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Slow segmentation of faces in Autism Spectrum Disorder

C. van den Boomen[a,b,*], J.J. Fahrenfort[c], T.M. Snijders[d,e], C. Kemner[a,b,f]

[a] Dept. of Experimental Psychology, Helmholtz Institute, 3584 CS Utrecht, the Netherlands
[b] Dept. of Developmental Psychology, Utrecht University, 3584 CS Utrecht, the Netherlands
[c] Department of Experimental and Applied Psychology, Vrije Universiteit Amsterdam, 1081 BT Amsterdam, the Netherlands
[d] Max Planck Institute for Psycholinguistics, 6500 AH Nijmegen, the Netherlands
[e] Donders Institute for Brain, Cognition, and Behaviour; Donders Centre for Cognitive Neuroimaging, Radboud University, 6525 EN Nijmegen, the Netherlands
[f] Dept. of Child and Adolescent Psychiatry, Rudolf Magnus Institute of Neuroscience, University Medical Center, 3584 CX Utrecht, the Netherlands

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ABSTRACT

Atypical visual segmentation, affecting object perception, might contribute to face processing problems in Autism Spectrum Disorder (ASD). The current study investigated impairments in visual segmentation of faces in ASD. Thirty participants (ASD: 16; Control: 14) viewed texture-defined faces, houses, and homogeneous images, while electroencephalographic and behavioral responses were recorded. The ASD group showed slower face-segmentation related brain activity and longer segmentation reaction times than the control group, but no difference in house-segmentation related activity or behavioral performance. Furthermore, individual differences in face-segmentation but not house-segmentation correlated with score on the Autism Quotient. Segmentation is thus selectively impaired for faces in ASD, and relates to the degree of ASD traits. Face segmentation relates to recurrent connectivity from the fusiform face area (FFA) to the visual cortex. These findings thus suggest that atypical connectivity from the FFA might contribute to delayed face processing in ASD.

1. Introduction

Autism Spectrum Disorder (ASD) is generally known as a disorder with symptoms in social behavior, communication, and behavioral flexibility. An increasingly recognized problem is the hypo- or hypersensitivity to visual information (DSM-V, APA, 2013). For instance, many report problems in basic visual processes, such as a bias towards local details instead of the global configuration (Dakin and Frith, 2005; Simmons et al., 2009). More complex visual processes are also impaired, such as discrimination of emotions in a face (Uljarevic and Hamilton, 2013). Visual segmentation is an intermediate process that includes integration of local visual elements into a global object, detection of its borders, and segregation of the object from its background (Lamme and Roelfsema, 2000). Because segmentation involves combining details into an object (Hochstein and Ahissar, 2002) and seems to guide or interact with explicit object recognition (Koivisto et al., 2011; van den Boomen et al., 2015; van Loon et al., 2016), it was proposed that atypical segmentation might underlie problems in both basic and complex vision (Kemner et al., 2007).

Segmentation plays an important role in perception: it leads to conscious perception of an object (Fahrenfort et al., 2012). Furthermore, recurrent neural processes involved in segmentation are also involved in gaining detailed information about an object (Ahissar et al., 2009; Hochstein and Ahissar, 2002). The process of segmentation is, in both typical and atypical populations, often studied using two types of stimuli that contain multiple (line) elements: textured objects and homogeneous images (Fig. 1; e.g. Bach and Meigen, 1992, 1998; Caputo and Casco, 1999; Kemner et al., 2007; Kemner et al., 2009; Lamme et al., 1992). In textured objects, the lines differ in orientation to form an object on a background. In homogeneous images, all lines have the same orientation. Segmentation is reflected in the difference in brain or behavioral responses evoked by a textured versus a homogeneous stimulus (Bach and Meigen, 1992; Fahrenfort et al., 2012; Lamme et al., 1992; Scholte et al., 2008). Multiple studies in primates and humans led to neural models explaining the role of recurrent processing in segmentation and perception of detail (Hochstein and Ahissar, 2002; Lamme and Roelfsema, 2000). These models broadly propose that coarse global visual information is first processed via feedforward connectivity from lower to higher areas in the visual hierarchy. Higher areas include for instance the fusiform face area (FFA), which strongly responds to faces (Kanwisher et al., 1997). Subsequently, higher areas interact with lower areas via recurrent connectivity. This recurrent connectivity supposedly brings about segmentation (Lamme and Roelfsema, 2000) and perception of detail (Hochstein and Ahissar,

* Correspondence to: Utrecht University, Department of Experimental Psychology, Heidelberglaan 1 – room H0.66, 3584 CS Utrecht, the Netherlands.
E-mail address: C.vandenboomen@uu.nl (C. van den Boomen).

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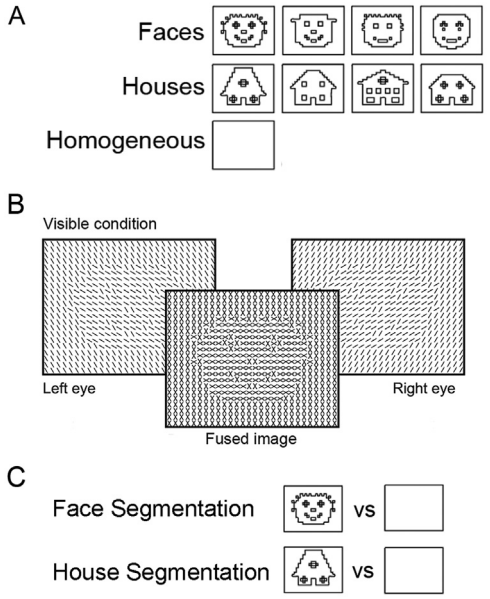

Fig. 1. Examples of stimuli presented to study segmentation and categorization of texture-defined objects. (A) Schematic versions of the images of faces, houses, and homogeneous textures. Black lines represent object borders, defined by differences in Gabor orientation. (B) Schematic versions of the stimuli. For clarity purposes, black lines instead of Gabor patches are depicted in this figure. (C) Contrasts to separate face- and house-segmentation responses.

