

The N170 is not modulated by attention in autism spectrum conditions

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Face processing deficits are characteristic of autism spectrum conditions. However, event-related potential studies of autism spectrum conditions have found inconsistent results for the face selective N170 component. In this study, 15 adult males with autism spectrum conditions and 15 matched, typically developing controls completed a task in which pictures of faces were either attended to or ignored. In the control group, the N170 was larger when faces were attended to. However, there was no such modulation in the autism spectrum conditions group. This finding helps clarify the results from the earlier eventrelated potential studies of face processing in autism spectrum conditions and suggests that visual attention does not enhance face processing in autism spectrum conditions as it does in typical development. *NeuroReport* 00:000–000 © 2010 Wolters Kluwer Health | Lippincott Williams & Wilkins.

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Introduction

Problems processing information from faces are frequently found in behavioural studies of people with autism spectrum conditions. Deficits are found in the recognition of unfamiliar faces [1], in the recognition of familiar faces [2] and in the labelling of facial emotions [3]. Given this, event-related potential (ERP) studies might be expected to show that people with autism spectrum conditions have a decreased amplitude of the face selective N170 component [4]. However, results from such studies have been mixed, as detailed in a recent review [5]. This inconsistency may be because of the different tasks used in these ERP studies. When participants attend to faces because task demands are high, the anticipated difference in N170 amplitude between participants with and without autism spectrum conditions is found [6]. However, when participants do not attend carefully to faces because the task demands are low [7] or because their attention is deliberately directed away from faces [8] then this difference is not seen.

ERP studies of typically developing adults in which visual attention has been manipulated have shown that the amplitude of the N170 component is affected by attention. Specifically, the N170 is larger to faces when they are the attended to category in a stream of different objects [9]. Positron emission tomography suggests that this enhancement of early visual processing relies on connections between the frontal and parietal sites [10].

However, there is evidence from the functional MRI literature that this attentional enhancement of face

processing systems is reduced in autism spectrum conditions [11]. This is consistent with findings of weaker connectivity between distant brain regions in autism spectrum conditions [12] and ERP studies of patients with lesions to the prefrontal cortex show that early ERP components such as the N170 are reduced when this attentional enhancement from the prefrontal cortex is not available [13].

To the authors' knowledge, this is the first ERP study of autism spectrum conditions in which attention to faces is experimentally controlled. We predicted that the task instructions would have a different effect on the N170 in the two groups. Specifically, we predicted that the N170 would be larger when attention was directed at the faces but that this increase would be larger in the control group than in the autism spectrum condition group.

Methods

Participants

The School of Psychology Research Ethics Board at the University of Cambridge approved this methodology. Electroencephalography was recorded from 30 right handed, male participants (15 autism spectrum conditions, 15 control). Exclusion criteria for autism spectrum conditions participants were an uncorrected impairment in eyesight or hand movement, a personal or family history of any psychological or genetic disorder apart from an autism spectrum conditions and a period of unconsciousness in the last 5 years. Exclusion criteria for control participants were the same points with the addition of a self or family history of autism spectrum conditions. All autism spectrum conditions participants were diagnosed with Asperger syndrome according to the international criteria (*Diagnostic and Statistical Manual of Mental Disorders-IV-TR*, [14]) by a professional experienced with the diagnosis of autism spectrum conditions.

Before participating, participants completed the Autism-Spectrum Quotient questionnaire (AQ; [15]) and Wechsler Adult Intelligence Scale (3rd UK edition; [16]). The groups were matched on age (autism spectrum conditions M = 31.4 years SD = 6.7, control M = 29.3 years SD = 4.6) and IQ (autism spectrum conditions M = 119.3 SD = 13.4, control M = 118.9 SD = 13.6). Higher scores on the AQ reflect a greater number of traits indicative of autism spectrum conditions and the autism spectrum conditions group scored significantly higher on the AQ (M = 35.3, SD = 7) than the control group [M = 15.7, SD = 6.8, t(27) = 7.57, P < 0.001].

Procedure

Participants were seated in a darkened room approximately 60 cm from the monitor on which the stimuli were presented. The stimuli consisted of 30 neutral faces (15 male and 15 female) from the NimSTIM database [17] and 30 chairs from Photo Clip Art by Hemera. All stimuli were edited in Photoshop CS3 (*www.adobe.com*), transformed to grayscale, mounted on a white background, equated for average luminance and contrast and resized to 5×7 cm.

