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Sustained-attention slowing and trial-level P3-reaction time coupling in university students with generalized anxiety disorder and self-reported habitual short sleep: a secondary cross-sectional study

Ce Shi
Henan University of Traditional Chinese Medicine
Haiping Li
Henan University of Traditional Chinese Medicine
Lihua Wu
Henan University of Traditional Chinese Medicine
Saisai Li
The Third Affiliated Hospital of Henan University of Chinese Medicine
Mengyu Wang
Henan University of Traditional Chinese Medicine
Yajing Guo
Henan University of Traditional Chinese Medicine
Jing Wen
Henan University of Traditional Chinese Medicine
Jing Gao
First Affiliated Hospital of Henan University of Traditional Chinese Medicine
Wen Fu
First Affiliated Hospital of Henan University of Traditional Chinese Medicine
Xinwang Chen
Henan University of Traditional Chinese Medicine
Yiming Wu
wuyimingsir@163.com

The Third Affiliated Hospital of Henan University of Chinese Medicine

Research Article

Keywords: generalized anxiety disorder, habitual short sleep, sustained attention, psychomotor vigilance test, electroencephalography, event-related potentials, P3, reaction time

Posted Date: October 7th, 2026

DOI: https://doi.org/10.21203/rs.3.rs-11088532/v1

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Additional Declarations: No competing interests reported.

Abstract

Background

Generalized anxiety disorder and habitual short sleep commonly coexist. Sustained attention is clinically relevant, but objective performance and electroencephalographic correlates in young adults with this combined presentation remain insufficiently characterized.

Methods

This secondary cross-sectional analysis used baseline data from a parent study. Sixty-nine university students (32 healthy controls and 37 with clinically diagnosed generalized anxiety disorder and self-reported habitual short sleep) completed a 10-min psychomotor vigilance test during 32-channel electroencephalography. Analyses included participant-level behavior, averaged event-related potentials and time-frequency measures, and single-trial P3-reaction time models. Unadjusted analyses were primary; age- and sex-adjusted models were sensitivity analyses.

Results

Median reaction time was 33.00 ms longer in the combined clinical group (95% CI 12.35–53.65; Hedges' $g = 0.745$; $p_{FDR} = 0.00873$). The age- and sex-adjusted difference was 31.48 ms (95% CI 3.46–59.50; $p = 0.0283$; sensitivity-family $p_{FDR} = 0.0449$). Participant-level reaction-time progress slopes differed, but the trial-level group × progress interaction did not, providing inconsistent evidence for a differential vigilance decrement. None of 42 principal averaged electroencephalographic group contrasts survived the relevant family and global multiplicity corrections. Across 3,231 trials with complete model data from 60 participants, larger within-participant P3 amplitudes predicted faster responses ($\beta = -0.02384$; SE = 0.00593; $p = 5.89 \times 10^{-5}$; $p_{FDR} = 0.000648$). The adjusted association remained negative ($\beta = -0.02332$; 95% CI -0.03929 to -0.00736; $p = 0.00420$), while the P3 × group interaction was nonsignificant ($\beta = 0.01595$; $p = 0.144$).

Conclusions

University students with generalized anxiety disorder and self-reported habitual short sleep showed slower sustained-attention responses than healthy controls. Within-person P3 amplitude tracked trial-level response speed across groups, but no corrected averaged electroencephalographic group marker or group-specific P3 relationship was identified. The design cannot separate effects of generalized anxiety disorder from those of habitual short sleep.

Background

Generalized anxiety disorder (GAD) commonly emerges by early adulthood [1] and includes persistent worry, hyperarousal, and difficulty concentrating. Objective cognitive testing can clarify whether concentration complaints are accompanied by measurable inefficiency in sustained attention. Attentional control theory proposes that anxiety may reduce processing efficiency without uniformly lowering accuracy [2]. The psychomotor vigilance test (PVT) provides a low-complexity measure of sustained attention with limited working-memory, rule-switching, and emotional-processing demands. Laboratory performance, however, does not establish academic or everyday impairment.

Sleep is central to this question because poor sleep, stress, and anxiety frequently co-occur in university students [3–5]. Hyperarousal, impaired emotion regulation, and reduced alertness may link anxiety and insufficient sleep to cognitive inefficiency [6,7], and anxiety may moderate sleep-related memory effects [8]. The psychomotor vigilance test (PVT) is sensitive to sleep loss and offers a low-complexity measure of sustained attention [9,10]. Because GAD and habitual short sleep co-occurred by design here, their independent contributions cannot be estimated.

Electroencephalography (EEG) can address two related questions. Averaged participant-level event-related potential and oscillatory measures test group differences, whereas single-trial models test whether within-person physiological variation covaries with behavior. A null averaged group comparison therefore does not rule out a trial-level association. P1/N1, N2, and P3/LPC components, theta and alpha activity, inter-trial phase consistency, response-locked potentials, and response-related beta describe different aspects of attention and motor processing. P3 amplitude has been related to stimulus evaluation and phasic arousal, prestimulus alpha to momentary attentional state, and beta changes to motor-system state [11–17]. Vigilance and sleep-loss studies have also reported time-dependent ERP, theta, and waking EEG changes [18–22], while anxiety-related ERP findings vary with clinical composition, task demands, and analytic choices [23,24].

We examined sustained-attention performance in university students with clinically diagnosed GAD and self-reported habitual short sleep and in healthy student controls. The primary hypothesis was that the combined GAD-HSS group would show slower PVT responses. Secondary analyses examined time-on-task, averaged event-related potentials, time-frequency measures, response-related beta, and within-participant P3-reaction time coupling, including possible group moderation. GAD-HSS is operational shorthand rather than a diagnostic entity. Because the study did not include GAD-normal-sleep or non-GAD-short-sleep groups, every between-group inference concerns the combined presentation.