2002). Possible abnormalities in segmentation thus reflect atypical recurrent connectivity, and could affect the success of subsequent visual processes that rely on segmentation.

Contrary to the expectations, previous research using geometric forms revealed that segmentation is typical in ASD (Kemner et al., 2007; Vandenbroucke et al., 2008, 2009). However, young children with ASD were less accurate in behaviorally identifying textured non-social everyday objects than controls (Evers et al., 2014). Furthermore, a formal meta-analyses of behavioral studies revealed that while persons with ASD are as good in segmentation as controls, they are slower in behaviorally integrating local elements into global whole (van der Hallen et al., 2015). This suggests that segmentation might be impaired in ASD for more complex objects and that impairments are particularly present in the temporal domain. Of all complex images, segmentation of faces is particularly interesting. Face processing is atypical in ASD (e.g. Dawson et al., 2005), but it is unknown whether this abnormality could arise from atypical segmentation. Previous imaging studies suggested that atypical face processing relates to decreased functional connectivity within and from the FFA (e.g. Khan et al., 2013; Kleinhans et al., 2008). No studies reported decreased connectivity from the FFA to the early visual cortex, which would relate to atypical segmentation. Nevertheless, previous results might suggest that atypical connectivity may contribute to atypical face processing. By studying segmentation using electroencephalography (EEG), we can investigate whether there are specific impairments in the quality or speed of face segmentation in ASD.

The current study investigated whether visual segmentation of faces is impaired in ASD. As such, we presented texture-defined faces and houses (Fig. 1). We presented stimuli for a short duration, as was done in previous research in non-ASD adults (van den Boomen et al., 2015). Short presentation duration prevented a possible ceiling effect of segmentation, which could partly explain an absence of group differences in previous studies (Kemner et al., 2007). Segmentation was evaluated using behavioral and neurophysiological (EEG) measures. Impairments would appear as a lower segmentation performance, in addition to slower responses, in ASD for the behavioral task. In the EEG signal, we were interested in the peak difference in activity evoked by textured objects versus homogeneous stimuli, representing the segmentation process. We expected this difference to be smaller and to occur later, if segmentation would be affected in ASD. This group difference was hypothesized to be particularly present for the face stimuli. In addition, we investigated whether neural and behavioral segmentation abilities related to ASD traits. We expected a positive correlation between the degree of segmentation impairment and the level of ASD traits.

Table 1
Subject characteristics of the ASD and Control groups. Means and standard errors are provided, as well as t-values; Asterisk represents significant difference between groups ($p < .001$). TIQ = total scale IQ; VIQ = verbal IQ; PIQ = performance IQ; AQ = autism quotient.

	ASD group	Control group	$t(28)$
Sample size	16	14	
Gender	4 female; 12 male	2 female; 12 male	
Age	24 (0.21)	23 (0.16)	1.3
TIQ	117 (3.2)	120 (3.6)	−0.6
VIQ	118 (3.1)	121 (3.0)	−0.5
PIQ	112 (3.5)	116 (4.9)	−0.6
Acuity (logMar)	−0.19 (0.01)	−0.20 (0.04)	0.2
AQ	132 (4.0)	100 (2.7)	6.6*

2. Methods

2.1. Participants

Sixteen adults with ASD (4 females, 12 males) and 14 control subjects (2 females, 12 males) participated in the study. Three additional subjects (1 ASD; 2 Controls) were tested but excluded because they deviated more than 3 inter quartile range from the group median (EEG latency: N = 2; raw reaction times: N = 1). Please see Table 1 for descriptive statistics for included participants in both groups.

ASD subjects were recruited through an existing database and through organizations for patients with ASD. The diagnostic evaluation of subjects in the ASD group included a psychiatric observation and a review of prior records (developmental history, child psychiatric and psychological observations and tests). ASD was diagnosed by a child psychiatrist, using the DSM-IV criteria. In addition, parents of fourteen ASD subjects were administered the Autism Diagnostic Interview (Lord et al., 1994), and thirteen of the participants with ASD were administered the Autism Diagnostic Observation Schedule- generic (Lord et al., 1989), both by a trained rater. The ASD group consisted of 11 participants diagnosed with Asperger's Syndrome, 5 diagnosed with Autism, and 1 diagnosed with PDD-NOS. Control subjects were recruited on campus and were screened for ASD, ASD in their family, and history of psychopathology using self-report. None of the control subjects reported ASD or psychopathology, although two subjects had first- degree relatives diagnosed with ASD.