Participants viewed two blocks of stimuli between which only the order of the images varied. In each block, all faces and chairs were repeated three times pseudorandomly. In each block, ten faces (five male and five female) and ten chairs were randomly selected and were inserted as immediate repetitions. No other stimuli were immediately repeated. All stimuli subtended $5.1 \times 7.3^{\circ}$ of visual angle and were presented for 500 ms with an interstimulus interval that varied randomly between 1200 and 1400 ms. Participants rested for approximately 5 min between the blocks.

At the start of each block, participants were asked to attend to one of the stimulus categories (either faces or chairs) and to press a response button when they saw an immediate repetition of the same stimuli in that category. Importantly, participants were told that all stimuli in the other category, including immediate repetitions, were to be ignored.

Each block began with a practice run of ten stimuli (including two immediate repetitions). The order of the two blocks, the attended category and the hand used to respond with were counterbalanced across participants. Total testing time was approximately 15 min. The stimulus train is illustrated in Fig. 1.

Fig. 1



Example stimulus trains for the two conditions. Stimuli that required a button press from the participant are shown here underlined and stimuli used to form the averages in each condition are shown here in boxes.

Electrophysiology

Electroencephalography was recorded from 32 electrodes using a modified Quickcap 10/10 system (Compumedics Neuroscan, Charlotte, North Carolina, USA). Reference was at the tip of the nose and ground at FPZ. Vertical and horizontal eye movements were recorded in bi-polar channels with electrodes above and below the left eye (vertical electrooculogram) and 1 cm from the outer canthus of each eye (horizontal electrooculogram). Impedances at all sites were maintained below 5 k Ω . All channels were recorded using a Synamps amplifier (Compumedics Neuroscan), which sampled the analogue signal at 1000 Hz with a bandpass filter between 0.1 and 100 Hz.

Of the original sample, 12 control and 13 autism spectrum conditions participants provided at least 55 movement and α free trials for averaging and were included in the final analysis. Offline, eye blink artefact was corrected using an eye movement subtraction algorithm [18]. Epochs were formed for the period from 100 before to 900 ms after the presentation of each face stimulus that was not an immediate repetition. These were baseline corrected against the prestimulus interval. Separate average epochs were formed for the faces when they were attended to and when they were ignored. Average waveforms were lowpass filtered at 20 Hz (24 dB) using a zero-phase shift finite impulse response filter.

The mean amplitude of the N170 component was calculated for the window 130–190 ms post stimulus onset. This interval was chosen to correspond with the waveforms across hemisphere, condition and group and with the interval used by Eimer [9]. Peak latency was calculated as the time from stimulus onset to the minimum (most negative) point within this same window. Epochs were visually inspected to ensure that this represented a true local minimum. Statistical

analyses were conducted for the sites at which the N170 was maximal: P7 (left hemisphere) and P8 (right hemisphere). The analysis consisted of a mixed threeway analysis of variance with hemisphere (left, right), attention (attend to faces, ignore faces) and group (autism spectrum conditions, control) as factors and mean amplitude and peak latency as outcome measures. Only interactions involving the between patients factor of group, which were necessary to address the hypothesis, are reported here.

Results

Behavioural results

The number of targets in both the conditions (either immediately repeated faces or immediately repeated chairs) that participants correctly identified (out of the maximum of 10) was investigated as a measure of performance. There was no significant difference between the number of correctly identified faces and chairs in either the autism spectrum condition group (faces median (Mdn) = 9, chairs Mdn = 10, T = 3.5, P = 0.07) or the control group (faces Mdn = 10, chairs Mdn = 10, T = 2.5, P = 0.32).

Electrophysiological results

For mean amplitude, there was no main effect of hemisphere [F(1,23) = 0.96, P = 0.34] or interaction between hemisphere and group [F(1,23) = 0.31, P = 0.58]. Subsequent analyses were conducted across hemispheres. There was no main effect of group across the levels of attention [F(1,23) = 0.74, P = 0.4]. However, as predicted, there was a significant interaction between group and attention [F(1,23) = 5.81, P = 0.02] and a significant main

effect of attention [F(1,23) = 16.43, P < 0.001] with a larger (more negative) amplitude to faces when attention was directed at them $(M = -0.61 \,\mu\text{V}, \text{SD} = 2.74)$ than when faces were ignored $(M = 0.45 \,\mu\text{V}, \text{SD} = 2.72)$.