Methods

Study design ethics and registration

This report is a secondary cross-sectional analysis of baseline data from the parent study described in the published protocol by Shi et al. [25]. The protocol reported the planned parent-study design but no

participant-level results. The behavioral and EEG findings reported here have not previously been published. The study was conducted in accordance with the Declaration of Helsinki. The study was conducted in accordance with the Declaration of Helsinki. The Ethics Committee of the Third Affiliated Hospital of Henan University of Chinese Medicine approved the study (2023SH-147), and all participants provided written informed consent. The parent trial was registered in the Chinese Clinical Trial Registry on 25 March 2024 (ChiCTR2400082221). The present article reports only a cross-sectional analysis of baseline PVT-EEG data acquired after registration, between 28 September and 28 November 2024; it does not report intervention outcomes.

Participants and clinical characterization

Participants were recruited from the Longzihu and Dongming Road campuses of Henan University of Chinese Medicine. Earlier project documents referenced recruitment beginning in June 2023, but pre-enrollment logs were unavailable for independent confirmation. GAD was diagnosed according to the International Statistical Classification of Diseases and Related Health Problems, 10th Revision (ICD-10), through a clinical assessment conducted by a licensed clinical psychologist who had received systematic training in the application of the ICD-10 criteria for GAD. The assessment comprised a face-to-face clinical interview supported by questionnaire-based evaluation. The GAD-7 was not used as the sole diagnostic basis; a score of ≥ 5 was used as an additional symptom-severity criterion among participants who had already met the ICD-10 diagnostic criteria for GAD. The Generalized Anxiety Disorder 7-item scale (GAD-7) and Hamilton Anxiety Rating Scale (HAMA) were previously published instruments and were not developed for this study. The GAD-7 was used as an additional symptom-severity criterion rather than as the sole diagnostic basis [26], whereas HAMA scores were used for clinical characterization [27].

Clinical-group eligibility additionally required self-reported habitual weekday nocturnal sleep of 6 h or less for at least one month and written informed consent. Students were ineligible if they had received anti-anxiety medication or anti-anxiety acupuncture during the previous 30 days. Other exclusions included organic mental disorders, anxiety or depression secondary to surgery or psychoactive substances, a family history of severe psychiatric disorders, and cognitive impairment that prevented EEG acquisition.

Healthy controls were recruited from the same university population and were currently enrolled undergraduate or postgraduate students. They were required not to meet criteria for GAD or another major psychiatric disorder. Controls were not matched on short-sleep status. Sleep duration was assessed retrospectively by self-report without a sleep diary, actigraphy, or polysomnography.

The analyzed cohort comprised 69 currently enrolled university students: 32 healthy controls (all undergraduates) and 37 students with clinically diagnosed GAD and self-reported habitual short sleep (35 undergraduates and two postgraduates). All clinical-group participants were aged 18–25 years, had GAD-7 scores of at least 5, and reported weekday sleep of 6 h or less. This clinical group is hereafter

referred to as GAD-HSS. The sample size was determined by the number of eligible participants with complete baseline PVT and EEG data available from the parent study. No separate a priori power calculation was performed for this secondary cross-sectional analysis.

Psychomotor vigilance task

The PVT was implemented in E-Prime 3.0. Participants sat approximately 75 cm from the monitor and watched a central outlined rectangle. When the target appeared, an elapsed-time counter was displayed. As confirmed by the study team, participants responded with the right hand by pressing the '1' key on a Lenovo keyboard. The task folder contained reaction-time, too-fast, and too-slow feedback screens.

The event stream showed one continuous run per participant lasting 577.5–603.6 s (median 593.8 s) and 79–81 target events. Recorded S11 target, S1 response, and S3 feedback events supported trial reconstruction. A 10-s response timeout was used. The available task files did not permit recovery of the exact programmed foreperiod or feedback duration. These parameters were described from the recorded event stream and were not inferred beyond the available evidence.

EEG acquisition

EEG was acquired with a Brain Products actiCHamp plus system and an electrode cap fitted with Ag/AgCl electrodes. The montage followed the international 10–20 system. Electrode impedances were reduced to 5 kOhm or lower before recording, and data were sampled at 500 Hz. All BrainVision headers identified the actiCHamp amplifier and Cz as the online reference. The physical configuration contained 29 connected scalp data channels and bilateral mastoids TP9 and TP10. Cz was restored during preprocessing, yielding 30 analyzable scalp locations. Recordings took place in a quiet, dimly lit room while participants maintained central fixation and minimized movement.

EEG preprocessing and quality control

Processing used MATLAB R2020b and EEGLAB 2019 [28] under Windows 11. Reporting of EEG acquisition, preprocessing, artifact handling, and data reduction followed recommendations for EEG/MEG studies [29], and reporting of frequency-domain and time-frequency procedures followed the corresponding Psychophysiology recommendations [30]. Raw BrainVision files were read-only and were not overwritten. Structural labels marked as not connected were removed before quality control, and Cz was restored as the acquisition reference channel. Zero-phase finite-impulse-response filters produced three branches: 0.1–30 Hz for ERP analysis, 0.5–40 Hz for time-frequency analysis, and 1–40 Hz for independent component analysis (ICA). No additional notch filter was used because 50 Hz lay outside the analysis passbands.

Bad-channel decisions combined flatness, robust variance, peak-to-peak amplitude, sustained high amplitude, step changes, robust interchannel correlation, and fixed-copy detection. A scalp channel was rejected for a structural or fixed-copy failure, flatness, or at least two convergent abnormal metrics. TP9 and TP10 were assessed separately and were not interpolated. Gross continuous artifacts were removed before ICA.

