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Poster · May 2024

DOI: 10.1371/journal.p.02311498

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The Perceptual and Physiologic Correlates of Decreased Sound Tolerance Disorders in Autistic and Non-autistic Adults

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Background

- Decreased Sound Tolerance (DST)¹ refers to the pathological inability to tolerate sounds in everyday life. DST is a common feature of autism¹, although multiple types exist (including hyperacusis² and misophonia³) based on the specific ways in which environmental sounds are perceived as aversive.
- Little is known about the mechanistic underpinnings of DST disorders, particularly in autistic individuals, and electrophysiologic studies to date rarely, if ever, include individuals with multiple distinct DST disorders.
- The present study represents the first relatively large-scale transdiagnostic study of the (differential) neural mechanisms underpinning DST disorders (hyperacusis and misophonia) in both autistic and non-autistic adults.

Methods

- Sample of 113 adults (33 autistic)
 - $M_{age}=33.04$, $SD=9.92$; 74.3% female, 71.7% non-Hispanic White; 43.4% at least some graduate school; Full-scale IQ = 116.58, $SD=11.18$, $Min-Max$: 78–141
 - Autistic and non-autistic groups matched on demographics and cognitive functioning
- Characterized DST status using a structured clinical interview (DISSS) as well as a newly-validated dimensional questionnaire (MIST-A)⁴
- Completed comprehensive battery of psychological and audiological tests
 - Categorical loudness scaling (CLS) with calculation of individual loudness-intensity functions⁵
 - Electrophysiologic tests of central auditory processing (binaural click ABR, 40 Hz ASSR, and N1b ERP to 1-second pure tones during a passive listening task)
- Electrophysiologic “intensity growth slopes” created by taking parameter (amplitude or latency) and regressing it on stimulus intensity for each participant
 - Bayesian hierarchical models used to calculate growth slope values for all participants simultaneously
- Comparison of autistic and non-autistic individuals with and without DST disorders using Bayesian hierarchical regression models

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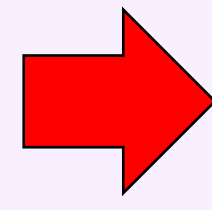
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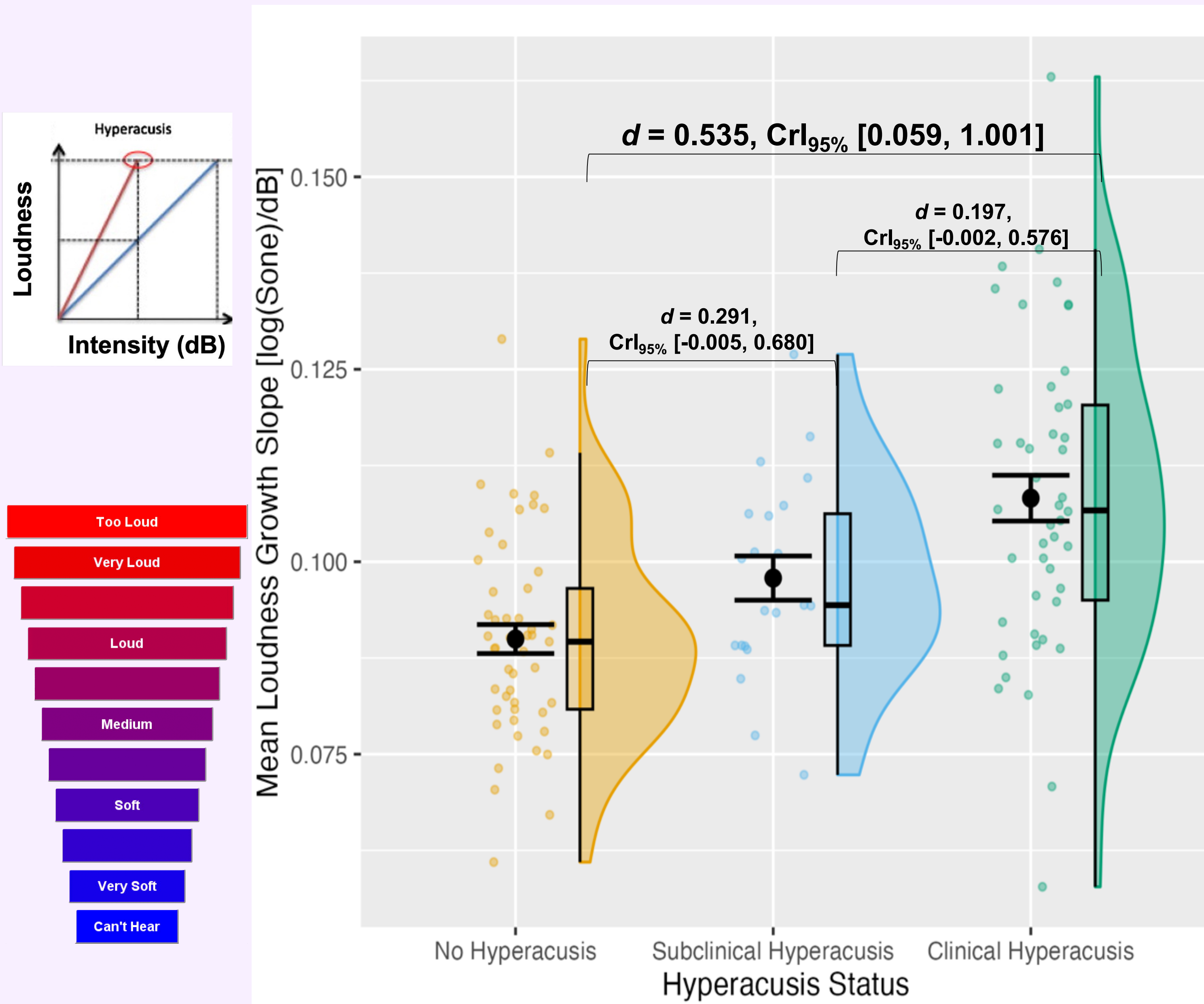
Funding