The statistical interaction between group and attention was further investigated with paired *t*-tests for each group of participants on attention. As predicted, this showed that there was a significant difference in the N170 amplitude to faces in the control participants because of attention, with a larger amplitude when attention was directed at the faces $(M = -1.44 \,\mu\text{V}, \text{SD} = 3.06)$ than when faces were ignored $(M = 0.32 \,\mu\text{V}, \text{SD} = 2.92, t(11) = -4.76, P = 0.001)$. However, as shown in Fig. 2, there was no significant difference in the N170 amplitude to faces in autism spectrum conditions participants between the attention conditions [t(12) = -1.13, P = 0.28].

As with the results for the amplitude of the N170, there was no main effect of hemisphere on peak latency [F(1,23) = 0.2, P = 0.66] or interaction between the hemisphere and group [F(1,23) = 1.15, P = 0.3]. However, unlike the results for amplitude, there was no main effect of attention on latency [F(1,23) = 2.51, P = 0.13] or interaction between the group and attention [F(1,23) = 0.07, P = 0.8].

Discussion

This study aimed to examine whether the enhancement that directed visual attention gives to the N170 ERP component is present in patients with autism spectrum conditions to the same extent that it is in controls. The results show that while the amplitude of the N170



Grand average waveforms for the face stimuli in the left hemisphere (P7) and right hemisphere (P8). ASC, autism spectrum condition.

increased in the control group when attention was directed at faces, this increase was absent in the autism spectrum condition group.

The modulating effect of attention in the typically developing group is consistent with previous ERP studies. In particular it replicates the findings of Eimer [9] who used the same one-back task to load attention as was used here. The lack of attentional enhancement of the N170 component in the autism spectrum conditions group in this study is also consistent with functional MRI results [11] which show that face selective brain regions are not modulated by attention in autism spectrum conditions participants.

Unlike the results for amplitude, there was no effect of attention on the peak latency of the N170. This is inconsistent with an earlier ERP study [19], which found that when faces were attended to, the N170 peaked earlier. However, this earlier study used a different experimental design in which faces always occurred at the same points in the stimulus train (i.e. face then landscape, then face, then landscape, then test image). As participants knew when a face would be the next image, attentional resources could be engaged earlier.

The behavioural results showed that there was no difference between the number of correctly identified faces and chairs in either the autism spectrum condition group or the control group. This is inconsistent with earlier finding of impaired recognition of unfamiliar faces but normal recognition of other unfamiliar objects in autism [1]. However, the lack of significance between the conditions should be treated with caution because there was a strong negative skew in the distribution.

Our research supports a body of literature on face processing in autism spectrum conditions, which has seen some initially contradictory results explained by the addition of attention as a factor. In the functional MRI literature, the fusiform gyrus was initially reported as being hypoactive in people with autism spectrum conditions [20] but was subsequently found to be equivalent [21]. A recent review [22], suggested that the difference between these results is explained by the use of a fixation cross in the second study which reduced the attention of the typically developing participants to the faces and hence reduced their apparent fusiform gyrus activation to the level shown by participants with autism spectrum conditions. Source localization suggests that the fusiform gyrus is a likely generator of the N170 [23] and this study shows that the same effect is evident in ERP studies. That is, the reduced N170 amplitude in autism spectrum conditions is only evident when attention is directed at faces because under these conditions, typically developing people show an increased N170.

The ability to add additional neural resources to the processing of faces at 170 ms post stimulus onset would be of great value in social interaction. Attention enhances

the influence of relevant stimuli at further processing stages while diminishing the influence of irrelevant stimuli [24]. Furthermore, the biased competition model of attention [25] suggests that stimuli which do not engage with attentional systems are not available to subsequent memory and motor systems. Thus, a lack of attentional enhancement of the N170 may contribute to the impairments in unfamiliar face recognition [1], familiar face recognition [2] and emotional facial expression [3] found in autism spectrum conditions.

Conclusion

In summary, this study has shown that attention modulates the N170 in typically developing people but not in people with autism spectrum conditions. This suggests that a lack of face processing enhancement because of attention is a possible explanation for the behavioural findings of impaired face perception in autism spectrum conditions.

