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Impaired Visual Processing in Glaucoma – Insights from Task and Resting EEG

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ABSTRACT

Glaucoma is traditionally considered as an ocular disease primarily characterized by visual field loss. However, growing evidence suggests cortical involvement. This study investigated cortical differences and potential compensatory mechanisms through dominant eye plasticity. Using task-related and resting-state EEG, we aimed to understand visual processing alterations in glaucoma patients compared to their healthy peers.

OBJECTIVES

- **Task EEG:** Identified cortical biomarkers related to severity of glaucoma using visual evoked potentials (VEP)
 - Amplitude → processing strength
 - Latency → processing speed
- **Resting EEG:** Examined the impact of glaucoma on cortical function
 - Alpha Suppression → a cortical mechanism of attentional engagement, operating independently of retinal input
 - Alpha Peak Frequency (APF) → a biomarker indicating the state of neural oscillation
- **Dominant Eye:** Determined whether ocular dominance modulated the cortical responses of glaucoma, suggesting compensatory plasticity

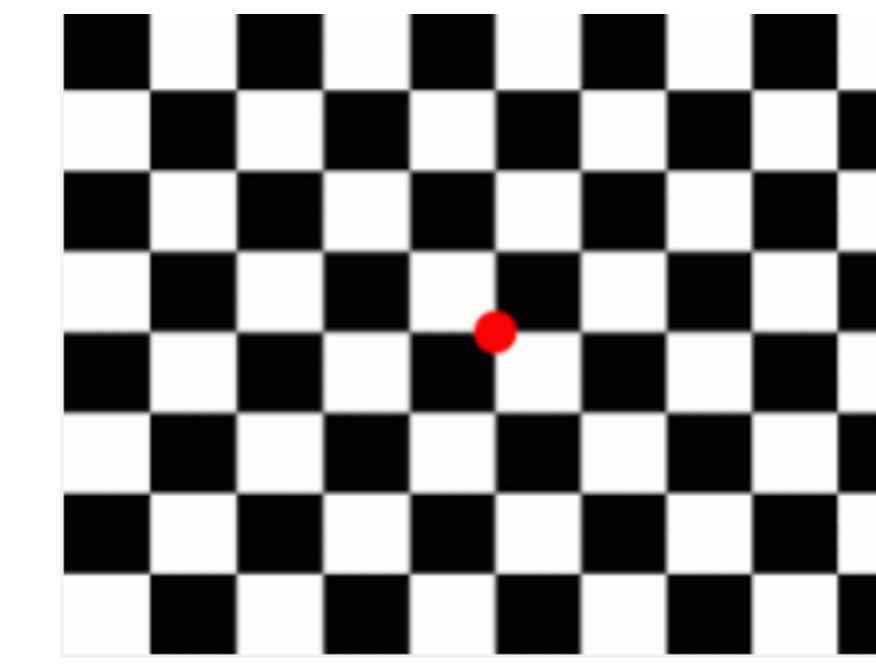
PARTICIPANTS

- **30 Bilateral Glaucoma Patients**
 - Mean Deviation: -15.41 ± 6.34 dB in HFA 24-2
 - Age: 63 ± 8.26 years
- **28 Age-matched Normal Controls**
 - Mean Deviation: -0.16 ± 1.18 dB
 - Age: 62.82 ± 9.23 years

METHODS

Protocol: 64-channels EEG

- **Task EEG:** Checkerboard reversal induced VEP (monocular)
 - 30 seconds x 2 times
- **Resting EEG:** 3 min eye close and 3 min open
- **Ocular dominance**
 - Test by motor dominance



