Getting to know you: the acquisition of new face representations in autism spectrum conditions

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Social difficulties form a part of the canonical description of autism spectrum conditions (ASC), and the development of familiarity with new faces is a key ability required to navigate the social world. Here, we investigated the acquisition of new face representations in ASC by analysing the N170 and N250 event-related potential components induced by a previously unfamiliar face that was embedded in a series of other unfamiliar faces. We found that participants with ASC developed a smaller N250 component to the target face, indicating that the development of new face representations is impaired. We also found that the participants with ASC showed a smaller N170 component to both the target and the nontarget faces. This highlights the role of the early stages of face detection, structural encoding and attention in the formation of face memories in the typical population and implicates the dysfunction of these stages in the manifestation of the social difficulties observed in ASC. ***NeuroReport*** **23:668–672 © 2012 Wolters Kluwer Health | Lippincott Williams & Wilkins.**

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Along with unusually narrow interests, repetitive behaviours and difficulties with communication, social difficulties are a defining feature of autism spectrum conditions (ASC). In particular, the recognition of face identity is a particularly problematic area for individuals with autism. Children with ASC are less able to recognize pictures of their peers than typically developing children [1] and are less able to recognize pictures of their teachers [2].

Two event-related potential (ERP) studies have found deficits in components related to face recognition in children with ASC. Dawson *et al.* [3] found that typically developing children aged 3–4 years showed a differential response in the P400 and the Nc components for pictures of their mother compared with pictures of unfamiliar women. However, no such difference was found in the Nc or the P400 of age-matched children with ASC, suggesting that familiar faces do not activate the same neural system in young children with ASC as they do in typical development. Similarly, Gunji *et al.* [4] analysed a negative going wave between 200 and 400 ms over lateral posterior sites called the early posterior negativity (EPN). For typically developing children, the EPN was larger to the participants' mother's face than an unfamiliar face. However, in the ASC group, no significant difference was found in the amplitude of the EPN between these faces.

In typical adults, the N250 ERP component has been found to be larger to familiar faces [5] and to repetitions of unfamiliar faces that become familiar during the experimental session [6,7]. The N250 is a negative going wave that peaks between 200 and 400 ms after stimulus and is maximal over lateral occipitoparietal sites. The size of the N250 amplitude is related to the degree of familiarity with a face. Herzmann *et al.* [8] found that the N250 to repeated familiar faces is larger than the N250 to repeated unfamiliar faces. Tanaka *et al.* [7] found that the N250 is largest to one's own face and that previously unfamiliar faces induce an increasingly large N250 as familiarity is increased across the course of an experiment.

Preceding the N250 is the well-studied N170 ERP component. The N170 is an index of earlier face detection mechanisms [9] and is also a negative going wave typically recorded at the same occipitoparietal sites as the N250 but that peaks between 130 and 190 ms after the presentation of a face [7]. Importantly, the N170 is reduced in ASC in tasks that direct attention at face stimuli such as the task used in the current study [10].

The current study investigates the acquisition of new face representations in ASC by analysing face-specific and target-related ERP components induced by a previously unfamiliar face inserted as the target into a string of other unfamiliar faces. We hypothesized that individuals with ASC would show a decreased N250 to the target face compared with typical controls and a decreased N170 to both the target and the nontarget faces.

Methods

Participants

This study was approved by the Psychology Research Ethics Committee at the University of Cambridge and consent was obtained from all participants. Of a sample of

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15 ASC and 15 controls, 11 with ASC and 11 controls provided artefact-free data in at least 50% of the trials for averaging and were included in the final analyses. All participants were male and right-handed. Participants in the ASC group were diagnosed with Asperger syndrome according to international criteria (*Diagnostic and Statistical Manual of Mental Disorders*, 4th ed., text revision [11]) by a clinical psychologist or psychiatrist experienced in the diagnosis of ASC. The exclusion criteria for ASC participants were an uncorrected impairment in eyesight or hand movement, a personal or a family history of any psychological or genetic disorder apart from an ASC or a period of unconsciousness in the last 5 years. The exclusion criteria for the typical controls were the same, with the addition of a personal or a family history of ASC.

The participants with ASC had a mean age of 31.82 years (SD = 6.88) and a mean full-scale intelligence quotient ([11]) of 120.18 (SD = 13.99). The controls had a mean age of 30.1 years (SD = 4.92) and a full-scale intelligence quotient of 116.1 (SD = 15.03). There were no significant differences between the groups for these measures ($P > 0.5$). However, as expected, there was a significant difference between the groups on the autism spectrum quotient ([12]), with the ASC group scoring higher ($M = 33.64$, SD = 7.21) than the control group [$M = 15.4$, SD = 6.9, $t(19) = 5.91$, $P < 0.0001$].