NIDCD grant F30-DC019510 (ZJW); Misophonia Research Fund (ZJW, CJC, TGW); Family S Endowed Autism Scholarship (ZJW); NCATS grant UL1-TR002243; Autism Science Foundation (DJB)

Competing Interests

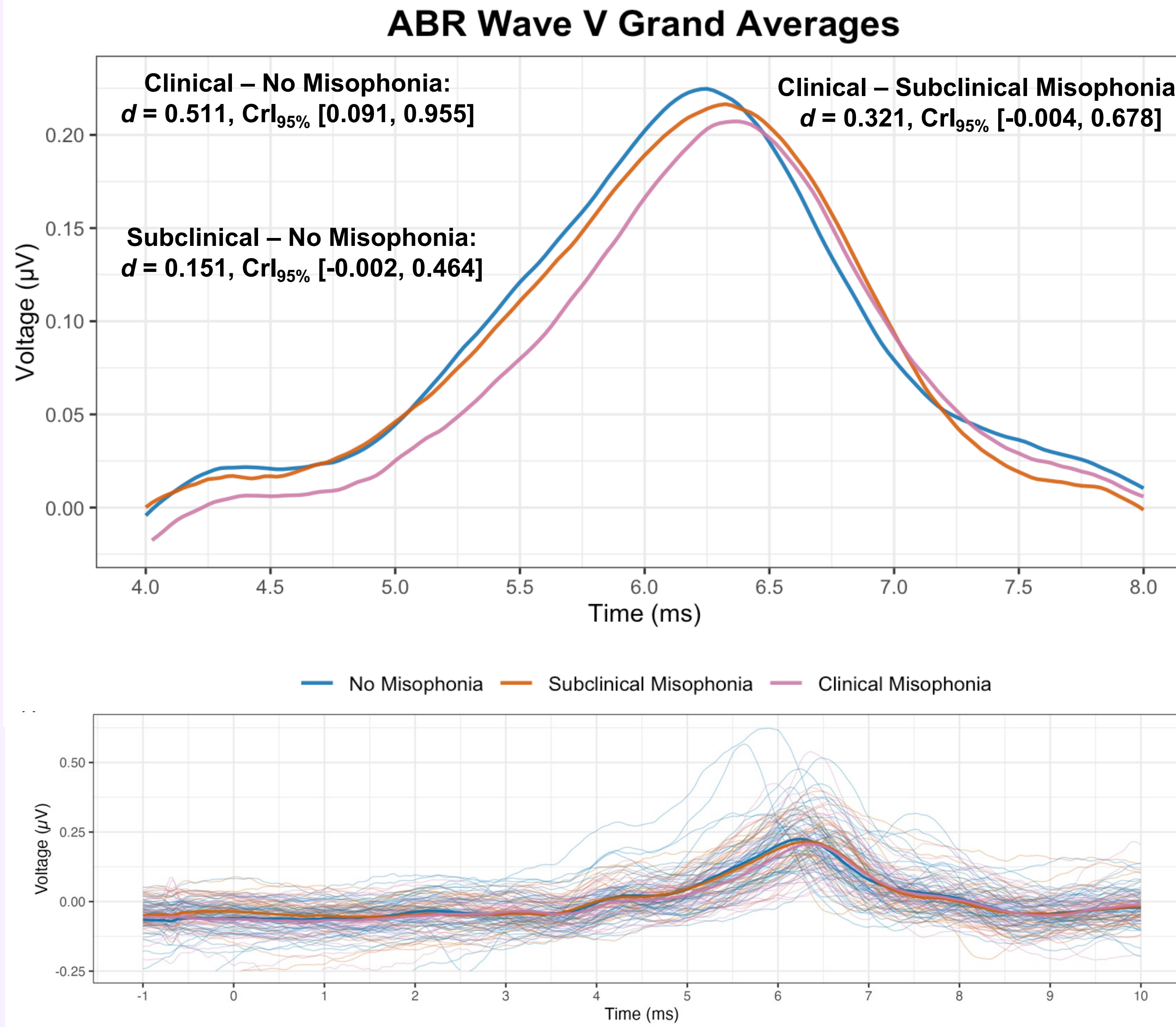
ZJW has received consulting fees from Roche. He is also a member of the Autism Intervention Research Network on Physical Health (AIR-P) ANSWER Committee. TGW is the parent of an autistic adult.