RESULTS

1. Glaucoma group had **decreased amplitude** and **prolonged latency** in VEP

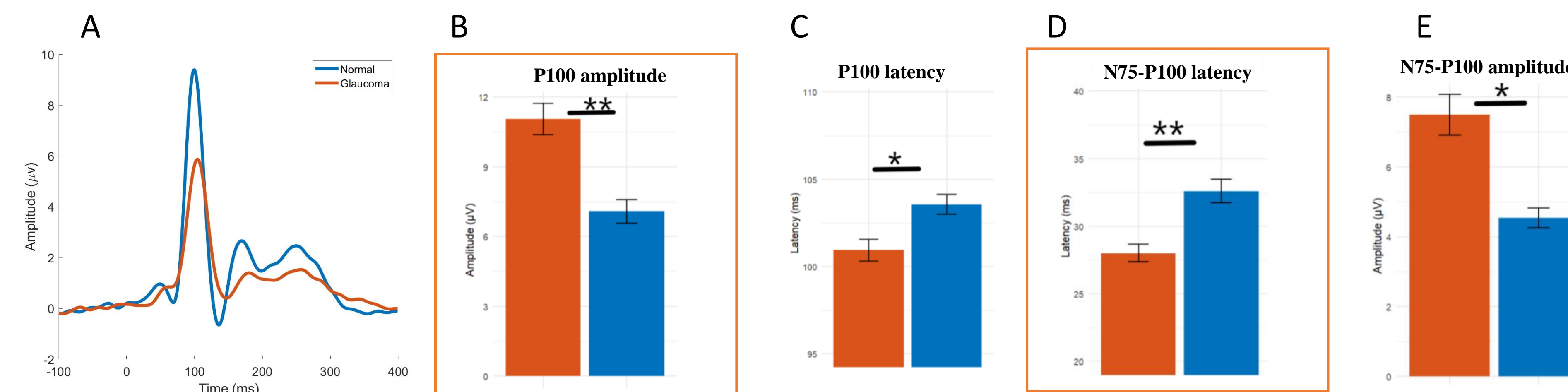


Fig 1. Comparison of VEP waveforms between glaucoma and normal controls (A). Specific ERP measures group, highlighting group differences in P100 amplitude/latency and N75-P100 amplitude/latency (B to E).

2. Glaucoma group had **reduced alpha suppression**

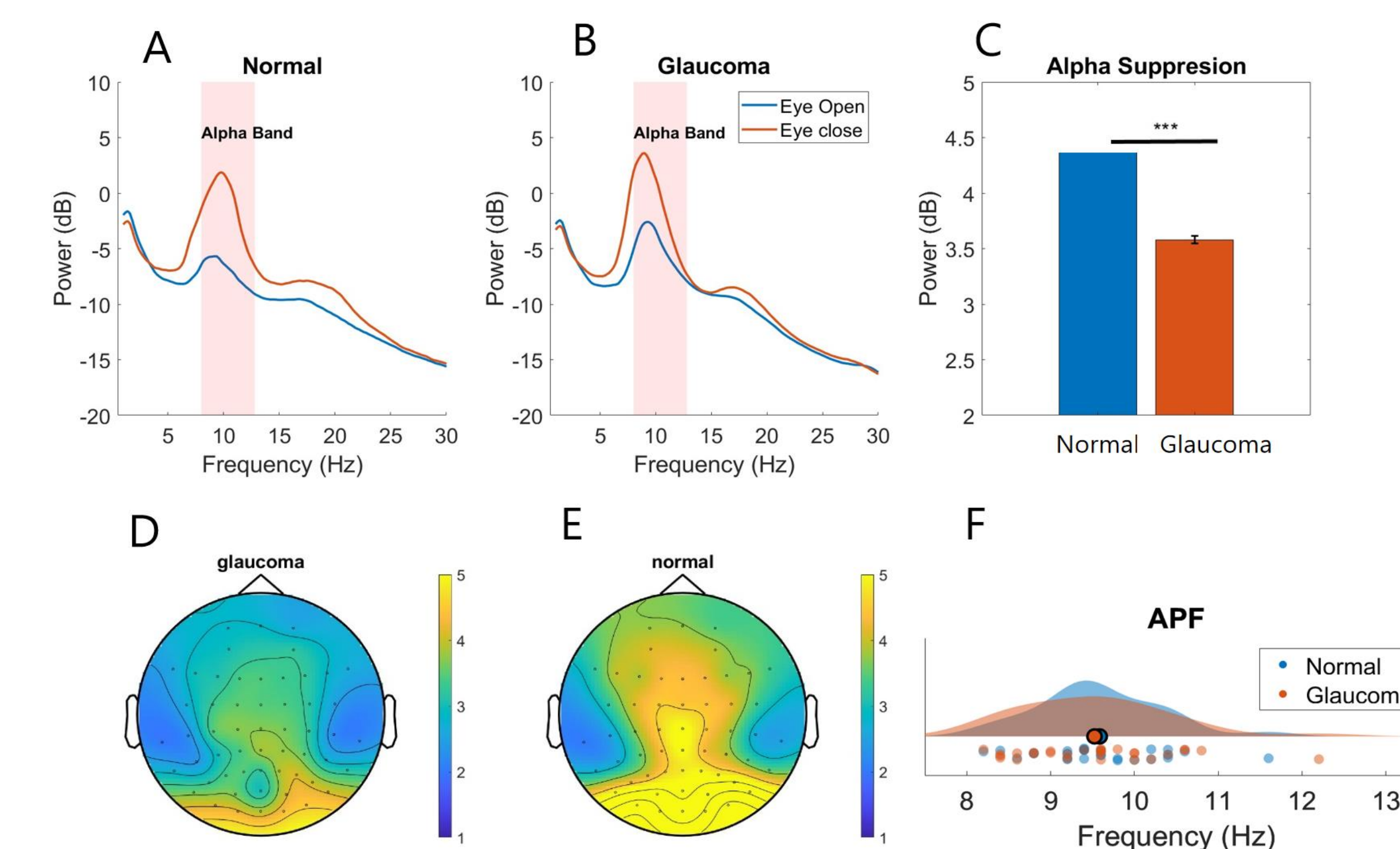


Fig 2. Panels A and B indicate the power distribution in eye-open and eye-close states for glaucoma patients and normal controls, with the alpha band range highlighted. Panel C is the alpha suppression, representing the difference between these states for both groups. Panels D and E show the topographic distributions of alpha suppression across the scalp for glaucoma and normal controls, respectively. Panel F is the raincloud plot of Alpha Peak Frequency (APF) for both groups.