Extended Infomax ICA was trained on the 1-40-Hz branch using the stable covariance rank [31–33]. ICLabel supported component review [34]. Components were removed only when classification probabilities agreed with topographic, spectral, and temporal evidence. Cleaned ICA weights were transferred to ERP and time-frequency branches after channel-order and reference validation. Rejected scalp channels were spherically interpolated.

The average of TP9 and TP10 was the primary offline reference. Six records failed the defined mastoid-quality assessment and were excluded from primary-reference EEG inference. These records remained eligible for an average-reference sensitivity branch. Saved datasets were reopened to verify integrity, finite values, external data size, and rereferencing algebra. The maximum number of rejected scalp channels was 3 of 29 physically connected scalp channels. The maximum removed continuous-data fraction was 3.48%.

Trial reconstruction and behavioral outcomes

Trials were reconstructed from each S11 event to the next S11 event. The first legal S1 event was the primary response, and later S1 events were logged as extra presses. Trials were invalid when they crossed an EEG boundary, contained an S1/S3 conflict, or yielded a negative or nonfinite RT. Responses below 100 ms were anticipatory. Responses from 100 to below 500 ms were normal, responses from 500 to below 10,000 ms were lapses, and S3 events without S1 were omissions.

The first and last quarters of normalized trial progress defined early and late periods. Within-participant lower and upper RT quartiles defined fast and slow trials. Primary participant-level behavioral outcomes were median RT, RT coefficient of variation, lapse proportion, and reaction-time progress slope. Secondary measures included mean RT, RT standard deviation, response speed, omission proportion, and early and late median RT.

Stimulus-locked and response-locked event-related potentials

Stimulus-locked epochs extended from − 500 to 1,000 ms and used a − 200 to 0 ms baseline. Artifact rejection excluded epochs with absolute amplitude above 100 μV, peak-to-peak amplitude above 200 μV, robust log-root-mean-square z score above 5, kurtosis above 15, boundaries, or nonfinite values. Acquisition-supported regions of interest and windows were defined before group contrasts. P1 was measured from 70 to 130 ms and N1 from 130 to 200 ms at O1/Oz/O2. N2 was measured from 200 to 350 ms at Fz/Cz. P3/LPC was measured from 300 to 600 ms at Cz/Pz. Mean amplitude was primary.

Response-locked epochs extended from − 1,000 to 800 ms. The primary baseline was − 800 to -600 ms, with − 600 to -400 ms used for baseline sensitivity. Response-locked outcomes were readiness activity at Cz from − 500 to -100 ms, motor activity at C3/Cz/C4 from − 100 to 100 ms, and post-response activity at Cz/Pz from 100 to 400 ms. All valid estimates required at least 30 clean trials. Conditional ERP contrasts required at least 15 clean trials.

Time-frequency analysis

Time-frequency decomposition used complex Morlet wavelets from 2 to 40 Hz, with cycles increasing linearly from 3 to 9. Stimulus-locked epochs extended from − 1,500 to 2,000 ms and used − 1,000 to -500 ms as the power baseline. Outcomes were poststimulus theta power at Fz/Cz, alpha suppression and prestimulus alpha at O1/Oz/O2/Pz, and delta/theta inter-trial phase coherence. Response-related beta power was quantified from 13 to 30 Hz at C3/Cz/C4 using a -1,000 to -600 ms baseline. Power changes are reported in decibels. Frequency bands, temporal windows, regions of interest, baseline intervals, and trial-retention rules were fixed before group contrasts; Additional file 1 reports endpoint-specific retention and reference sensitivity.

Single-trial models

Single-trial neural predictors were standardized within participant, so their coefficients represent within-person associations between trial-level neural variation and behavior. Separate mixed models tested prestimulus alpha, P1, and P3 as predictors of log RT. Models adjusted for normalized trial progress, foreperiod, and previous-trial log RT. The original neural models included participant random intercepts. The time-on-task behavioral model included participant-specific progress slopes when convergence permitted. Neural predictors were modeled separately to avoid combining correlated measures.

The P3 sensitivity analysis added participant-specific P3 slopes. Both the primary random-intercept model and the age- and sex-adjusted random-slope model used 3,231 trials from the same 60 participants. Thirty-five rows in the 3,266-row analysis table were excluded because standardized foreperiod and/or previous-trial log-RT derivatives were missing. The preferred adjusted model used a correlated random intercept and P3 slope; uncorrelated and participant-clustered sensitivity models were also fitted.

Statistical analysis and sensitivity framework

Participants were the independent unit for group contrasts. Welch tests were primary for continuous outcomes. Rank-sum and 10,000-label permutation tests provided sensitivity analyses. Hedges' g and participant-level bootstrap confidence intervals accompanied group effects. group × speed and group × time effects were contrasts of within-participant slow-versus-fast and late-versus-early differences.

Benjamini-Hochberg FDR correction [35] was applied within behavioral, early ERP, late ERP, response-locked ERP, time-frequency, response-beta, and single-trial families. These families were specified in the

analysis pipeline before examination of group contrasts but were not described as formally preregistered analysis families. A global sensitivity correction combined all 42 principal EEG group contrasts without replacing the family-based primary correction.

Post-response beta robustness analyses included rank-based, permutation, bootstrap, robust-regression, average-reference, near-threshold, and leave-one-participant-out checks. Full implementation details and participant-level diagnostics are reported in the Additional file 1. The primary 15-trial rule was unchanged.

Participant-level age and sex data were available for all 69 participants. The original unadjusted analyses remained primary, and age- and sex-adjusted models were performed as sensitivity analyses. Continuous participant-level outcomes used HC3 inference and Huber sensitivity analyses. Lapse counts were additionally assessed with an NB2 model using log(valid trials) as an offset because the aggregate binomial model was overdispersed. Adjusted p values for the original endpoint models were corrected within their corresponding analysis families. The NB2 lapse model was an additional overdispersion sensitivity analysis and was not included in the adjusted behavioral FDR family.