Stimuli

The stimuli were eight previously unfamiliar faces, four of which were male and four were female, all taken from the Karolinska Directed Emotional Faces DVD [13]. All faces were a front portrait view and had a neutral expression. The images were edited in Photoshop CS3 (*http://www.adobe.com*), transformed into greyscale, mounted on a white background, equated for average luminance and contrast and resized to 5 × 7 cm.

Procedure

Participants were seated in a darkened room ~60 cm from the monitor on which the stimuli were presented. Electroencephalography (EEG) was recorded using a modified Quickcap (Compumedics Neuroscan, Charlotte, North Carolina, USA). Thirty-two electrodes were arranged according to the 10–20 system [14]. Reference was at the tip of the nose and ground at FPZ. Vertical and horizontal eye movements were recorded in bipolar channels with electrodes 1 cm above and below the left eye and from the outer canthus of each eye. All channels were recorded using a Synamps amplifier (Compumedics Neuroscan) that sampled the analogue signal at 1000 Hz with an analogue bandpass filter between 0.1 and 100 Hz. Impedance at each electrode was reduced to below 5 kΩ before the experiment was started.

Stimuli subtended $5.1 \times 7.3°$ of visual angle and were presented for 500 ms. The interstimulus interval was randomized between 1700 and 1900 ms. The stimuli were presented to participants in three blocks with a brief rest in between each. The total testing time was ~25 min. The stimulus train is described in Fig. 1. Each of the eight faces was presented to participants a total of 100 times in a random order. This order was constant for all participants. During a learning phase before the experiment, one of the faces was identified to the participants as the target face. The target face was counterbalanced across participants.

Participants were instructed to press one button with the index finger of one hand when they saw the target face and another button with the index finger of the other hand when they saw any other face. The hand used was counterbalanced across participants. A practice trial of 10 stimuli, including two presentations of the target face, was shown to participants following the instructions before the test phase.

Electrophysiology

Offline, the raw EEG was inspected visually before epoching, and major sections of artefact were excluded. Deflections because of eye blinks were corrected [15]. The continuous EEG was epoched from 150 ms before to 900 ms after the onset of the stimulus. The N170 and N250 were analysed at the sites at which they were maximal: P7 (left hemisphere) and P8 (right hemisphere).

Fig. 1

Example stimulus train including the learning phase (a) and a part of the test phase (b).

The mean amplitude of the N170 was calculated for the interval between 130 and 190 ms after stimulus onset, with the peak latency calculated for the maximum negative value within the same interval. The mean amplitude of the N250 was calculated for the interval between 230 and 320 ms after stimulus onset. However, as found previously by Tanaka *et al.* [7], an N250 peak was not always visible in the nontarget condition; thus, peak latency could not be measured.

Statistical analysis

The percentage of correctly identified faces in each category was entered into a two-way mixed analysis of variance with face type (target, nontarget) and participant group (ASC, typical control) as factors. The N170 and N250 were analysed using three-way mixed analysis of variances with hemisphere (left, right), face type (target, nontarget) and participant group (ASC, typical control) as factors.

Results

Behavioural results

For the percentage of faces identified correctly, there was a main effect of face, type showing that nontarget faces ($M = 88.52\%$, SD = 1.86) were identified more accurately than the target face [$M = 70.73\%$, SD = 8.48, $F(1,20) = 81.58$, $P < 0.0001$]. No main effect or interaction was found involving participant group.

Event-related potential results

The N170

The N170 was larger (more negative) in the control group ($M = -4.23\,\mu V$, SD = 2.91) than in the ASC group [$M = -1.3\,\mu V$, SD = 2.44, $F(1,20) = 6.45$, $P = 0.02$] across face type and hemisphere. No other main effects or interactions were significant for the N170 amplitude and no significant effects were found for the N170 latency.

The N250

Across participant group and hemisphere, the N250 was larger to target faces ($M = -1.53\,\mu V$, SD = 5.64) than to nontarget faces [$M = -5.09\,\mu V$, SD = 3.83, $F(1,20) = 67.7$, $P < 0.000$]. This was qualified by an interaction between face type and participant group [$F(1,20) = 5.73$, $P = 0.03$] in which there was no significant difference in the amplitude of the N250 between the participant groups to nontarget faces [$t(20) = 1.11$, $P = 0.28$], but a significant difference between the groups to target faces [$M = 1.29\,\mu V$, SD = 5.24, $t(20) = 2.67$, $P = 0.02$].

