

Impact of Cooccurring Conditions on EEG Biomarkers in Young Autistic Children

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Introduction

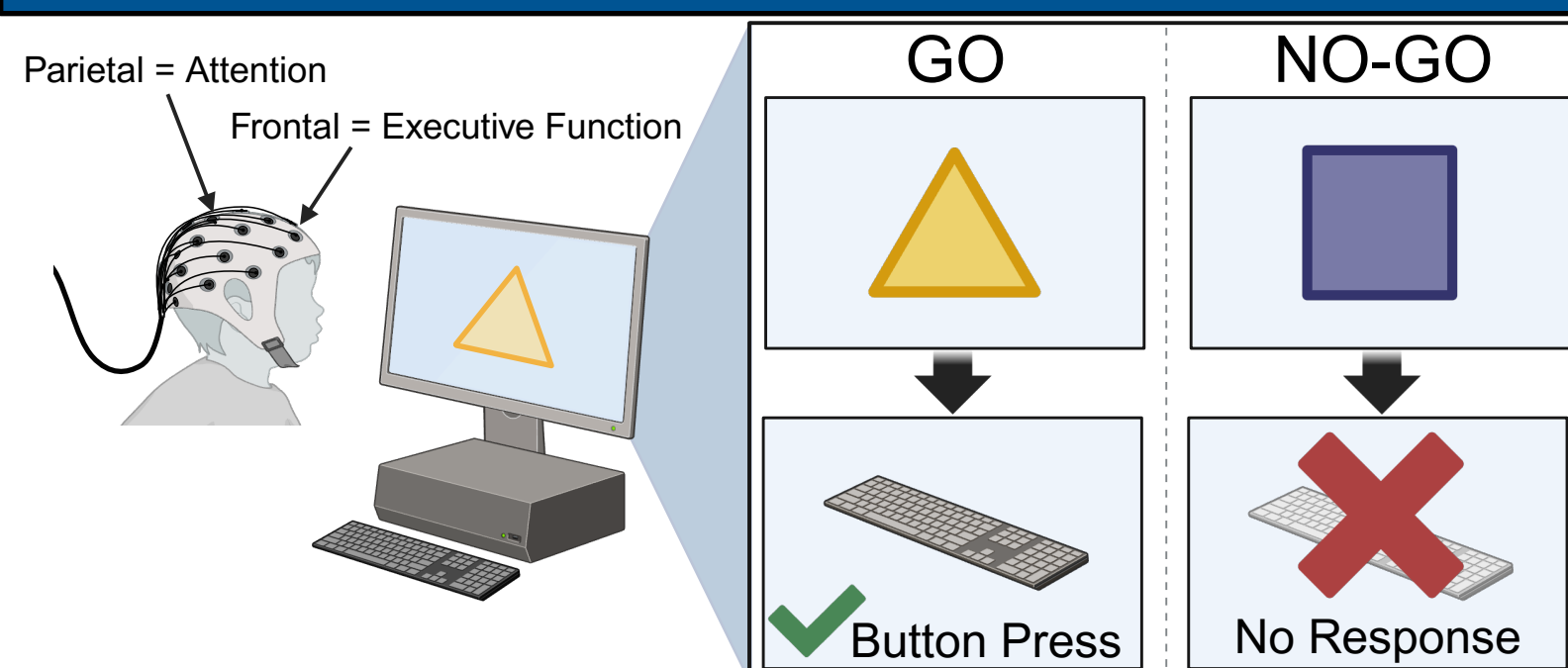
- Autistic features present heterogeneously, complicating interpretation of clinical measures and impacting intervention efficacy.
- Cooccurring anxiety and/or ADHD (present in 40-60% of autistic children) may impact heterogeneity and influence outcomes.
- Reliable biomarkers, which account for cooccurring conditions, are needed to phenotypically stratify young autistic populations for clinical studies.
- We utilized 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.