Use of generative AI-assisted technologies

During manuscript preparation, the authors used OpenAI ChatGPT solely for language editing and polishing. The authors reviewed and revised the AI-assisted text and take full responsibility for the final content.

Results

Participant characteristics and data flow

The available records contained 69 completed baseline PVT-EEG datasets: 32 healthy controls and 37 clinical participants. Pre-enrollment screening counts could not be reconstructed from the available records. Endpoint-specific samples were behavior n = 32/37, primary-reference ERP n = 25/33, stimulus-locked time-frequency n = 24/31, all valid post-response beta n = 24/33, paired beta n = 20/19, and single-trial P3 n = 26/34 (3,231 trials with complete model data). Controls were all undergraduates; the clinical group included 35 undergraduates and two postgraduates.

All 69 participants contributed behavioral data. The 5,512 target events yielded 5,508 valid trials, including 5,010 normal responses, 437 lapses, 52 anticipatory responses, and 9 omissions. Primary-reference ERP inference included 25 healthy controls and 33 GAD-HSS participants. Primary time-frequency inference included 24 healthy controls and 31 GAD-HSS participants for stimulus-locked measures. All valid post-response beta included 24 and 33 participants, respectively. The late-versus-early beta contrast required both periods and included 20 healthy controls and 19 GAD-HSS participants. Participant characteristics are summarized in Table 1.

Table 1
Characteristics of the university-student sample

Characteristic	Healthy controls (n = 32)	GAD-HSS (n = 37)	p value
Age, years	21.28 ± 0.92	20.00 ± 2.09	0.00148
Female/male, n	17/15	28/9	0.0757
Weight, kg	59.02 ± 12.54	60.38 ± 8.74	0.610
Undergraduate/postgraduate, n	32/0	35/2	0.495
Current smoking, yes/no	0/32	0/37	1.000
Alcohol history, yes/no	4/28	8/29	0.359
GAD-7 score	2.75 ± 2.63	11.05 ± 3.15	< 0.001
HAMA score	4.16 ± 3.34	21.03 ± 7.60	< 0.001
Weekday sleep, h/night	7.54 ± 1.13	5.73 ± 0.42	< 0.001
Weekend sleep, h/night	9.39 ± 1.17	7.10 ± 1.18	< 0.001

Values are mean ± SD unless otherwise indicated. Continuous variables used two-sided Welch tests. Categorical variables used two-sided Fisher exact tests. GAD-7, Generalized Anxiety Disorder 7-item scale; HAMA, Hamilton Anxiety Rating Scale. Sleep duration was retrospectively self-reported.

Sustained-attention performance

Median RT was longer in GAD-HSS than in healthy controls (353 vs 320 ms). The primary unadjusted group difference was 33.00 ms (95% CI 12.35–53.65; g = 0.745; p = 0.00218; p_{FDR} = 0.00873; Fig. 1A). In the age- and sex-adjusted HC3 model, the difference was 31.48 ms (95% CI 3.46–59.50; p = 0.0283; sensitivity-family p_{FDR} = 0.0449), representing 4.6% attenuation. Huber regression produced a 27.74-ms estimate (95% CI 5.05–50.44; p = 0.0166).

RT coefficient of variation did not differ in primary or adjusted analyses (adjusted coefficient = 0.0182, 95% CI − 0.220 to 0.256; p_{FDR} = 0.879; Fig. 1B). Lapse proportion was higher in GAD-HSS (10.02% vs 5.61%) but did not survive the primary family correction (p_{FDR} = 0.0606). An age- and sex-adjusted NB2 sensitivity model estimated an IRR of 1.887 (95% CI 1.110–3.205; p = 0.0189; dispersion parameter α = 0.986). Because this additional overdispersion model was not included in the adjusted behavioral FDR family, the lapse finding was treated as a secondary, model-dependent result. Principal behavioral and psychophysiological findings are summarized in Table 2.

Table 2
Principal behavioral and psychophysiological findings

Domain	Endpoint	Sample	Primary unadjusted result	Age- and sex-adjusted result
Behavior	Median RT	32/37	Δ = 33.00 ms (12.35–53.65); g = 0.745; p_{FDR} = 0.00873	Δ = 31.48 ms (3.46–59.50); p = 0.0283; p_{FDR} = 0.0449
Behavior	RT coefficient of variation	32/37	Δ = −0.043; g = −0.112; p_{FDR} = 0.648	β = 0.0182 (− 0.220 to 0.256); p = 0.879; p_{FDR} = 0.879
Behavior	Lapse outcome	32/37	Δ proportion = 0.044; g = 0.474; p_{FDR} = 0.0606	NB2 IRR = 1.887 (1.110–3.205); p = 0.0189
Time-on-task	Participant reaction-time progress slope	32/37	Δ = 101.25 ms; g = 0.588; p_{FDR} = 0.0373	β = 100.26 ms (16.56–183.95); p = 0.0196; p_{FDR} = 0.0449
Time-on-task	Trial-level group × progress	69; 5,377 trials	β = 0.0169; SE = 0.0115; p_{FDR} = 0.259	β = 0.01687 (− 0.00560 to 0.03934); p = 0.141
ERP	P1 all valid	25/33	Δ = -0.480 μV; p_{FDR} = 0.417	β = -0.570 μV; p = 0.163; p_{FDR} = 0.325
ERP	N2 all valid	25/33	Δ = 0.733 μV; p_{FDR} = 0.487	β = 0.020 μV; p = 0.987; p_{FDR} = 0.987
ERP	P3/LPC all valid	25/33	Δ = -2.249 μV; p_{FDR} = 0.325	β = -3.408 μV; p = 0.049; p_{FDR} = 0.129
Time-frequency	Poststimulus theta	24/31	Δ = -0.222 dB; p_{FDR} = 0.645	β = -0.235 dB; p = 0.481; p_{FDR} = 0.902
Time-frequency	Alpha suppression	24/31	Δ = 0.193 dB; p_{FDR} = 0.906	β = 0.363 dB; p = 0.550; p_{FDR} = 0.912
Response beta	Late-versus-early	20/19	Δ = −0.833 dB; g = −0.896; p_{FDR} = 0.0431	β = −0.931 dB (− 1.741 to − 0.122); p = 0.0254; family p_{FDR} = 0.0778; global p_{FDR} = 0.363
Single trial	P3 predicting log RT	60; 3,231 trials	β = −0.02384; SE = 0.00593; p = 5.89×10^{-5}; p_{FDR} = 0.000648	β = −0.02332 (− 0.03929 to − 0.00736); p = 0.00420

