Impact of Cooccurring Conditions on EEG Biomarkers in Young Autistic Children

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Methodological Issue Being Addressed Studies of executive function in young autistic children often rely on caregiver reports or cognitive assessments with limited validity in young children. They often also lack real-time data and fail to account for cooccurring conditions, limiting translatability to interventions. Here we utilize electroencephalography (EEG) analysis during a go/no-go task to identify neural signatures of executive function in young autistic children, with and without cooccurring ADHD and/or anxiety.

Introduction Since autistic features present heterogeneously, clinicians could benefit from reliable biomarkers early in development to phenotypically stratify and monitor treatment of young autistic individuals. Executive function is one cognitive domain that lends itself to EEG-based biomarkers and could benefit from early intervention. Despite the fact that 40-60% of autistic children have cooccurring anxiety and/or ADHD, the contribution of these disorders to cognitive function in autism remains mostly unknown. This study highlights the use of EEG recordings during a go/no-go behavioral paradigm, which measures the ability to inhibit a dominant response, as a biomarker of executive function in autistic children, with and without cooccurring anxiety and/or ADHD.

Methods 154 autistic children aged 48-95 months with or without cooccurring anxiety and/or ADHD from 4 groups participated: autism alone, autism+anxiety, autism+ADHD, autism+anxiety+ADHD. Of these children, 91 completed an EEG go/no-go task in which participants responded (via button press) to a visual target while inhibiting responses to a lure (i.e., no-go trials). Averaged event-related potentials (ERPs) generated from frontal and parietal electrodes provided neural signatures of executive function and attentional responses, respectively.

Results For no-go trials, the presence of cooccurring ADHD led to significantly quicker frontal N200 latencies, reflecting faster neural reaction times ($p=0.036$), compared to participants with autism alone. Cooccurring ADHD also led to larger frontal P300 amplitudes, suggestive of greater recruitment of cognitive resources to inhibit a response compared to participants with autism alone ($p=0.040$). The addition of cooccurring anxiety (autism+anxiety+ADHD) did not significantly alter the impact of ADHD on frontal ERPs. However, participants with autism+anxiety displayed significantly quicker parietal N200 latencies and larger parietal N200 amplitudes compared to those with autism alone ($p=0.036$, $p=0.023$, respectively), suggesting that cooccurrence of anxiety leads to faster stimulus perception and less neural resource allocation for response inhibition.

Conclusion This study highlights the utility of EEG recordings as potential biomarkers for executive function and target engagement in young autistic populations. This work also demonstrates the need for detection of cooccurring conditions, such as ADHD and anxiety, which are highly prevalent in autistic populations and influence both symptom presentation and response to interventions. Unique ERP signatures were found for the presence of cooccurring ADHD and anxiety in frontal and parietal regions, respectively, suggesting this go/no-go paradigm could be used to stratify autistic cohorts based on cooccurring conditions to decrease heterogeneity of clinical samples.

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Keywords

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autism
EEG
executive function
ADHD
anxiety

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Disclosures Nothing to disclose.