $p = .509$, $\eta p^2 = .01$	

Note. Group = (low-scoring, high-scoring); Person = (own, close-other, unknown); Hemisphere = (left, right) * Bonferroni correction for multiple comparisons applied.