Participants

	Full HERO Sample	Sample with EEG	P-Value
Age (Mean in months, Std.Dev.)	64.6 (12.7)	72.2 (15.0)	0.0013
Sex			
Female	46	21	N.S.
Male	106	46	N.S.
Reported Race			
White	111	56	N.S.
Black/African American	19	3	N.S.
Asian	5	0	0.011
More than one race	14	6	N.S.
Other	3	2	N.S.
Reported Ethnicity			
Non-Hispanic	130	56	0.035
Hispanic	22	11	N.S.
On Stimulant			
No	132	56	0.019
Yes	19	11	N.S.
Group			
Autism	36	15	N.S.
Autism and ADHD	36	15	N.S.
Autism and Anxiety	30	17	N.S.
Autism, ADHD, and Anxiety	49	20	N.S.

152 autistic children age 48-95 months, with or without cooccurring ADHD and/or anxiety. 4 groups: autism alone, autism+ADHD, autism+anxiety, autism+ADHD+anxiety. 67 completed an EEG go/no-go task.

Methods



- EEG go/no-go task: participants responded (via button press) to a visual target while inhibiting responses to a lure.
- Averaged event-related potentials (ERPs) generated from frontal and parietal electrodes provided neural signatures of executive function and attentional responses, respectively.

Results — Frontal Findings

Fig 1. Frontal Grand Average ERPs for No-Go Trials

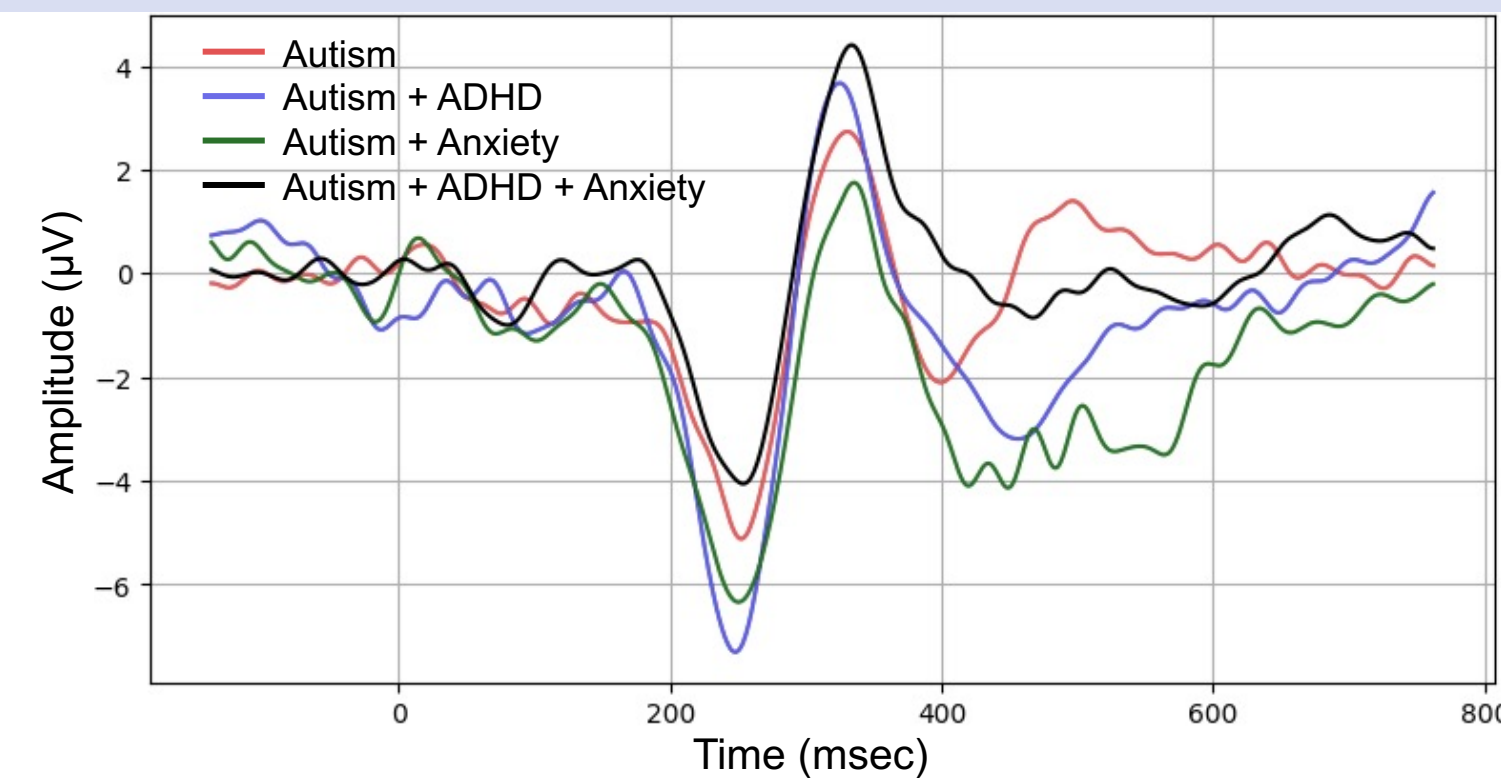


Fig 2. Participants with cooccurring ADHD had quicker frontal N200 latencies compared to participants with autism alone ($p=0.036$), reflecting faster neural reaction times.