The Dutch translation of the Autism Spectrum Quotient (AQ; Baron-Cohen et al., 2001a, 2001b) was administered to all subjects to investigate ASD traits. The AQ was coded according to the Dutch guidelines, which results in a score between 50 and 200 (Hoekstra et al., 2008). The ASD group had a significantly higher AQ score than the control group (ASD: $M = 131$; $SE = 3.8$; Controls: $M = 98$; $SE = 2.7$; $t(31) = 7.1$; $p < .001$). The Wechsler Adult Intelligence Scale III, Dutch edition (WAIS-III) was used to determine IQ. The full scale was used for the ASD group, and the abbreviated scale (WASI) was used for the control group. The Freiburg Visual Acuity Test (Bach, 1996) was used to measure visual acuity and Weber contrast sensitivity (i.e. sensitivity for contrast between feature and background luminance). The ASD and control groups did not differ significantly on age, IQ, visual acuity or contrast sensitivity, and all subjects had normal or corrected-to- normal vision.

Both the subjects with ASD and the control participants received a

monetary reward for their participation. The medical ethics committee of the University Medical Centre Utrecht approved the study. Informed consent was obtained from all individual participants included in the study.

2.2. Procedure and stimulation

The procedure and stimulation are similar to those described in van den Boomen et al. (2015). We recorded behavioral performance and brain activity using EEG, while participants viewed texture-defined faces or houses, or homogeneous textures (Fig. 1). To keep stimulus parameters the same as in previous studies (Fahrenfort et al., 2012; van den Boomen et al., 2015), we created a visible and invisible stimulus condition. The invisible condition was presented in the current experiment as well, but not of interest to the current research purposes and hence not analyzed (see van den Boomen et al., 2015) for further details on the invisible condition). For stimulus presentation, we used 3 stimulus categories: faces, houses and homogeneous. All stimuli contained a matrix of Gabor elements of specific orientations. Face- and house-categories were created using different orientations (22.5°; 67.5°; 112.5° or 157.5°) for fore- and background. Gabor elements of the homogeneous stimuli had one of the four orientations per stimulus. Stimuli were followed by a mask, which consisted of a field of Gabor elements with random orientation. Visibility was manipulated by presenting a stimulus to each of the eyes separately (referred to as dichoptic stimulation; (Fahrenfort et al., 2012; Moutoussis and Zeki, 2002; Wolfe, 1983)). Monocular presentation was achieved by having participants view a screen with a presentation rate of 120 Hz through shutter glasses, blocking the visual field to each eye alternatingly at a rate of 60 Hz. Consequently, each eye processed a different version of the same stimulus at a rate of 60 Hz. When different stimuli are presented to the left and the right eye for a short time, such as 92 ms in the current experiment, the brain fuses stimuli of the two eyes into a single percept rather than inducing binocular rivalry (Wolfe, 1983). Both eyes processed the same object, but the object contained differently oriented Gabor elements for the left and the right eye. The figure was only visible when the orientations between figure and background were different in the respective eyes (Fig. 1), and this was the only condition we analyzed here. Even though the monocular presentation was not necessary for the current research question, it is unlikely that the required fusion into a binocular percept hampers the interpretation of the specific results of this study. Binocular fusion is required in all conditions (i.e. face, house, but also homogeneous). The here reported results represent the difference in evoked activity between face and homogeneous or house and homogeneous stimuli. Possible effects of fusion are therefore likely canceled out. Nevertheless, a possible interaction of binocular processing abilities and participant group cannot be excluded in the current study. The stimulus sequence consisted of object presentation for 92 ms, followed by a mask for 50 ms, and an inter-stimulus-interval for 1600–2000 ms (grey screen with fixation cross). Participants used a chinrest to stay at a distance of 45 cm from the screen, such that stimuli measured 16.9° × 12.7° of visual angle.

During EEG measurement, stimulus presentation contained two blocks of 32 trials per stimulus condition (i.e. face, house, or homogeneous), resulting in a total of 192 randomly presented trials. An ellipse was presented surrounding the object, which appeared to be hovering in front or behind the stimulus screen due to a slight offset in the left and right eye (see Fahrenfort et al., 2012). During the EEG measurement, the participant indicated whether the ellipse appeared in front or behind the stimulus screen. The purpose of this task was to facilitate dichoptic fusion and prevent differences in attention between stimulus categories, which could affect the EEG signal. During the behavioral run, stimulus presentation contained one block of 21 trials per stimulus condition (i.e. face, house, or homogeneous), resulting in a total of 63 randomly presented trials. Participants indicated by button-press whether they perceived a face, house, or homogeneous image.

2.3. EEG recording and analyses

2.3.1. Recording

A Biosemi Active Two EEG system (Biosemi, Amsterdam, The Netherlands) recorded EEG activity from 32 electrodes. We positioned electrodes at standard EEG recording locations according to the international 10/20 system. Electrodes above and below the left eye recorded vertical EOG to detect blinks, and electrodes near the outer canthi of the eyes recorded horizontal EOG to detect horizontal eye movements. Two additional electrodes were placed at the left and right mastoid to maintain the possibility of offline re-referencing to these electrodes. During recording, the EEG sampling rate was 2048 Hz. Two electrodes in the cap, the CMS (Common Mode Sense) and DRL (Driven Right Leg), provided an 'active ground'.

