AN ERP SOURCE LOCALIZATION STUDY OF PERFORMANCE MONITORING IN AUTISM SPECTRUM DISORDER

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Background

Autism spectrum disorder (ASD) is characterized by a wide range of cognitive and social impairments including monitoring, joint attention, theory of mind and initiation/modification of social behaviors [1]. Efficient response monitoring may be essential to the learning and responding to social cues as well as evaluating the reward value of social stimuli [2,3,4]. Converging evidence points to structural and functional abnormalities of the anterior cingulate cortex (ACC), particularly the dorsal ACC (dACC), underlying these impairments. The error-related negativity (ERN) and correct-response negativity (CRN) have been widely used as an indices of ACC-related response monitoring. Previous studies have shown that the ERN was reduced in adolescents with ASD compared with controls [5] and, in ASD children, larger ERNs predicted fewer social impairments [6]. To date, there are no studies examining the ERN in adults with ASD and/or in relation to social impairments.

Goal: To examine the neural activation associated with error processing in relation social functioning in high functioning ASD adults and healthy adults while performing a flanker task. We examined the ERN, CRN and Pe and LORETA current source density underlying the ERN in regions associated with error responses.

Hypotheses: Based on previous research, we expected adults with ASD to show diminished ERN and Pe amplitudes following errors and poor differentiation between error and correct responses. Moreover, ASD adults were expected to show a reduction in dACC error-related activity, and this activity would be predictive of social impairment.

Flanker Task

200 ms — 0 – 3000 ms — 200 ms

Close incongruent — Far congruent

- Participants were presented with an array of three digits and were asked to identify the centre number by key press, using a left key if the centre number was 3, and a right key for the number 4.
- There were two congruency conditions and two distance conditions.
  - For congruent trials, the flanking numbers matched the centre number (e.g., 333, 444); for incongruent trials, the flanking numbers differed from the centre number (e.g., 343, 434).
  - For close trials, the number arrays were presented in close configuration; for far trials, number arrays were in a more widely spaced configuration.

Method

Participants

- 15 high-functioning adults with ASD (12 men, Mean age 35.5 yrs ± 7.6; IQ: 64 - 136) and 16 healthy adults (12 men, Mean age 35.7 yrs ± 10.6; IQ: 88 - 121).

Self-report measure of social functioning

- Autism-Spectrum Quotient (AQ): social skills, communication skills, imagination, attention to detail, and attention switching/tolerance of change[ref].

EEG recording and reduction

- EEG was recorded continuously during the flanker task using a 128-channel sensor array.
- ERP epochs were extracted -200 to 800 ms following error and correct responses at Fz, FCz, and Cz where the ERN, CRN and Pe were scored.
- Epochs were filtered at 1 to 20 Hz and a -200 to 0 ms pre-response baseline was used.

Low Resolution Electromagnetic Tomography (LORETA)

- The intracerebral current density underlying the ERN in Brodmann areas 24 and 32 was examined using LORETA.
- Current density was computed within a 50 to100 time window which captured the mean latency of the ERN (77 ms).

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ERP Results

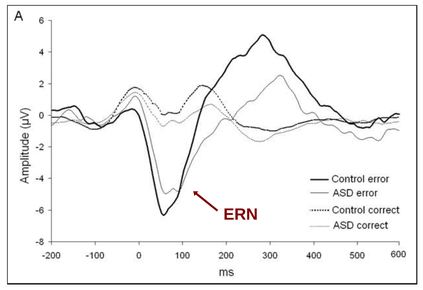

Figure 1. Averaged ERP waveforms for error and correct trials.

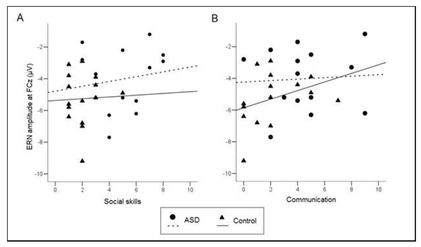

Figure 2. Scatter plot of the ERN at FCz and (A) AQ social skills and (B) AQ communication for ASD and controls

- A main effect for Accuracy [$F(1,28) = 130.66$, $p < .001$] indicated that the ERN was larger than the CRN across groups at FCz, Figure 1.
- An Accuracy by Group interaction [$F(1,28) = 4.81$, $p = .037$] indicated that the ERN was smaller for the ASD group compared with the control group [$t(28) = 2.11$, $p = .04$], with no difference between groups for the CRN [$t(28) = .69$, $p > .49$], Figure 1.
- A CRN minus ERN difference score revealed that the difference between components was smaller for the ASD compared to the control group [$t(28) = 2.19$, $p = .037$].
- There were no differences in the Pe between groups ($p = .23$).
- Better social ($r = .31$, $p = .05$) and communication ($r = .31$, $p = .05$) skills were related to larger ERNs, Figure 2A and B.
- Social/communication skills accounted for 11.2% of the variance in the ERN; group differences did not remain after accounting for social/communication skills; the direction of the relation was similar in both groups.

LORETA Source Localization

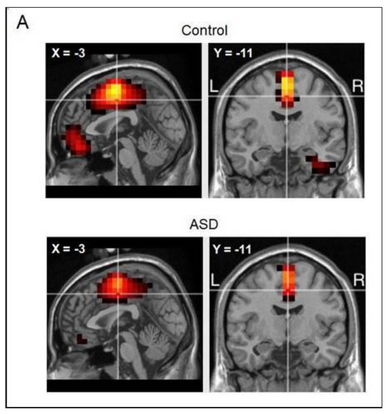

Figure 3A. Areas of error activation for controls and ASD adults.

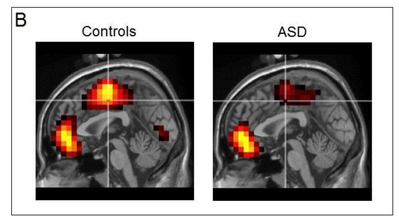

Figure 3B. Error minus correct for controls and ASD adults.

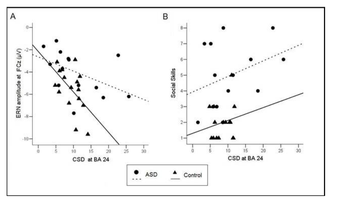

Figure 4. Scatterplot for the Pearson correlation between LORETA current density in the dACC (log A/m^2) and (A) the ERN and (B) social skills.

- Controls had higher activity in the ACC (BA 24) than ASD adults, Figure 3A.
- Figure 3B shows the difference between controls and ASD adults in BA 24 activity for error minus correct trials; similar activity between groups was seen in the prefrontal cortex regions (BA 10/11).
- As shown in Figure 4A, more negative ERNs were related to higher current density in BA 24 ($r = -.37$, $p = .04$); while better social skills was related to higher current density in BA 24 for both groups.

Conclusions

- Adults with ASD showed reduced ERN amplitudes and although the ERN was localized to the dorsal region of the ACC (BA 24) for both groups, activity in this region was reduced for individuals with ASD.
- Adults with ASD also showed less differentiation between error and correct ERP components.
- We found that poor social and communication skills was related to reduced ERNs and dACC hypoactivity.
- These findings are the first to link reduced ACC activity to social deficits in adults exhibiting autistic traits and suggest that reduced ACC activity may reflect a putative brain mechanism involved in the origins and maintenance of social impairments in ASD.

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