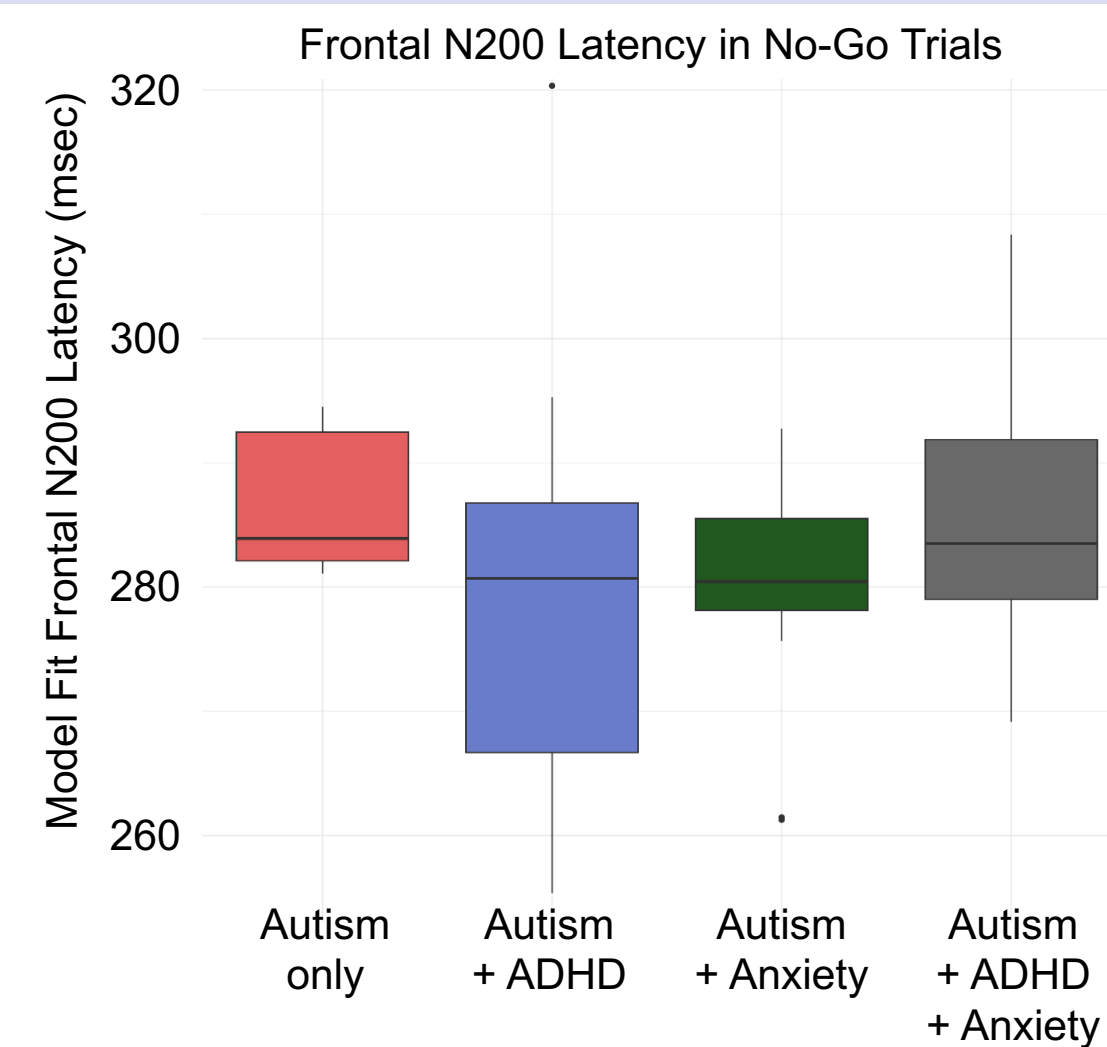
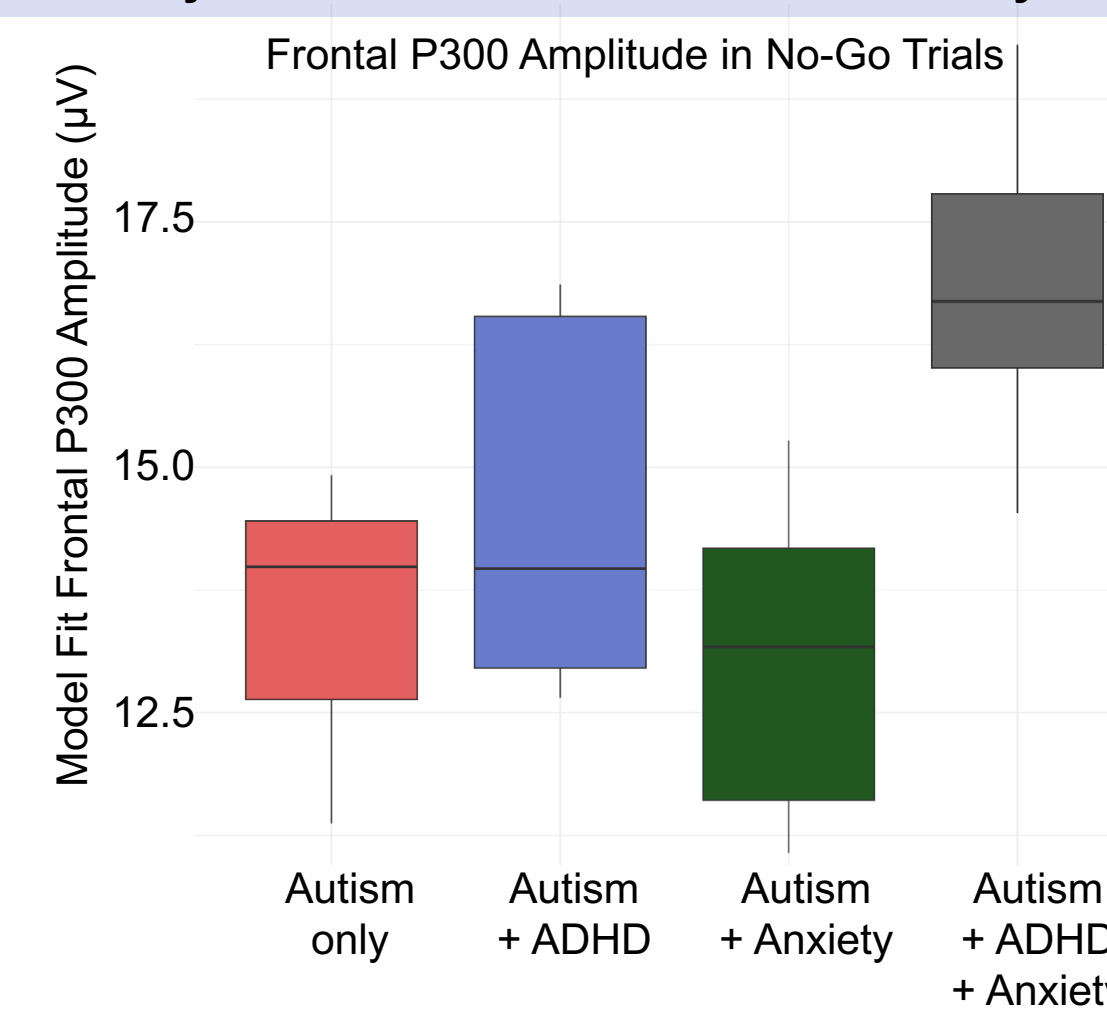


Fig 3. Participants with cooccurring ADHD showed larger frontal P300 amplitudes compared to autism alone ($p=0.040$), suggestive of greater recruitment of cognitive resources to inhibit a response. Effect is driven by those with autism + ADHD + anxiety.