2.3.2. Preprocessing analyses

Preprocessing analyses were performed in Brain Vision Analyzer (Amsterdam, the Netherlands). First, we resampled data offline to 512 Hz, and filtered them with a high-pass filter of 0.1592 Hz (24 dB/oct), a low-pass filter of 20 Hz (24 dB/oct) and a notch filter of 50 Hz. In order to compute ERPs, epochs of 100 ms pre-stimulus (baseline) until 800 ms post-stimulus were extracted from the continuous data. Epochs with large artifacts were removed. Activity was an artifact when amplitudes were below -200 or above 200 μV. A regression analysis based on eye-movements detected by vertical EOG (blinks) and horizontal EOG electrodes (horizontal eye-movements) removed ocular artifacts from the EEG (Gratton et al., 1983). Then, additional artifacts were rejected for each individual electrode. Activity was an artifact when there was a voltage change of 50 μV per sampling point, a difference of 1 μV per 100 ms, or amplitudes below −50 or above 50 μV. Activity was re-referenced to the average of all 32 cap-electrodes. We corrected for baseline activity, with baseline defined from −100 ms. to stimulus onset. Finally, data was averaged per condition.

We used ERP peak latency analyses to reveal the timing of segmentation-related responses, and amplitudes to reveal their strength. To perform these analyses, we first contrasted activity evoked by object (average of face and house) versus homogeneous stimuli, and by face or house separately versus homogeneous stimuli. This created segmentation difference waves. Then, peaks were detected as the local maximum of activity in each difference wave between 0 and 350 ms after stimulus onset. Peak detection was visually checked to detect cases in which no clear peak was visible (ASD: faces: N = 3; houses: N = 4; Control: faces: N = 1; houses: N = 3) or in which a double peak was visible (ASD: faces: N = 4; houses: N = 5; Control: faces: N = 3; houses: N = 1). Please see the Supplementary material S1 for examples of no, one, and two peaks. When no clear peak or when two peaks were present, we checked whether the automatically detected peak was within the timeframe of major peaks in the raw ERP of the participant (evoked by the house, face, and homogeneous stimulus separately). Previous studies indicate that typical segmentation-related activity is observed at the timeframe of the major ERP peaks evoked by the individual stimuli (e.g. van den Boomen, 2015). If the detected peak was not within this timeframe, we adjusted it to the highest peak in the difference wave within this timeframe. Participants without a clear peak were included in the analyses as not to bias the results, but results were qualitatively the same when excluding these participants. Segmentation-related activity was analyzed at the Oz electrode, based on previous research (Bach and Meigen, 1992; Lamme et al., 1992; van den Boomen et al., 2015).

2.3.3. ERP group analyses

Two independent *t*-tests analyzed group differences on segmentation-related activity evoked by all objects (average of faces and houses), for peak amplitude and latency. In addition, group differences on face or house segmentation were evaluated using repeated measures ANOVAs with group (ASD; controls) as between subject independent

variable, stimulus (face-homogeneous; house-homogeneous) as within subject independent variable, and peak amplitude and latency as dependent variables.

2.4. Behavior analyses

Behavioral performance was computed separately for face and house segmentation. For both types of segmentation, we subtracted the false alarm rate (FAR) from the hit rate (HR). Responses were defined as face segmentation hit when a face was detected as face. This was divided by the number of presented face stimuli to calculate face hit rate (HR). Responses were defined as face segmentation false alarms when a homogeneous image was detected as face. This was divided by the number of homogeneous stimuli to calculate face false alarm rate (FAR). In the same way, HR and FAR were calculated for house responses. Furthermore, interest was in reaction times related to face or house segmentation. Subtracting median reaction times for correct responses on homogeneous stimuli from those for correct responses on face stimuli led to face segmentation reaction times. This calculation was also applied for house segmentation.

Performance and reaction time were each evaluated by a repeated measures ANOVA using stimulus type (faces, houses) as within, and group (ASD, controls) as between subject independent variables, and performance (HR-FAR) or reaction time (face minus homogeneous; houses minus homogeneous) as dependent variable.

2.5. Correlations to ASD traits

To investigate whether variations in segmentation abilities relate to variations in the ASD phenotype, we correlated the segmentation results to the total score on the Autism Quotient (AQ) questionnaire. Specifically, we performed eight bivariate correlation analyses, namely on the ERP amplitude and latency of face and house segmentation-related activity, and on the behavioral segmentation performance and reaction time for face- and house segmentation. P-values of the Pearson correlation coefficients were evaluated with an alpha of 0.006 to correct for multiple testing.

3. Results

3.1. ERP group results

Figs. 2 and 3, and Table 2 present group averages of the segmentation-related EEG responses, and Fig. 4 presents individual results. Please see Supplementary materials S2 for further individual results. All assumptions for *t*-tests and ANOVA were confirmed. T-tests on group differences in segmentation-related ERP peak latency, averaged over faces and houses, revealed a significantly longer latency for the ASD than control group ($t(28) = 2.2$; $p = .038$; ASD: $M = 250$; $SE = 7.4$; Control: $M = 223$; $SE = 10.1$). Repeated measures ANOVA with stimulus type as an additional variable revealed an interaction between stimulus and group on peak latency ($F(1,28) = 6.4$; $p = .017$; $\eta^2 = .187$). Follow-up *t*-tests revealed that this was due to slower segmentation of faces in the ASD compared to the control group ($t(24.4) = 3.3$; $p = .003$). No group difference was found for the house stimuli ($t(20.9) = 0.37$; $p = .712$). Furthermore, control subjects segmented faces faster than houses ($t(13) = -4.3$; $p = .001$) whereas this was not significant in persons with ASD ($t(15) = -1.8$; $p = .084$).

For ERP peak amplitude, there was no difference between groups for the averaged face and house segmentation-related response ($t(28) = -0.124$; $p = .902$). Repeated measures ANOVA with stimulus as an additional variable again revealed no interaction between stimulus and group ($F(1,28) = 1.0$; $p = .318$; $\eta^2 = .036$), and no group main effect ($F(1,28) = 0.004$; $p = .953$; $\eta^2 < .000$) on ERP peak amplitude. There was a main effect of stimulus ($F(1,28) = 6.2$; $p = .019$; $\eta^2 = .181$), revealing that the face-homogeneous contrast evoked a larger peak amplitude than house-homogeneous contrast.