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References

- 1 Boucher J, Lewis V. Unfamiliar face recognition in relatively able autistic children. *J Child Psychol Psychiatry* 1992; **33**:843–859.
- 2 Boucher J, Lewis V, Collis G. Familiar face and voice matching and recognition in children with autism. J Child Psychol Psychiatry 1998; 39:171-181.
- 3 Tantam D, Monaghan L, Nicholson H, Stirling J. Autistic children's ability to interpret faces: a research note. J Child Psychol Psychiatry 1989; 30:623–630.
- 4 Bentin S, Allison T, Puce A, Perez E, McCarthy G. Electrophysiological studies of face perception in humans. *J Cogn Neurosci* 1996; 8:551–565.
- 5 Jemel B, Mottron L, Dawson M. Impaired face processing in autism: fact or artifact? J Autism Dev Disord 2006; **36**:91–106.
- 6 O'Connor K, Hamm JP, Kirk IJ. The neurophysiological correlates of face processing in adults and children with Asperger's syndrome. *Brain Cogn* 2005; **59**:82–95.
- 7 O'Connor K, Hamm JP, Kirk IJ. Neurophysiological responses to face, facial regions and objects in adults with Asperger's syndrome: an ERP investigation. *Int J Psychophysiol* 2007; **63**:283–293.
- 8 McPartland J, Dawson G, Webb SJ, Panagiotides H, Carver LJ. Event-related brain potentials reveal anomalies in temporal processing of faces in autism spectrum disorder. *J Child Psychol Psychiatry* 2004; **45**:1235–1245.
- 9 Eimer M. Attentional modulations of event-related brain potentials sensitive to faces. *Cognitive Neuropsychol* 2000; **17**:103–116.
- 10 Corbetta M, Miezin FM, Shulman GL, Petersen SE. A PET study of visuospacial attention. *J Neurosci* 1993; **13**:1202–1226.
- 11 Bird G, Catmur C, Silani G, Frith C, Frith U. Attention does not modulate neural responses to social stimuli in autism spectrum disorders. *Neuroimage* 2006; **31**:1614–1624.
- 12 Just MA, Cherkassky VL, Keller TA, Minshew NJ. Cortical activation and synchronization during sentence comprehension in high-functioning autism: evidence of underconnectivity. *Brain* 2004; **127** (Pt 8):1811–1821.

- 13 Barcelo F, Suwazono S, Knight RT. Prefrontal modulation of visual processing in humans. *Nat Neurosci* 2000; **3**:399–403.
- 14 American Psychiatric Association. *Diagnostic and statistical manual of mental disorders*. 4th ed. Washington, DC: American Psychological Association; 2000.
- 15 Baron-Cohen S, Wheelwright S, Skinner R, Martin J, Clubley E. The autismspectrum quotient (AQ): evidence from Asperger syndrome/highfunctioning autism, males and females, scientists and mathematicians. *J Autism Dev Disord* 2001; **31**:5–17.
- 16 Wechsler D. Wechsler adult intelligence scale. London: Harcourt Assessment; 1999.
- 17 Tanaka JW, Leon AC, McCarry T, Nurse M, Hare TA, Marcus DJ, et al. The NimStim set of facial expressions: judgments from untrained research participants. *Psychiatry Res* 2008; **168**:242–249.
- 18 Semlitsch HV, Anderer P, Schuster P, Presslich O. A solution for reliable and valid reduction of ocular artifacts, applied to the P300 ERP. *Psychophysiology* 1986; 23:695–703.
- 19 Gazzaley A, Cooney JW, McEvoy K, Knight RT, D'Esposito M. Top-down enhancement and suppression of the magnitude and speed of neural activity. J Cogn Neurosci 2005; 17:507–517.

- 20 Schultz RT, Gauthier I, Klin A, Fulbright RK, Anderson AW, Volkmar F, et al. Abnormal ventral temporal cortical activity during face discrimination among individuals with autism and Asperger syndrome. Arch Gen Psychiatry 2000; 57:331–340.
- 21 Hadjikhani N, Joseph RM, Snyder J, Chabris CF, Clark J, Steele S, *et al.* Activation of the fusiform gyrus when individuals with autism spectrum disorder view faces. *Neuroimage* 2004; 22:1141–1150.
- 22 Klin A. Three things to remember if you are a functional magnetic resonance imaging researcher of face processing in autism spectrum disorders. *Biol Psychiatry* 2008; **64**:549–551.
- 23 Deffke I, Sander T, Heidenreich J, Sommer W, Curio G, Trahms L, *et al.* MEG/EEG sources of the 170-ms response to faces are co-localized in the fusiform gyrus. *Neuroimage* 2007; **35**:1495–1501.
- 24 Pessoa L, Kastner S, Ungerleider LG. Neuroimaging studies of attention: from modulation of sensory processing to top-down control. *J Neurosci* 2003; 23:3990–3998.
- 25 Desimone R, Duncan J. Neural mechanisms of selective visual attention. Annu Rev Neurosci 1995; 18:193–222.

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