Results — Parietal Findings

Fig 4. Parietal Grand Average ERPs for No-Go Trials

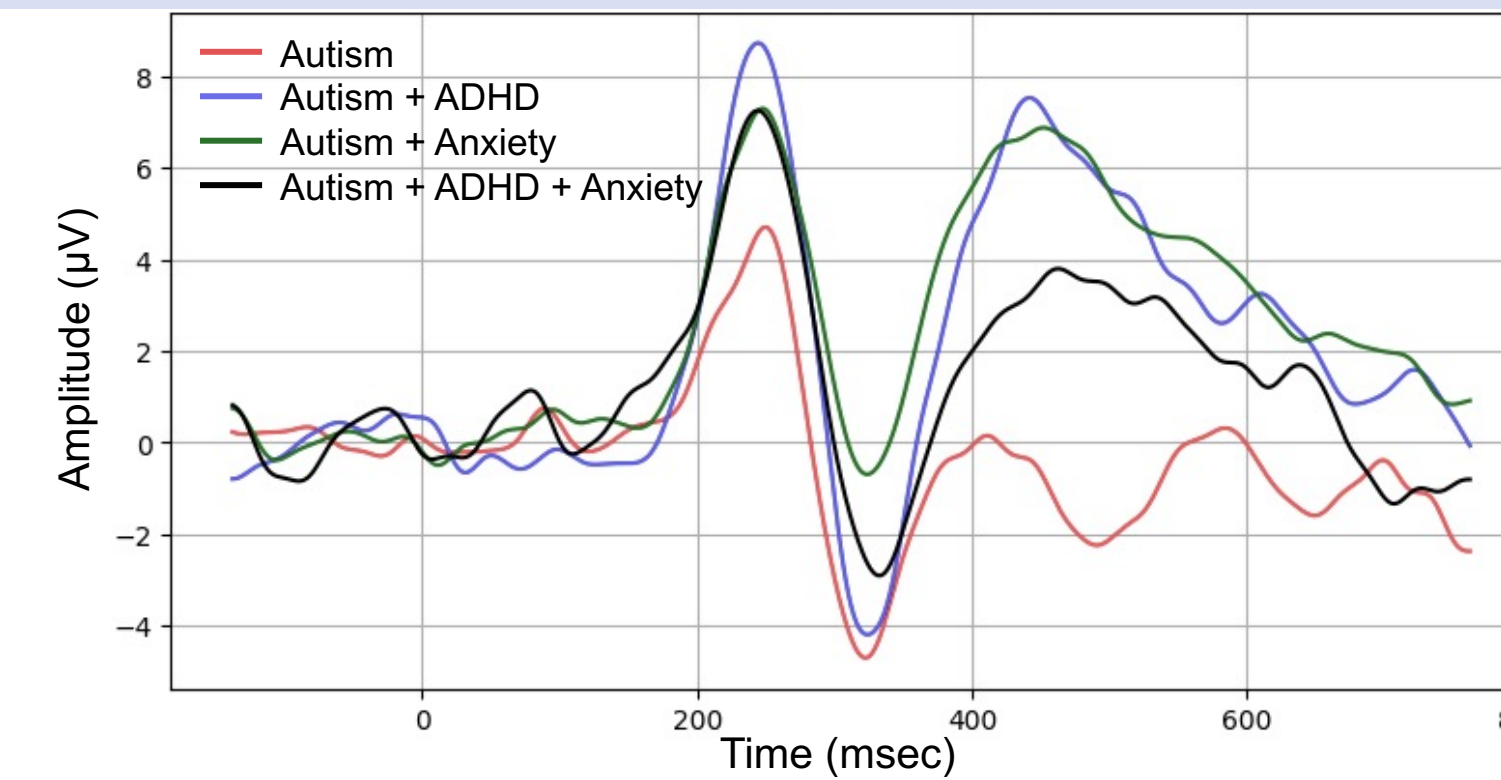


Fig 5. Participants with cooccurring anxiety had quicker parietal N200 latencies compared to autism alone ($p=0.036$), suggestive of faster stimulus perception.