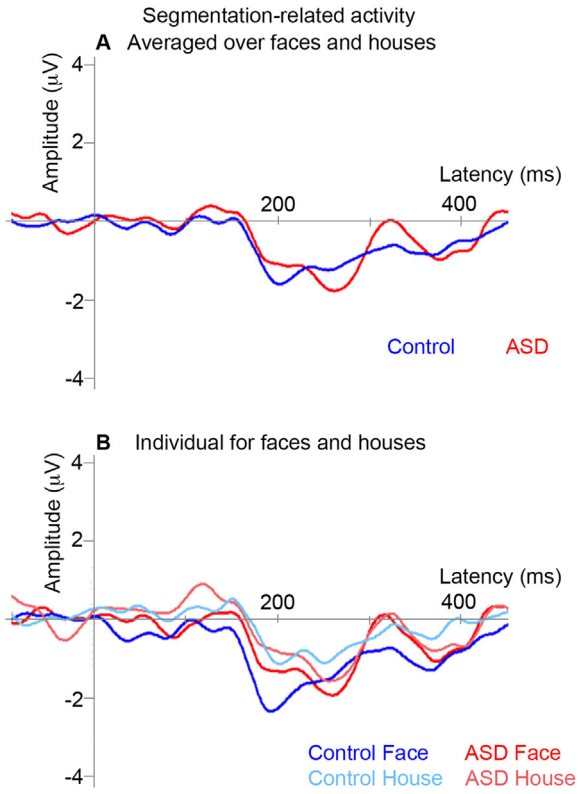

Fig. 2. Grand average ERP responses representing segmentation related brain activity for the Control (blue) and ASD (red) group. Note that grand averages are constructed by averaging the signal amplitude of all participants per timepoint, whereas statistical analyses are conducted on the peak amplitudes and latencies per participant. Consequently, the ERP pattern, peak amplitudes and latencies in the grand average in this figure could differ from the individual patterns in Supplementary materials S1 and the averages reported in Table 2. **A.** Segmentation averaged over faces and houses; **B.** Segmentation calculated individually for face and for house stimuli. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

3.2. Behavior group results

Table 3 provides average and standard errors of behavioral performance. The repeated measures ANOVA on segmentation performance revealed a main effect of stimulus ($F(1,28) = 4.5$; $p = .043$; $\eta^2 = .139$) on performance. Pairwise comparisons revealed that performance was better for face than house stimuli. There was no interaction ($F(1,28) = 2.5$; $p = .125$; $\eta^2 = .082$) or main effect of group ($F(1,28) = 1.6$; $p = .219$; $\eta^2 = .053$). Even though there is no interaction or group effect, the results suggest that the main effect of stimulus is driven by a lower performance on house segmentation in the control group. This lower performance was present in multiple individuals. Exploring the data further, this lower performance was due to both lower hit-rates and higher false alarm rates for house segmentation in the control group than other condition (See Supplementary materials S3).

The repeated measures ANOVA on segmentation reaction times revealed a main effect of group ($F(1,28) = 5.1$; $p = .032$; $\eta^2 = .154$). That is, the ASD group showed a larger difference in response to textured versus homogeneous stimuli than the control group did, suggesting that segmentation takes longer in ASD than control participants. There was no interaction of stimulus and group ($F(1,28) = 1.4$; $p = .254$; $\eta^2 = .046$) or main effect of stimulus ($F(1,28) = 4.0$; $p = .057$; $\eta^2 = .124$). The assumption of normal distribution was

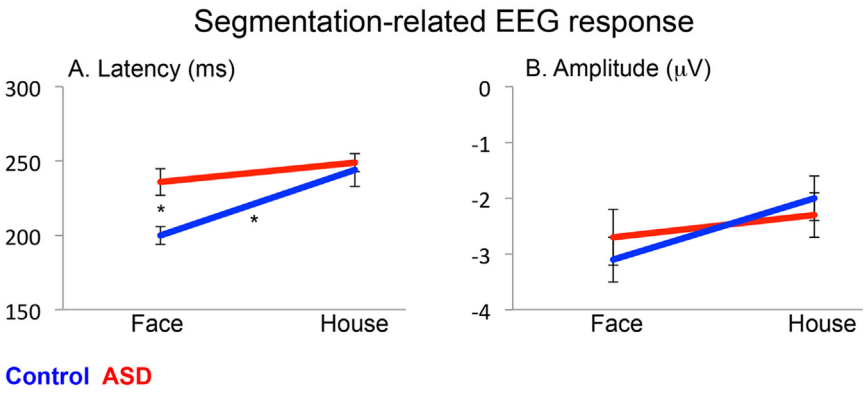

Fig. 3. Segmentation related EEG responses for the Control (blue) and ASD (red) group, individually for face and for house stimuli. **A.** Peak latency, showing significantly faster face-segmentation latency for the controls than ASD group; **B.** Peak amplitude. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

Table 2
Amplitude and latency of the face- and house-segmentation related peaks. Asterisk indicates significant group difference ($p < .01$).