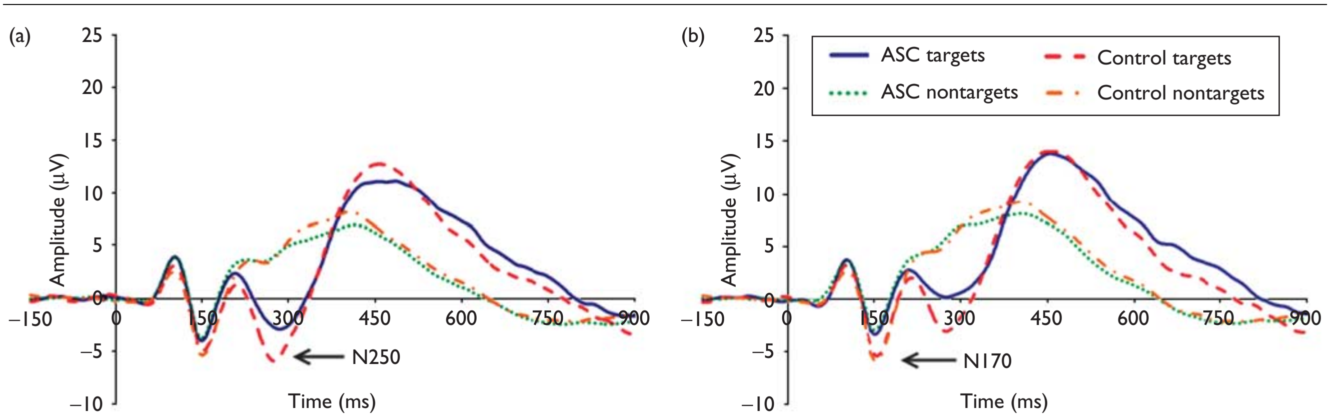
There was also a significant main effect of hemisphere [$F(1,20) = 5.86$, $P = 0.03$] in which the N250 was larger in the left hemisphere ($M = 1.14\,\mu V$, SD = 4.43) than in the right hemisphere ($M = 2.42\,\mu V$, SD = 4.59). There were no other main effects of interactions for the amplitude of the N250. The grand average waveforms are shown in Fig. 2.

Discussion

This study investigated the acquisition of new face representations in ASC. Consistent with the hypothesis, the N250 was reduced for the target face in the ASC group, suggesting that the development of new face representations is impaired in ASC. These results from an adult sample are consistent with two previous ERP studies of familiar face processing in ASC, involving children [3] and adolescents [4].

However, in contrast to the current results, another recent study of the N250 in adults with ASC found no difference between the ASC and typically developing groups [16]. The difference between these two findings can be attributed to differences in the paradigms used. In the study by Webb *et al.* [16], the attention of participants was directed away from the face stimuli by task instructions to press the response button to infrequently occurring

Fig. 2



Grand average waveforms for (a) P7, (b) P8 showing waveforms for target and nontarget faces for both participant groups. ASC, autism spectrum conditions.

houses. However, Neumann and Schweinberger [17] and Trenner *et al.* [18] reported that the N250 is reduced when attention is directed away from the face stimuli. Thus, the paradigm used by Webb *et al.* [16] is likely to have resulted in a reduced N250 in both groups and reduced the possibility of between-group differences.

The difference in attention created by this change in methodology also explains the different findings between the current study and those of Webb *et al.* [16] for the N170 component. In the current study, the N170 was larger in the control group than in the ASC group across both face types, whereas Webb *et al.* [16] found no difference between the groups in the N170. In agreement with the current findings, our earlier work also found that typical controls showed an increase in the amplitude of the N170 beyond that shown by individuals with ASC when participants' attention was directed towards faces but that this between-group difference was absent when participants' attention was directed away from the face stimuli.

Hence, when viewed together, the results of the current study and those of Webb *et al.* [16] emphasize the important relationship between attention and memory for faces and the deficits in this relationship in ASC. Building on the biased competition model of visual attention, Desimone ([19]) argues that 'Attention is often thought of as the gateway to memory, as we typically remember little about stimuli we ignore'. The results of Churches *et al.* [10] showed that in ASC, attentional mechanisms do not engage with the early face detection system indexed by the N170, and together with the results of Webb *et al.* [16], the current results indicate that this early attentional deficit impacts on the later processing of face familiarity, reducing the ability of individuals with ASC to develop the new face representations indexed by the N250.

Thus, a likely neurological basis for the decreased N250 in ASC is a decrease in the connectivity between the source of the N250 in temporal brain regions and the frontal brain regions that subserve attentional processes [20,21]. Imaging studies in the typical population and in animal models have shown that attention regulates activity in the temporal cortex through feedback from frontal regions [22]. Furthermore, there is evidence that this connectivity between frontal and temporal regions is reduced in ASC, especially during face perception tasks [23].

Conclusion

Participants with ASC showed a reduced N250 for target faces and a reduced N170 to both target and nontarget faces. This suggests that the acquisition of new face representations is impaired in ASC, but that predictable and repetitive tasks such as the one used here can be completed by individuals with ASC using neural strategies other than the development of new face representations. The reduced N170 component in the ASC group highlights the role of attention in the formation of new face representations in the typical population and implicates the dysfunction of these attentional systems in the manifestation of the clinical symptom of social difficulties in ASC.

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Conflicts of interest

There are no conflicts of interest.

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