RT, reaction time; ERP, event-related potential; LPC, late positive complex; FDR, Benjamini–Hochberg false discovery rate; HC, healthy controls. Samples are HC/GAD-HSS participant counts unless total participants and trials are stated. Effects are GAD-HSS minus healthy controls. Original unadjusted

analyses remained primary. Adjusted models included centered age and sex. The NB2 lapse model used log(valid trials) as an offset and was an additional overdispersion sensitivity analysis. It was not included in the adjusted behavioral FDR family; therefore, the reported NB2 p value is unadjusted. Full models and robustness analyses are provided in Additional file 1.

Time-on-task findings

Participant-level reaction-time progress slopes were more positive in GAD-HSS (57.52 ± 149.1 ms) than in healthy controls (− 43.73 ± 191.8 ms). The primary unadjusted difference was 101.25 ms (95% CI 17.51–185.00; g = 0.588; p = 0.0187; p_{FDR} = 0.0373; Fig. 1C). The adjusted HC3 estimate was 100.26 ms (95% CI 16.56–183.95; p = 0.0196; sensitivity-family p_{FDR} = 0.0449). Huber regression yielded 46.63 ms (95% CI 14.14–79.12; p = 0.00491), indicating sensitivity of magnitude to influential values.

The trial-level mixed model did not corroborate a differential progress effect. The primary group × progress coefficient was β = 0.0169 (SE = 0.0115; p = 0.141; p_{FDR} = 0.259). The adjusted correlated random-slope model gave an almost identical interaction (β = 0.01687, 95% CI − 0.00560 to 0.03934; p = 0.141). Participant-level summaries therefore suggested greater time-on-task slowing, but the trial-level analysis remained nonsignificant.

Averaged event-related potential findings

No averaged ERP group contrast survived FDR correction (Fig. 2; Table 2). Differences between GAD-HSS and healthy controls for all valid endpoints were − 0.480 μV for P1 (g = -0.374, p_{FDR} = 0.417), -0.899 μV for N1 (g = -0.374, p_{FDR} = 0.417), 0.733 μV for N2 (g = 0.187, p_{FDR} = 0.487), and − 2.249 μV for P3/LPC (g = -0.380, p_{FDR} = 0.325). Age- and sex-adjusted models were fitted for all 42 principal ERP, response-ERP, and time-frequency contrasts; none survived its original family correction or global correction.

Slow-versus-fast and late-versus-early ERP contrasts were also nonsignificant after correction. The largest uncorrected pattern was the N2 slow-versus-fast contrast (difference = 3.518 μV, g = 0.649, p = 0.0552, p_{FDR} = 0.300). Response-locked readiness, motor, and post-response potential contrasts were nonsignificant. These results do not establish equivalence, but they do not support broad averaged ERP abnormalities.

Time-frequency and post-response beta findings

No overall group difference survived FDR correction for poststimulus theta, alpha suppression, prestimulus alpha, delta inter-trial phase coherence, or theta inter-trial phase coherence (Fig. 3). All primary family-adjusted p values were at least 0.645. All valid stimulus-locked endpoints included 24 healthy controls and 31 GAD-HSS participants. No corresponding age- and sex-adjusted contrast survived its original family correction.

The all valid post-response beta group contrast was nonsignificant in both primary and adjusted analyses. The paired late-versus-early difference was − 0.833 dB (95% CI − 1.426 to − 0.240; g = − 0.896; p = 0.00719; family p_{FDR} = 0.0431; Fig. 4), but it failed global EEG correction (p_{FDR} = 0.302) and weakened under average reference (p = 0.0972). With age and sex included, the estimate was − 0.931 dB (95% CI − 1.741 to − 0.122; p = 0.0254) and failed its six-test family (p_{FDR} = 0.0778) and global 42-contrast correction (p_{FDR} = 0.363).

Single-trial P3-reaction time associations

The single-trial analysis table contained 3,266 EEG rows from 60 primary-reference participants. Both fitted P3 models used 3,231 trials because 35 rows lacked a standardized foreperiod and/or previous-trial log-RT derivative. In the primary random-intercept model, larger within-participant P3 amplitudes predicted shorter log RTs (β = −0.02384, SE = 0.00593; p = 5.89×10^{-5}; p_{FDR} = 0.000648). The adjusted correlated random-slope model retained the association (β = −0.02332, 95% CI − 0.03929 to − 0.00736; p = 0.00420), while the P3 × group interaction remained nonsignificant (β = 0.01595; p = 0.144).

Prestimulus alpha and P1 did not predict RT after correction, and their interactions with group were nonsignificant. Prestimulus alpha did not reliably predict lapse probability. The P3 result therefore represented a shared dimensional brain–behavior relationship rather than a group-specific mechanism.

Sensitivity analyses

Age- and sex-adjusted sensitivity models covered the full behavior sample and each endpoint-specific EEG sample. The adjusted median-RT and participant-slope findings retained direction and family-level support. The trial-level progress interaction remained nonsignificant, no adjusted averaged EEG contrast survived family FDR, and the adjusted P3 effect remained negative with a nonsignificant group interaction.