	Face segmentation		House segmentation	
	Control	ASD	Control	ASD
Amplitude (μV)	−3.1 (0.4)	−2.7 (0.5)	−2.0 (0.4)	−2.3 (0.4)
Latency (ms)	200 (6) *	236 (9)	244 (11)	249 (7)

Table 3
Average and standard error of behavioral performance, calculated as hit rate subtracted by false alarm rate, see methods.

	ASD	Control
Performance face (%)	34 (7)	33 (7)
Performance house (%)	31 (7)	18 (4)
Reaction times face minus homogeneous (ms)	162 (45)	64 (26)
Reaction times house minus homogeneous (ms)	225 (50)	81 (28)

violated for the face segmentation reaction time in the control group, which might affect the results from the ANOVA. Therefore, we also compared segmentation reaction times between groups using non-parametric tests. Because no equivalent of the repeated measures ANOVA is available, we performed a Mann-whitney *U*-test per stimulus. This revealed a difference between groups for the house ($p = .009$) but only a trend for the face segmentation reaction times ($p = .066$). For both stimuli, the difference in reaction time between figure versus homogeneous was larger in the ASD than control group, suggesting slower segmentation in ASD.

3.3. Correlation results

Autism Quotient score correlated significantly with latency of the face-segmentation related ERP peak ($r = 0.541$; $p = .002$), and with segmentation behavioral reaction times for both faces ($r = 0.515$; $p = .004$; although not confirmed by non-parametric Kendall's tau test: $r = 0.198$; $p = .125$) and houses ($r = 0.505$; $p = .004$). No other correlations were significant (house ERP latency: $r = 0.012$; $p = .949$; face ERP amplitude: $r = 0.018$; $p = .927$; house ERP amplitude: $r = -0.177$; $p = .350$; face performance: $r = 0.095$; $p = .618$; house performance: $r = 0.234$; $p = .213$). Moreover, the correlation between AQ and peak latency was significantly higher for face- than house-segmentation related responses ($z = 2.872$; $p = .002$). For the other measurements (ERP amplitude, reaction times, and performance) there was no significant difference between the correlations of AQ with face- and house-segmentation (all $p > .1$; calculation performed using the toolbox of Lenhard and Lenhard, 2014). Fig. 5 presents scatterplots of AQ score against the latencies and reaction times. Note that the correlations were no longer significant when correcting for diagnosis by subtracting the group mean from the individual scores.

4. Discussion

The current study investigated whether segmentation of faces is impaired in ASD. Persons with ASD and controls perceived textured

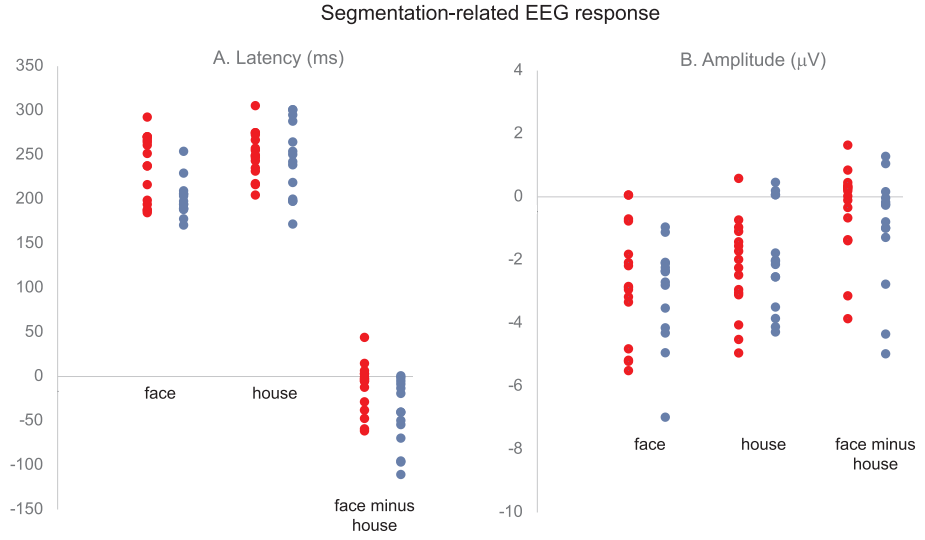

Fig. 4. Segmentation related EEG responses for individuals in the Control (blue) and ASD (red) group, for face and for house stimuli, and the difference in response evoked by face versus that evoked by house stimuli. **A.** Peak latency **B.** Peak amplitude. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