Hyperacusis is associated with steeper psychoacoustic loudness growth



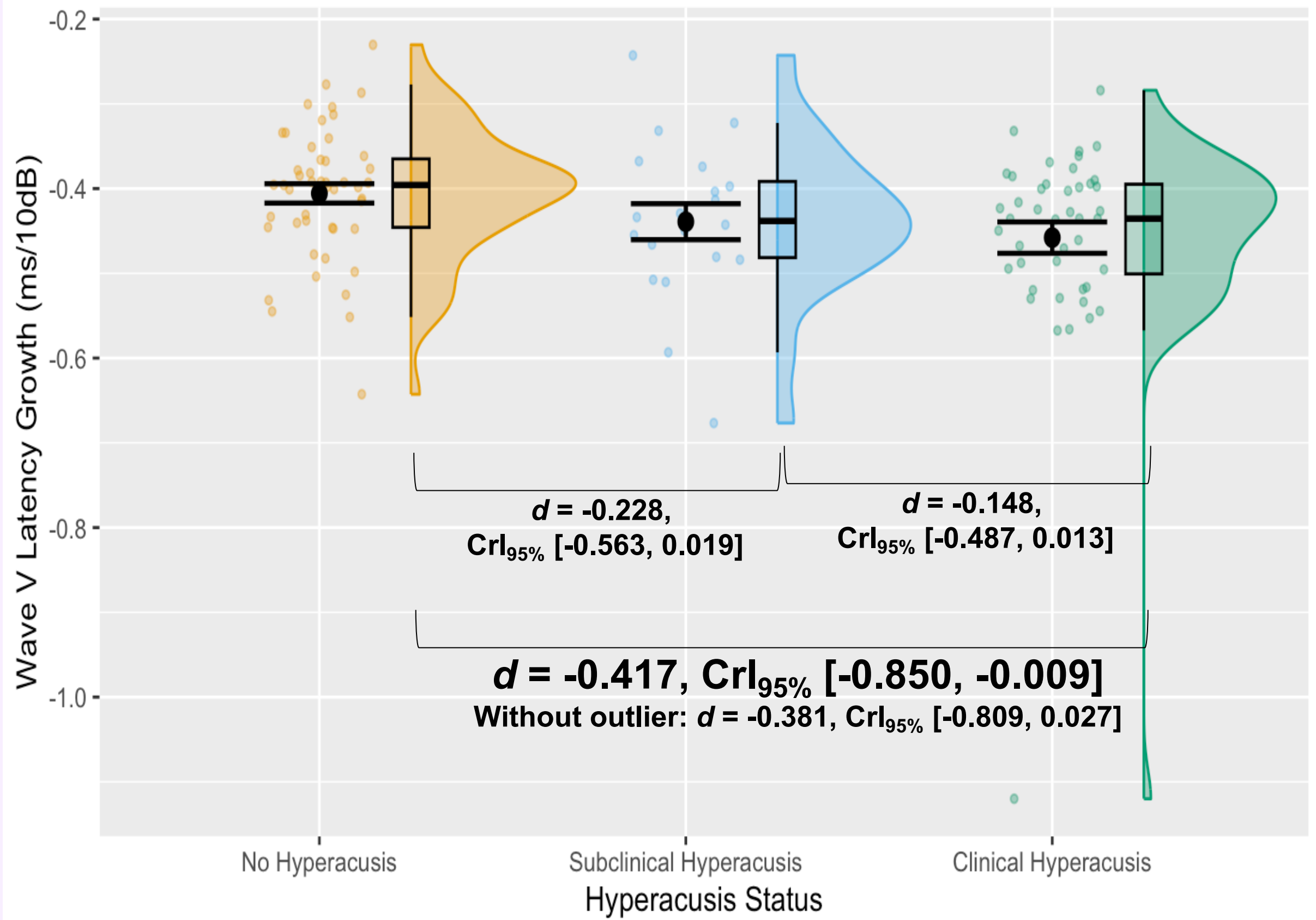
- CLS-based loudness-intensity growth slopes averaged across four tone conditions for each participant

Misophonia is associated with longer latencies of click-evoked auditory brainstem response (ABR) wave V

