Dissociating Social Functioning in ASD and Schizophrenia using Clinical Assessment and Neural Response to Gaze Cues
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Background
Social difficulties, including deficits in maintaining and interpreting social gaze and in recognizing faces and emotions, are hallmark features of autism spectrum disorder (ASD). Atypical social functioning and gaze processing are not, however, unique to ASD. Both are also affected in schizophrenia (SZ), a disorder with genetic, neurobiological, and phenotypic commonalities with ASD. This study utilized novel methods, integrating eye-tracking and electrophysiology (EEG), to study social behavior and brain function during simulated face-to-face interactions in individuals with ASD and SZ.

Specifically, we evaluated N170 and P300 response to direct and averted gaze in adults with ASD, SZ, and typical development (TD) to determine between-group differences in gaze, eye movement, and processing patterns in neural processes associated with face decoding. In parallel, we evaluated social functioning with a battery of diagnostic, clinical, and neurocognitive assessments. In this way, we evaluated whether specific abnormalities in gaze processing differed by diagnostic category or were general indicators of social dysfunction across neurodevelopmental disorders.

Objective: To evaluate relations among social function and dysfunction and neural markers of gaze processing during simulated face-to-face interactions in individuals with ASD, SZ, and TD controls.

Method

Participant Demographics

<table>
<thead>
<tr>
<th></th>
<th>N</th>
<th>Sex</th>
<th>Age (SD)</th>
<th>FSIQ* (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>ASD</td>
<td>17</td>
<td>13M</td>
<td>21.89 (3.81)</td>
<td>102.56 (19.29)</td>
</tr>
<tr>
<td>SZ</td>
<td>14</td>
<td>13M</td>
<td>28.81 (8.30)</td>
<td>95.79 (11.56)</td>
</tr>
<tr>
<td>TD</td>
<td>13</td>
<td>9M</td>
<td>28.80 (7.69)</td>
<td>105.42 (20.47)</td>
</tr>
</tbody>
</table>

Experimental Paradigm:
- Participants were presented with 80 distinct photorealistic, animated faces matched for low-level visual features.
- Contingent upon participants fixing on the face, stimuli responded by shifting eye gaze (either from direct to averted or averted to direct).

Clinical Measures:
To measure social and perceptual difficulties, participants completed:
- **ASD diagnostic assessment**: Autism Diagnostic Observation Schedule
- **SZ diagnostic assessment**: Structured Clinical Interview for DSM Diagnosis; Positive and Negative Syndrome Scale
- **ASD self-report measure**: Social Responsiveness Scale; Broad Autism Phenotype Questionnaire; Autism-Spectrum Quotient
- **SZ self-report measure**: Schizotypal Personality Questionnaire
- **Behavioral assessments**: Benton Facial Recognition Test

EEG and ET Data Acquisition and Collection:
- EEG recorded at 1000 Hz with a 128-electrode cluster rereferenced to vertex and ET data collected using an ElectroCap (Electrical Geodesics, Eugene OR) head cap. Analysis was performed using EEGLAB, a toolbox for processing and analyzing EEG data.

ERP Preprocessing and Analysis:
- Significant ERP differences were detected to gaze direction for each group (ASD, SZ, TD) and for each condition (direct, averted) using a repeated-measures ANOVA with group and condition as factors.

Results

1. **Clinical Measures**
- ASD presented a range of difficulties, including social communication and interaction, repetitive behaviors, and sensory sensitivity.
- SZ presented a range of difficulties, including social cognition and motor planning, and attention.

2. **Neural Measures**
- Significant differences were detected in the N170 and P300 response to direct and averted gaze in ASD, SZ, and TD.
- ASD had an increased N170 response to direct gaze compared to averted gaze.
- SZ had a decreased P300 response to direct gaze compared to averted gaze.

Conclusions
In line with a dimensional approach to understanding neurodevelopmental disorders, preliminary results of this study suggest that neural response to gaze-contingent shifts in eye gaze is a reliable marker of social dysfunction across individuals with ASD and SZ.
- **N170 latency** is related to social function and dysfunction. Across groups, those with better face perception skills during clinical assessment had faster N170 latencies, whereas those with greater ASD symptomatology had more delayed N170 to gaze cues.
- **N170 amplitude** did not differ significantly between groups, suggesting that amplitude may be a less sensitive marker of social dysfunction.

In contrast to ERP markers, clinical measures of ASD and SZ symptomatology are variably effective in differentiating the two clinical populations. Specifically, though self-report measures are reliable in differentiating clinical populations from TD, they are ineffective in differentiating between diagnostic categories.

These preliminary findings suggest that, across multiple neurodevelopmental disorders, neural indices of social processing can reveal differences in gaze processing related to clinically-relevant social difficulties that behavioral measures of overt symptomatology do not capture.