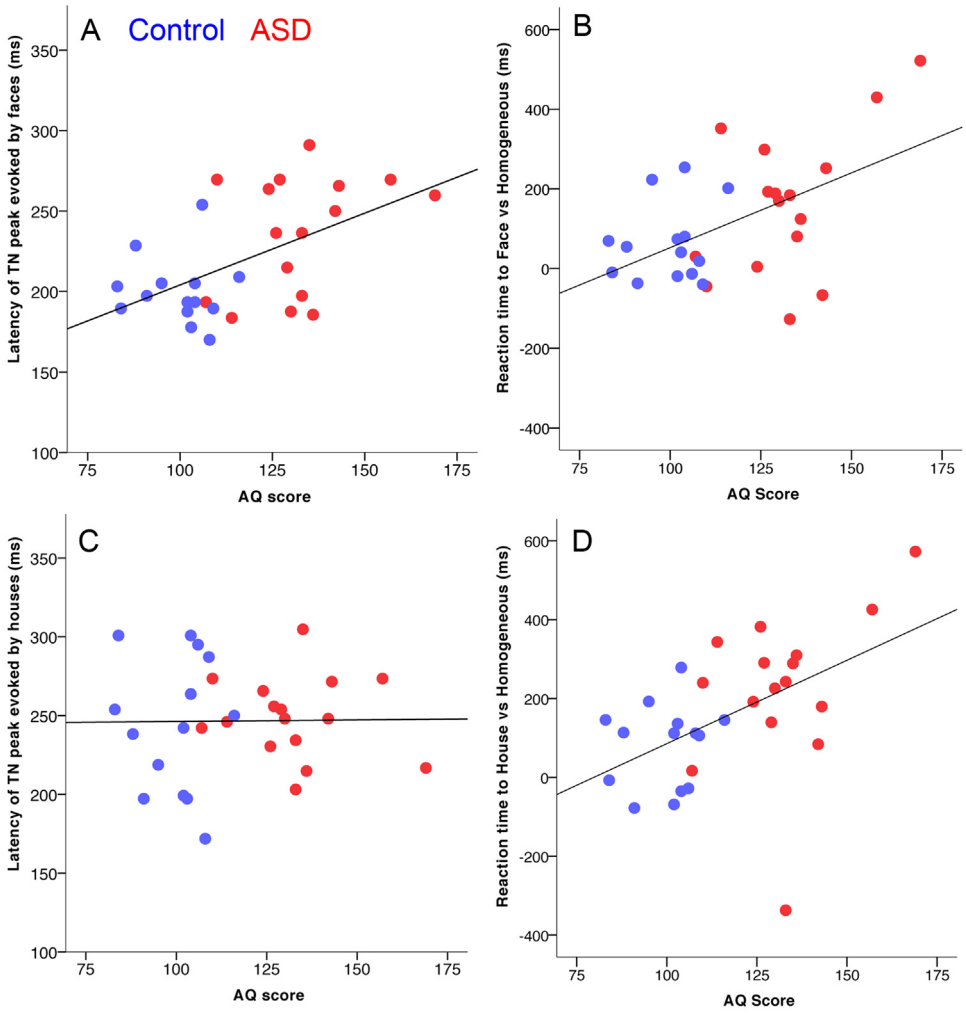

Fig. 5. Scatterplots of AQ score against **A.** Latency of the ERP TN peak evoked by faces. **B.** Reaction time to face versus homogeneous stimuli. **C.** Latency of the ERP TN peak evoked by houses. **D.** Reaction time to house versus homogeneous stimuli. Colors correspond to the Control (blue) and ASD (red) group. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

faces and houses, and homogeneous images. Face-segmentation related brain activity was slower in the ASD than control group. On the contrary, there was no group difference in house-segmentation related activity. Thus, segmentation-related activity is selectively impaired for faces in ASD. Furthermore, behavioral segmentation was delayed in the ASD compared to the control group. This was however not specific to faces and seemed even more evident for houses. Finally, these delays in neural and behavioral responses correlated positively with the degree of ASD traits.

Previous research already revealed delayed processing of photographic faces (McPartland et al., 2004; O'Connor et al., 2005). The current results suggest that delayed segmentation might contribute to delayed face processing. This is in line with the findings by van der Hallen et al. (2015) who showed slower integration of local details into a global percept, a crucial step in segmentation. Because previous fMRI research, using the same stimuli as the current study, showed that face segmentation relates to recurrent connectivity from the FFA to lower visual areas (Fahrenfort et al., 2012), the current results suggest that activity in these connections is abnormal. Specifically, recurrent connections seem to be activated later, but not less, in ASD than in typical individuals. Previous findings already revealed decreased connectivity within the FFA (Khan et al., 2013; Kleinhans et al., 2008). Adding the current results to this literature, it can now be suggested that connectivity is atypical both within the FFA and from the FFA to lower visual areas. This could contribute to delayed face processing in adults with ASD.

Interestingly, the correlation between face segmentation-related activity and ASD traits suggests that variations in basic visual processing relate to individual differences in ASD phenotype. This supports the theory that atypical basic visual perception plays an important role in ASD symptoms (e.g. Dakin and Frith, 2005; Simmons et al., 2009). Previous research already related autistic traits to behavioral aspects of face processing, such as the reading the eyes in the mind test (Baron-Cohen et al., 2001b) and adaptive coding of faces (specific for males; Rhodes et al., 2013). In addition, a study using functional Near Infrared Spectroscopy (fNIRS) revealed a correlation between AQ and activation near the STS during social interaction (Suda et al., 2011). The current correlation shows that individual differences can already be observed at a basic visual level, which might contribute to the previously revealed differences in higher-level face perception. However, the correlations where no longer present when corrected for diagnosis, indicating that the group differences explain a significant part of the correlation. This is to be expected, as the AQ (used to measure ASD traits) is designed to investigate ASD symptoms and thus to be affected by diagnosis. Nevertheless, the correlation between autism traits and EEG or behavioral test results provides a more subtle view on the effect of ASD on segmentation. The higher someone scores on the AQ, and thus possibly the more severe the ASD symptoms, the slower someone is in face segmentation. In addition ASD and control participants that are close to each other in symptomatology based on the AQ, but respectively did or

did not receive a diagnosis, are more similar in segmentation than ASD participants with severe symptoms versus controls with very few symptoms. The current correlations therefore support that ASD symptoms form a continuum rather than a binary phenomenon, and indicate that this is also the case for neurocognitive and behavioral impairments.