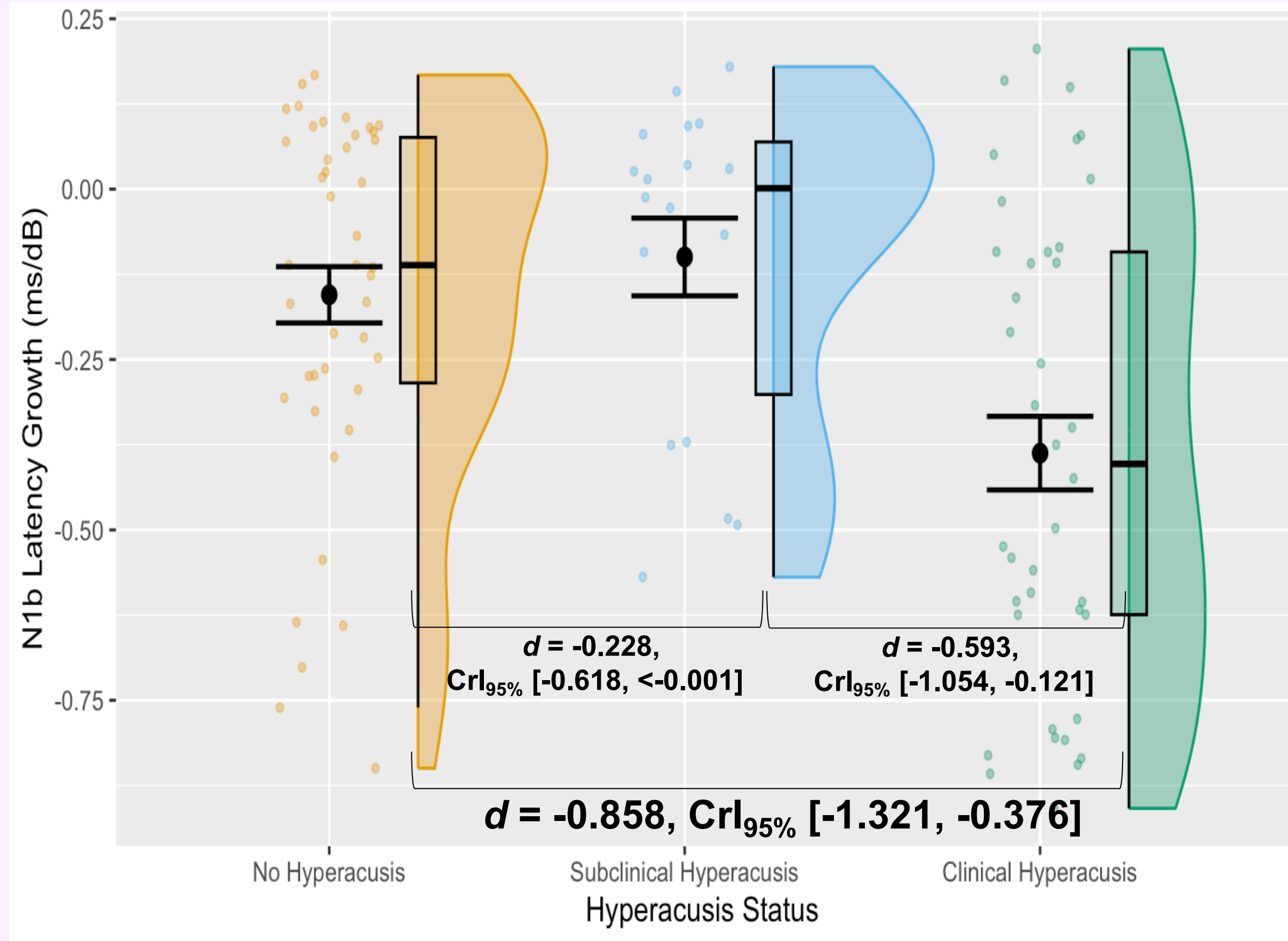


- Correlation with MIST-A Misophonia: $r_{rank} = 0.170$, $CrI_{95\%} [0.008, 0.323]$
- No significant misophonia × autism interaction for ABR wave V latencies
- Raises the possibility of “low level” auditory involvement in misophonia rather than this particular DST disorder being purely a disorder of emotion dysregulation

Hyperacusis is associated with steeper electrophysiologic latency-intensity growth slopes in brainstem and cortex



- Correlations with CLS: $r_{rank} = -0.220$, $CrI_{95\%} [-0.420, -0.025]$ and MIST-A: $r_{rank} = -0.220$, $CrI_{95\%} [-0.391, -0.056]$



- Correlation with CLS: $r_{rank} = -0.330$, $CrI_{95\%} [-0.522, -0.120]$ and MIST-A: $r_{rank} = -0.221$, $CrI_{95\%} [-0.379, -0.041]$
- No significant correlation between ABR wave V latency growth and N1b latency growth ($r_{rank} = -0.023$, $CrI_{95\%} [-0.191, 0.142]$) or significant hyperacusis × autism interaction for either outcome

Discussion/Conclusion

- Increased loudness-intensity growth slopes and two neural indicators of central auditory gain in hyperacusis provide empirical support for the “central gain theory” of the condition
 - Impressive dimensional association between brain, perception, and subjective symptoms – suggests genuine mechanism
 - Two uncorrelated “subtypes” of central gain (brainstem, cortex)
- Misophonia was associated with longer-latency ABR wave V
 - Potentially points towards an earlier locus of misophonia pathophysiology than previously realized

- No evidence to suggest differences in DST mechanisms between autistic and non-autistic adults

- Findings qualified by limitations, including use of operational DST disorder definitions, convenience samples, low interaction power