The beta contrast was directionally consistent across participant-level robustness analyses, but it was attenuated under average reference and did not survive global correction across the principal EEG contrasts. Complete rank-based, permutation, bootstrap, robust-regression, near-threshold, leave-one-out, and inclusion analyses are reported in Additional file 1: Table S7 and Figure S4.

Under average reference, the beta change difference was − 0.442 dB (n = 20/24, 95% CI − 0.968 to 0.084; p = 0.0972). Global BH-FDR across the 42 primary EEG contrasts yielded p_{FDR} = 0.302. In the age- and sex-adjusted primary-reference analysis, the beta estimate remained negative but failed both family correction (p_{FDR} = 0.0778) and adjusted global correction (p_{FDR} = 0.363).

The age- and sex-adjusted correlated P3 random-slope model converged without a singular fit. The within-participant P3 effect remained negative (β = -0.02332, 95% CI -0.03929 to -0.00736; p = 0.00420), whereas the P3 × group interaction remained nonsignificant (β = 0.01595, p = 0.144). The uncorrelated

random-slope and participant-clustered models gave similar conclusions. Age and sex coefficients were nonsignificant.

Discussion

Principal findings

The main finding was slower sustained-attention responding in the combined GAD-HSS group. The 33.00-ms median-RT difference changed little after age and sex adjustment. Evidence for a differential vigilance decrement was inconsistent because participant-level and trial-level analyses did not converge. None of 42 principal averaged EEG group contrasts survived family and global correction. Larger within-participant P3 amplitudes predicted faster trial-level responses across groups, but the P3 association did not differ significantly by group. The post-response beta result remained exploratory.

Sustained-attention slowing

The behavioral result is compatible with reduced processing efficiency during sustained monitoring. Attentional control theory proposes that anxiety can increase the resources needed to maintain performance, yielding slower or more effortful responses without uniformly increasing overt errors [2]. Because the PVT makes limited demands on working memory, rule switching, and emotional appraisal, the difference concerns response efficiency in a simple vigilance context rather than threat processing or complex executive control. Its clinical meaning should remain bounded: a 33.00-ms laboratory difference does not establish a particular degree of academic or everyday impairment.

Habitual short sleep offers a plausible, inseparable contributor. PVT performance is highly sensitive to cumulative sleep restriction [9,10], and recent work supports its sensitivity relative to other objective alertness tests [36]. Poor sleep is also clinically intertwined with stress and anxiety in student populations [3]. Because short sleep was part of the clinical-group definition and the design lacked factorial comparison groups, the observed slowing cannot be assigned to GAD alone. The appropriate inference is that students in the combined clinical group showed reduced sustained-attention efficiency relative to healthy student controls.

Time-on-task findings

Participant-level and trial-level models summarized time-on-task at different levels. The adjusted participant-level estimate was 100.26 ms, but Huber regression gave 46.63 ms, indicating influence from extreme values. The adjusted trial-level interaction was almost identical to the primary estimate and remained nonsignificant. Thus, a participant-summary difference was present without convergent evidence for a group-specific trial-level vigilance decrement.

Averaged electroencephalographic findings

No corrected group-level difference was identified for P1, N1, N2, P3/LPC, response-locked potentials, theta, alpha, or phase consistency. This conclusion was unchanged after age and sex adjustment across all 42 principal EEG contrasts. The nonsignificant findings do not demonstrate electrophysiological equivalence: endpoint-specific exclusions reduced precision, and confidence intervals remained compatible with moderate effects for some endpoints.

Behavioral performance and averaged EEG measures can differ because averaging reduces transient or temporally variable responses. Prior vigilance studies have related task duration and sleep loss to EEG and ERP changes [18–20,22], but those findings may not generalize to this combined GAD-HSS comparison. Task structure, sleep exposure, clinical composition, and ERP processing also influence estimates [23,24]. The present data support a behavioral difference without a corrected averaged EEG group difference.

P3-reaction time association

Across 3,231 trials with complete model data from 60 participants, larger within-participant P3 amplitudes predicted shorter reaction times after adjustment for task progress, foreperiod, and previous-trial reaction time. Because P3 was standardized within participant, this coefficient captures dimensional trial-to-trial brain–behavior coupling rather than a between-person amplitude difference. The association remained negative in the age- and sex-adjusted random-slope and participant-clustered sensitivity models, while group moderation was unsupported. This shared relationship may reflect variation in attention or evidence accumulation, but it does not establish a causal neural mechanism or a group-specific biomarker.

Exploratory beta finding

The post-response beta late-versus-early contrast was significant within the primary unadjusted response-beta family, but it weakened under average reference, failed global EEG correction, and did not survive the age- and sex-adjusted response-beta family. Although post-response beta may reflect motor-system state after action 11,12,16, the present contrast does not establish a mechanism or biomarker. Fatigue-related motor adaptation, response strategy, baseline drift, trial retention, and unmeasured differences remain plausible alternatives. The finding should therefore be regarded as exploratory and hypothesis-generating.

Clinical and research implications

For clinical practice and research, the behavioral result suggests that objective sustained-attention testing may complement reported concentration difficulties in young adults with GAD and habitual short sleep. The P3 association was shared across groups and is not a diagnostic marker or treatment target. Future studies should measure sleep objectively, include GAD-normal-sleep and non-GAD-short-sleep comparison groups, assess functional outcomes, and use prospective designs to clarify the course and clinical relevance of the observed slowing.