Combining the current results with previous findings on segmentation in ASD, we suggest that there are developmental changes in the type of objects that are atypically segmented in ASD. That is, early in life segmentation is atypical for complex objects but typical for basic geometric forms, developing into specific temporal deficits for segmentation of social stimuli but typical segmentation of other basic and complex objects (Evers et al., 2014; Kemner et al., 2007; Vandenbroucke et al., 2008, 2009). No current models can sufficiently explain this developmental pattern. Here, we propose factors that might affect the atypical development of face segmentation. The first two factors are the activity in a specific brain area and recurrent connectivity from this area, which both relate to the presence of segmentation (Hochstein and Ahissar, 2002; Lamme and Roelfsema, 2000). Activity in face processing areas and recurrent connectivity from these areas seem indeed atypical in ASD (e.g. McPartland et al., 2004; O'Connor et al., 2005; Scherf et al., 2010). Another factor is the specialization over development of areas that process faces or objects, which affects the response of an area to a specific stimulus. Research on face specialization in ASD showed a small but significant specialization in the FFA, but decreased specialization in the amygdala and medial prefrontal cortex which both interact with the FFA (Joseph et al., 2015). As such, face specialization seems atypical in ASD as well. Of importance is also myelination, which affects processing speed. If myelination of axons from the FFA to lower level visual areas would be decreased, this would lead to slower face processing. To our current knowledge, it is unknown whether persons with ASD show this specific decrease in myelin. Several studies suggest atypical myelination in other brain areas, although they differ in whether myelin is decreased (e.g. Ke et al., 2009; Lee et al., 2007) or increased (Ben Bashat et al., 2007; Cheng et al., 2010). Thus, it is unknown to what extend atypical myelination contributes to delayed face segmentation in ASD. Future research should reveal the interacting contribution of each of these factors to the atypical development of face and object perception. Then, the factors can be combined in a model on the atypical development of face segmentation in ASD.

The non-specific or even house-specific behavioral segmentation delay does not support the hypothesis that impairments are specific to faces. These results also oppose the ERP findings that impairments are face-specific. However, they are in line with the findings from a meta-analysis on behavioral studies that integration of local elements is delayed in ASD (van der Hallen et al., 2015). This meta-analysis only included non-face stimuli. It might thus be the case that persons with ASD behaviorally show slower segmentation for a wide range of complex visual objects, while neurocognitive measurements possibly indicate major impairments in face segmentation. Several factors could contribute to this discrepancy. Because face segmentation occurs early in the processing stream from perception to the action of responding, it is possible that other mechanisms such as motor preparation and decision making contributed to the behavioral delay. In addition, differences in task of the participant (i.e. object categorization during the behavioral and a non-object related ellipse task during the EEG measurement) could have evoked differences in perceptual processes or allocation of attention, which might partly explain the dissociation as well. Future studies should investigate which mechanisms contribute to differences in behavioral and neural segmentation. Nevertheless, both neural and behavioral results indicate that segmentation is impaired in the temporal domain in ASD.

One might suggest that the observed responses not only reflect segmentation, but categorization as well. It is likely that there is an interaction between segmentation and categorization processes for second-order objects such as the textured faces and houses. However, the current ERP activity most likely reflects segmentation. The activity is very similar in latency and pattern to the segmentation-related but not to the category-selective activity described in a previous study using the same design (van den Boomen et al., 2015). That is, textured objects evoke segmentation-related activity that peaks around 200 ms after stimulus onset and category-selective activity around 300 ms after stimulus. The category-selective activity is much later when evoked by textured objects than by photographic objects (usually around 170 ms after stimulus onset) and only occurs when stimuli are presented for a longer duration than in the current study (i.e. for 800 ms instead of 92 ms; see van den Boomen, 2015 for an in-depth comparison). This suggests that the here-described responses mainly reflect segmentation. Moreover, the comparison by van den Boomen (2015) highlights that textured faces are processed differently than real-life faces. This limits generalization to previous studies using photographic stimuli. Instead, this study adds to previous studies that under suboptimal conditions, i.e. when face categorization might be difficult, segmentation is delayed in persons with ASD. In addition, while interpreting the results, one should keep in mind that in some participants, segmentation-related brain activity was very small (i.e. no clear ERP peaks were present) or was present over a longer timeframe with more than one ERP peak. Particularly the absence of an ERP peak is relevant, as it suggests that faces or houses were not segmented. However, this only occurred in few participants and even though this was observed more often in the ASD group, on average this group did not differ in amplitude from the control group. Moreover, excluding these participants did not affect the results.

In conclusion, the current study revealed that persons with ASD show delayed segmentation of faces. Segmentation is an important process leading to face perception. We show that already at this early step in face perception, there is a delay in processing that is specific to faces. As such, delayed segmentation can be seen as one of the mechanisms contributing to delayed face processing. Furthermore, face segmentation reflects functional recurrent connectivity from the FFA to lower visual areas. Adding the current results to previous findings, we might now suggest that atypical connectivity within and from the FFA to the visual cortex contributes to delayed face processing in ASD.

CRediT authorship contribution statement

C. van den Boomen: Conceptualization, Methodology, Software, Data curation, Formal analysis, Investigation, Methodology, Project administration, Visualization, Writing - original draft, Writing - review & editing. **J.J. Fahrenfort:** Conceptualization, Methodology, Writing - review & editing. **T.M. Snijders:** Conceptualization, Methodology, Writing - review & editing. **C. Kemner:** Conceptualization, Resources, Writing - review & editing, Funding acquisition, Supervision.

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Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.neuropsychologia.2019.02.005.

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