3. Dominant eye mitigated the impact of **glaucoma severity** on VEP amplitudes

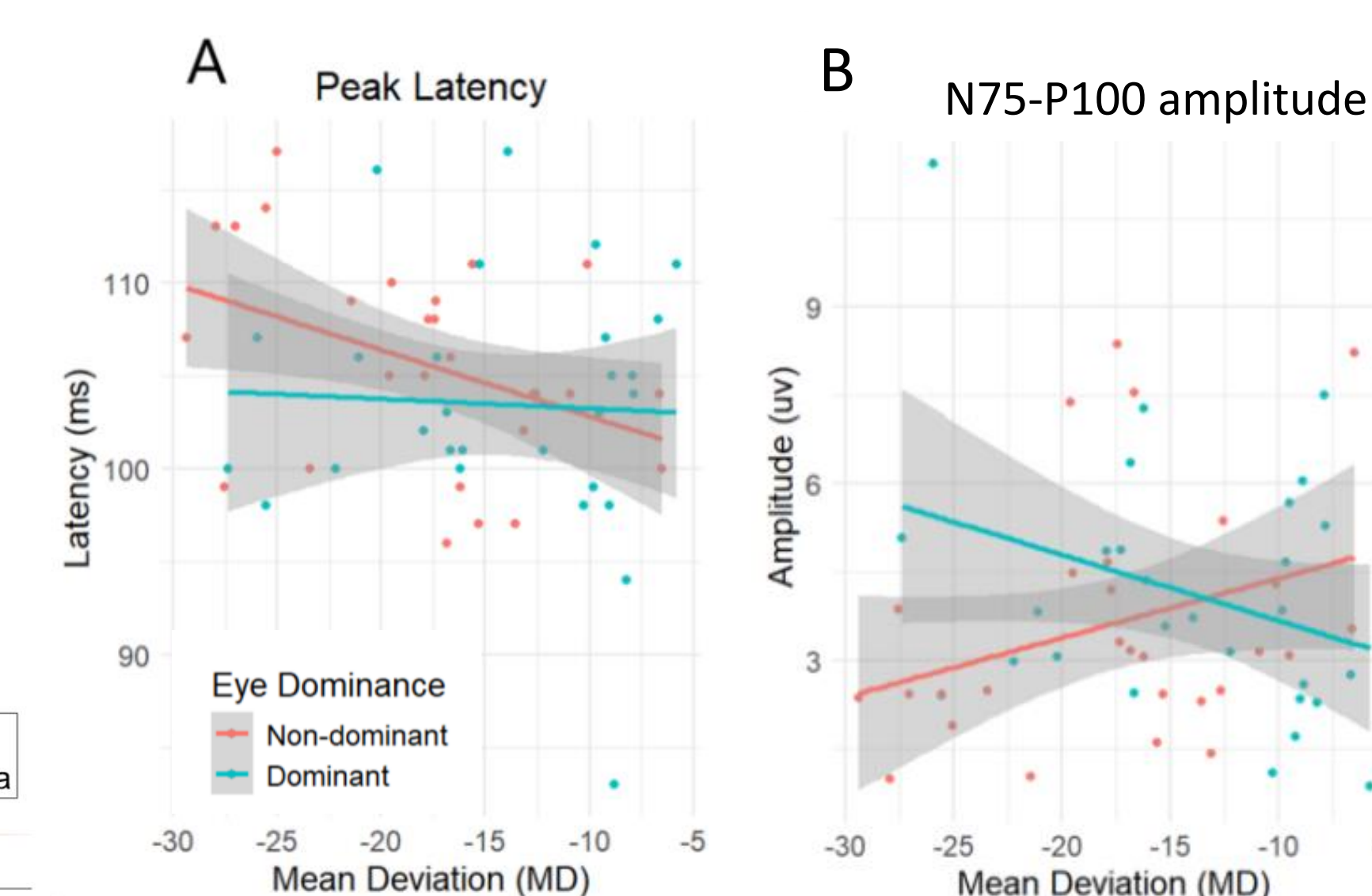


Fig 3. Interaction between ocular dominance and glaucoma severity on VEP responses, with a linear regression line showing the trend. Panel A shows that peak latency is similarly prolonged in both eyes as glaucoma severity worsens, suggesting a uniform slowing of visual processing. Panel B shows a significant interaction effect between ocular dominance and glaucoma severity on amplitude measure.

CONCLUSIONS

- Glaucoma **disrupts cortical visual processing**, affecting both strength (VEP amplitude) and speed (VEP latency) of responses
- It also alters the default network dynamics, as evidenced by changes in **alpha suppression**
- The **dominant eye shows adaptive plasticity**, suggesting potential avenues for developing new rehabilitation strategies

Clinical Implication

- **EEG biomarkers** might facilitate earlier detection of cortical dysfunction in glaucoma patients
- **Training the dominant eye** may enhance compensatory plasticity, offering a potential therapeutic approach for managing glaucoma

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