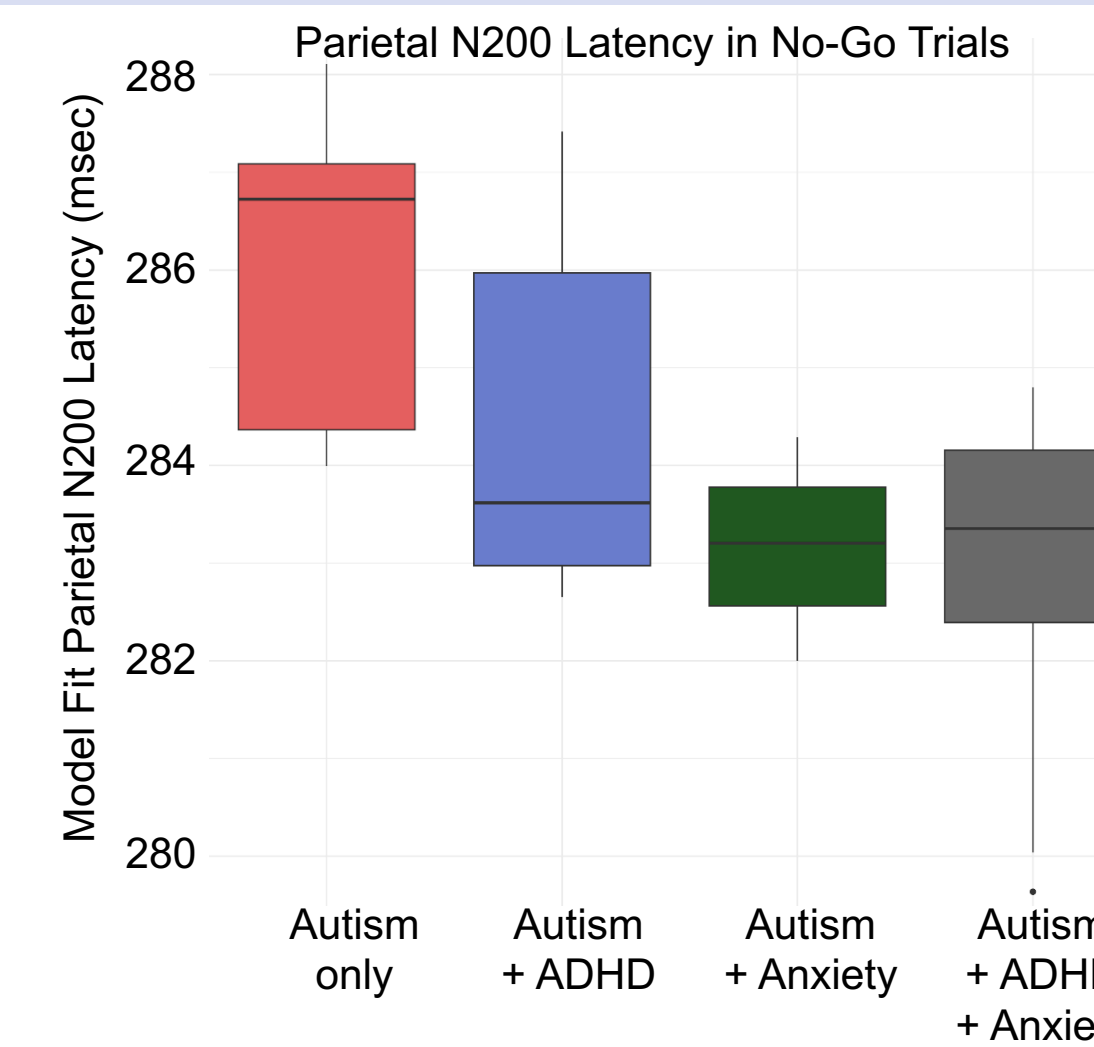
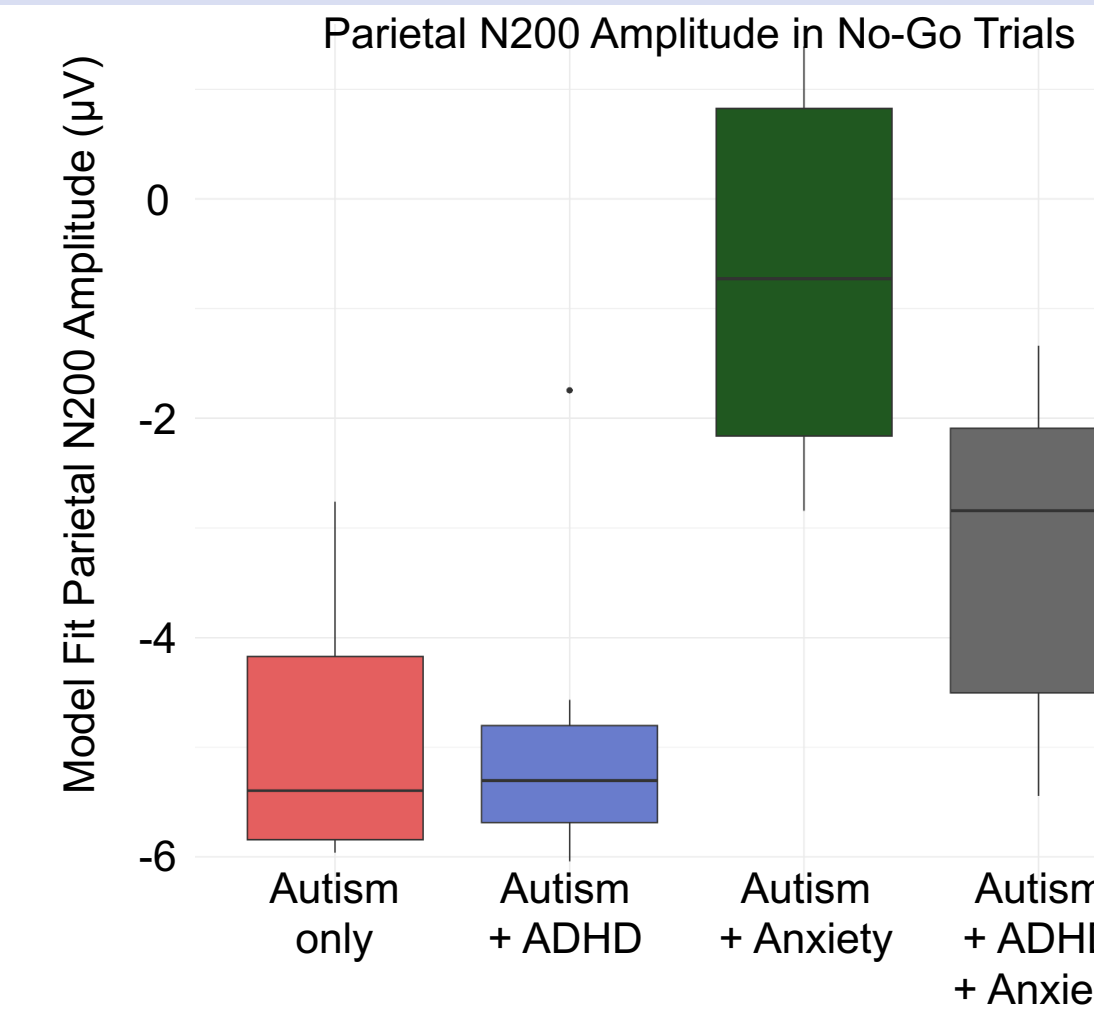


Fig 6. Participants with cooccurring anxiety displayed smaller parietal N200 amplitudes compared to those with autism alone ($p=0.023$), suggestive of less neural resource allocation for response inhibition.



Conclusions

- **Distinct EEG neural signatures were seen for autistic individuals with cooccurring ADHD versus cooccurring anxiety in frontal and parietal regions, respectively.**
- **Go/No-Go EEG paradigms could aide in stratifying autistic cohorts and decrease heterogeneity of clinical study samples.**

Statistical Analyses

- EEG data was preprocessed using MNE Toolbox via Python in conjunction with the EEGLab toolbox via MATLAB.
- Subject EEG data were included in the analysis only if a minimum of 30% of trials were retained and no less than 30% of channels in ROI were categorized as poor.
- Statistical models were computed using R-Studio, where each EEG variable of interest was the outcome of a multiple predictor linear regression accounting for the following potentially confounding predictors:
 - Age at EEG data acquisition (in months)
 - Stimulant usage (discrete variable)

Acknowledgements and Disclosures

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