Strengths and limitations

Strengths included a clinically diagnosed GAD-HSS group, recruitment of controls from the same university population, a low-complexity sustained-attention task, and integration of participant-level behavior, averaged ERPs, time-frequency measures, response-related beta, and single-trial models. Fixed processing windows, regions of interest, trial-retention rules, and endpoint-family FDR correction reduced analytic flexibility. Negative findings and reference and multiplicity sensitivities were retained rather than selectively omitted.

Several limitations constrain inference. The cross-sectional, single-center university sample limits causal interpretation and generalizability, and endpoint-specific EEG exclusions reduced precision. The groups differed in age and sex distribution; although the age- and sex-adjusted median-RT estimate was similar to the primary unadjusted result, residual and unmeasured confounding cannot be excluded. GAD diagnosis was based on ICD-10 clinical assessment by a trained licensed clinical psychologist. A named standardized structured diagnostic instrument was not documented in the available study materials, which may limit direct procedural reproducibility across studies. Pre-enrollment screening counts and the recruitment start referenced in earlier project documents could not be independently reconstructed, although all PVT-EEG recordings included in the present analysis were acquired after registry entry. The exact programmed foreperiod and feedback duration could not be recovered from the available task files. Sleep duration was retrospectively self-reported without diaries, actigraphy, or polysomnography. The absence of GAD-normal-sleep and non-GAD-short-sleep groups prevents separation of anxiety and sleep effects, and the minimal overlap in weekday sleep precluded an interpretable sleep-adjusted comparison. The number of EEG measures and analysis families increased inferential complexity; family-based FDR control and a global 42-contrast sensitivity correction were used, but they do not eliminate all multiplicity concerns. Nonsignificant EEG findings do not establish equivalence. Finally, the beta analysis used a reduced paired sample and produced a reference-sensitive result that did not survive global correction.

Conclusions

University students with clinically diagnosed GAD and self-reported habitual short sleep showed slower sustained-attention responses than healthy controls. No averaged EEG group difference survived the relevant family and global multiplicity corrections. Within-person P3 amplitude tracked trial-to-trial response speed across groups, and the P3-reaction time relationship did not differ significantly by group. The result is best interpreted as a shared performance-related association rather than a disorder-specific biomarker. The design cannot separate effects of GAD from those of habitual short sleep.

Abbreviations

CI
confidence interval
EEG
electroencephalography

ERP
event-related potential
FDR
false discovery rate
GAD
generalized anxiety disorder
GAD-7
Generalized Anxiety Disorder 7-item scale
HAMA
Hamilton Anxiety Rating Scale
HC
healthy control
HSS
habitual short sleep
ICA
independent component analysis
ICD-10
International Statistical Classification of Diseases and Related Health Problems,10th Revision
ITPC
inter-trial phase coherence
LPC
late positive complex
PVT
psychomotor vigilance test
ROI
region of interest
RT
reaction time.

Declarations

Ethics approval and consent to participate

The study was conducted in accordance with the Declaration of Helsinki. The Ethics Committee of the Third Affiliated Hospital of Henan University of Chinese Medicine approved the study (2023SH-147). All participants provided written informed consent.

Consent for publication

Not applicable. The manuscript contains no identifiable individual-level information.

Availability of data and materials

The datasets generated and/or analysed during the current study are not publicly available due to privacy, ethical, participant-consent, and institutional data-governance restrictions but are available from the corresponding author on reasonable request. Analysis code is also available from the corresponding author on reasonable request, subject to applicable ethical and institutional requirements.

Competing interests

The authors declare that they have no competing interests.

Funding

This work was supported by the Third Batch of Top-notch Talents Program in Traditional Chinese Medicine of Henan Province and the Science and Technology Research Project of Henan Province (grant no. 252102311264).

Authors' contributions

CS contributed to conceptualization, methodology, software, formal analysis, visualization, and writing the original draft. HL contributed to investigation, data curation, validation, and writing the original draft. LW contributed to investigation, data curation, formal analysis, and writing the original draft. SL contributed to investigation, resources, and reviewing and editing the manuscript. MW contributed to investigation and data curation. YG contributed to investigation and resources. JW contributed to investigation and project administration. JG contributed to methodology, validation, and reviewing and editing the manuscript. WF contributed to resources, project administration, and reviewing and editing the manuscript. XC contributed to conceptualization, methodology, supervision, funding acquisition, and reviewing and editing the manuscript. YW contributed to conceptualization, supervision, project administration, funding acquisition, and reviewing and editing the manuscript.

Acknowledgements

The authors thank all participants and the research and technical staff who assisted with recruitment, EEG acquisition, and study coordination.

Authors' information

Not applicable.

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Figures

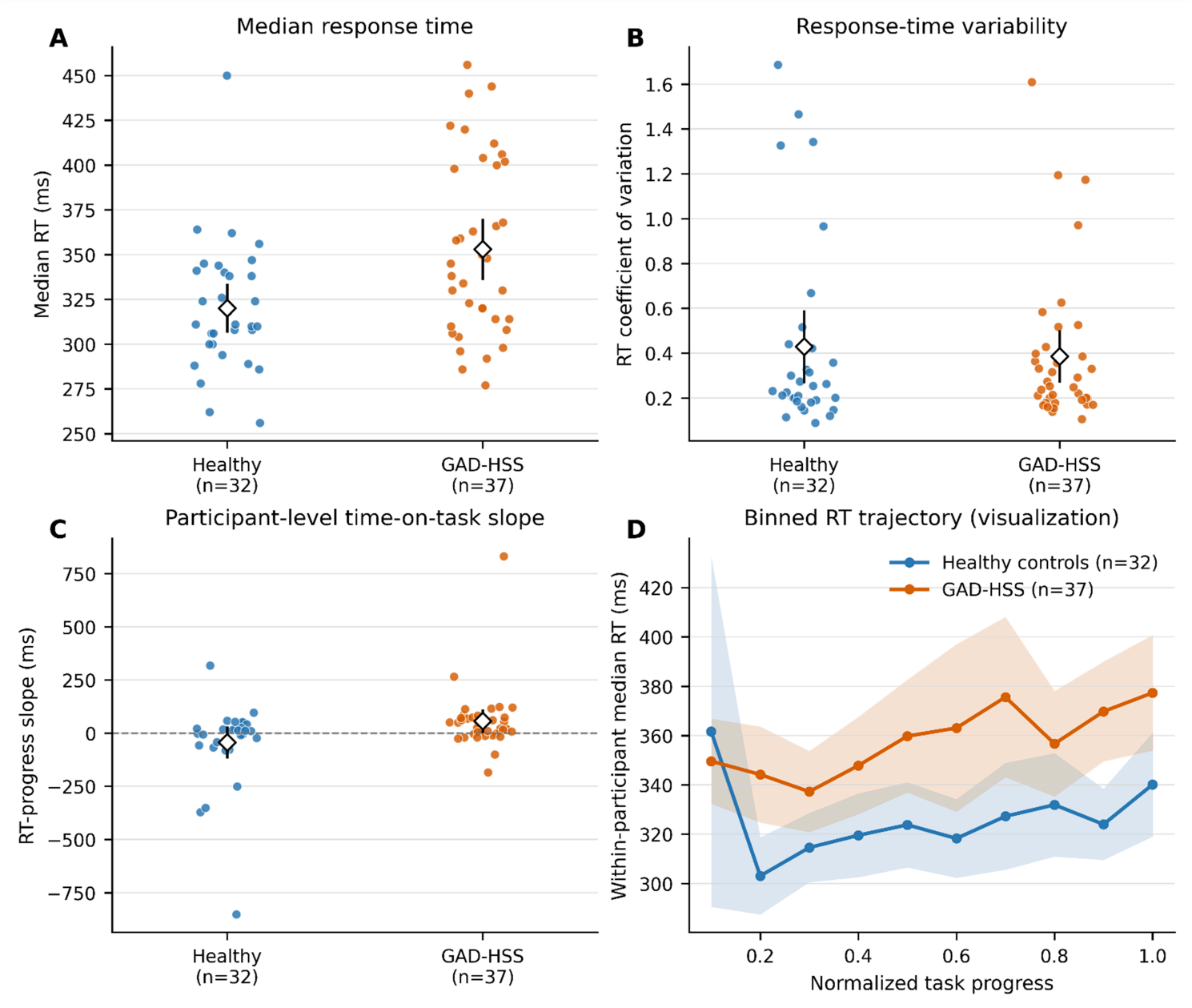


Figure 1

Sustained-attention performance and time-on-task. Panels A-C show participant-level median RT, RT coefficient of variation, and reaction-time progress slope with raw points, group summaries, and 95% confidence intervals. Panel D shows mean within-participant median RT across ten normalized task progress bins for visualization only; formal inference used the participant-level slope analysis and trial-level mixed model. Healthy controls n = 32; GAD-HSS n = 37.

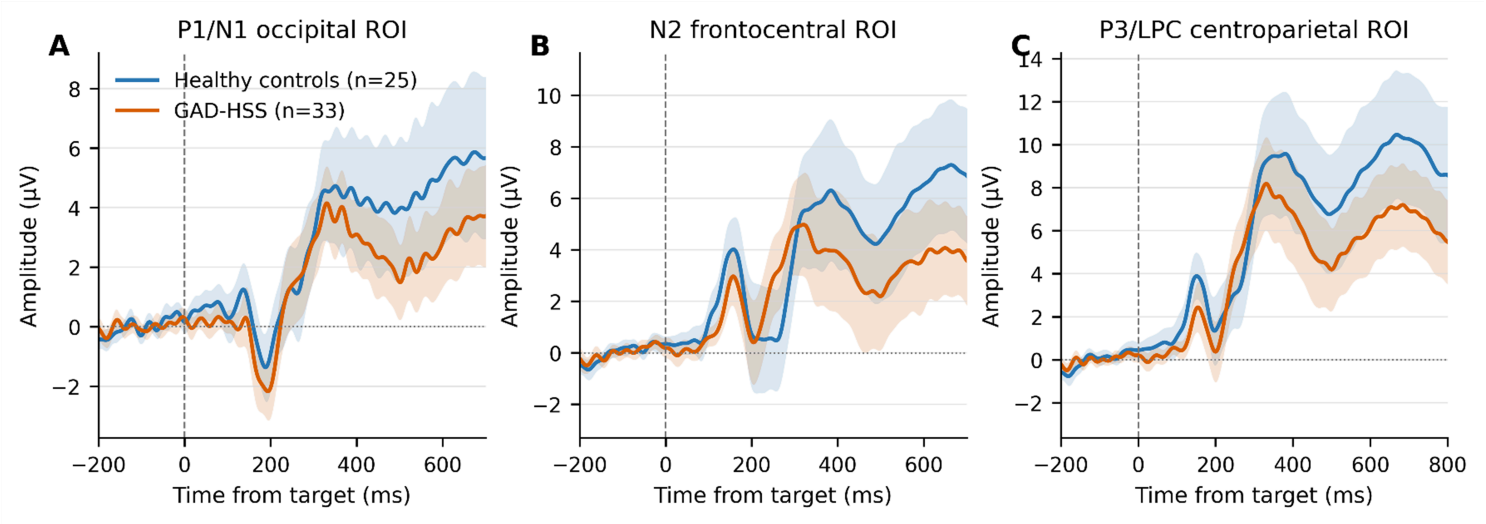


Figure 2

Stimulus-locked event-related potentials during the psychomotor vigilance test. Grand-average waveforms and 95% confidence intervals are shown for the P1/N1, N2, and P3/LPC regions of interest. Waveforms include the same participants contributing to each all valid endpoint.

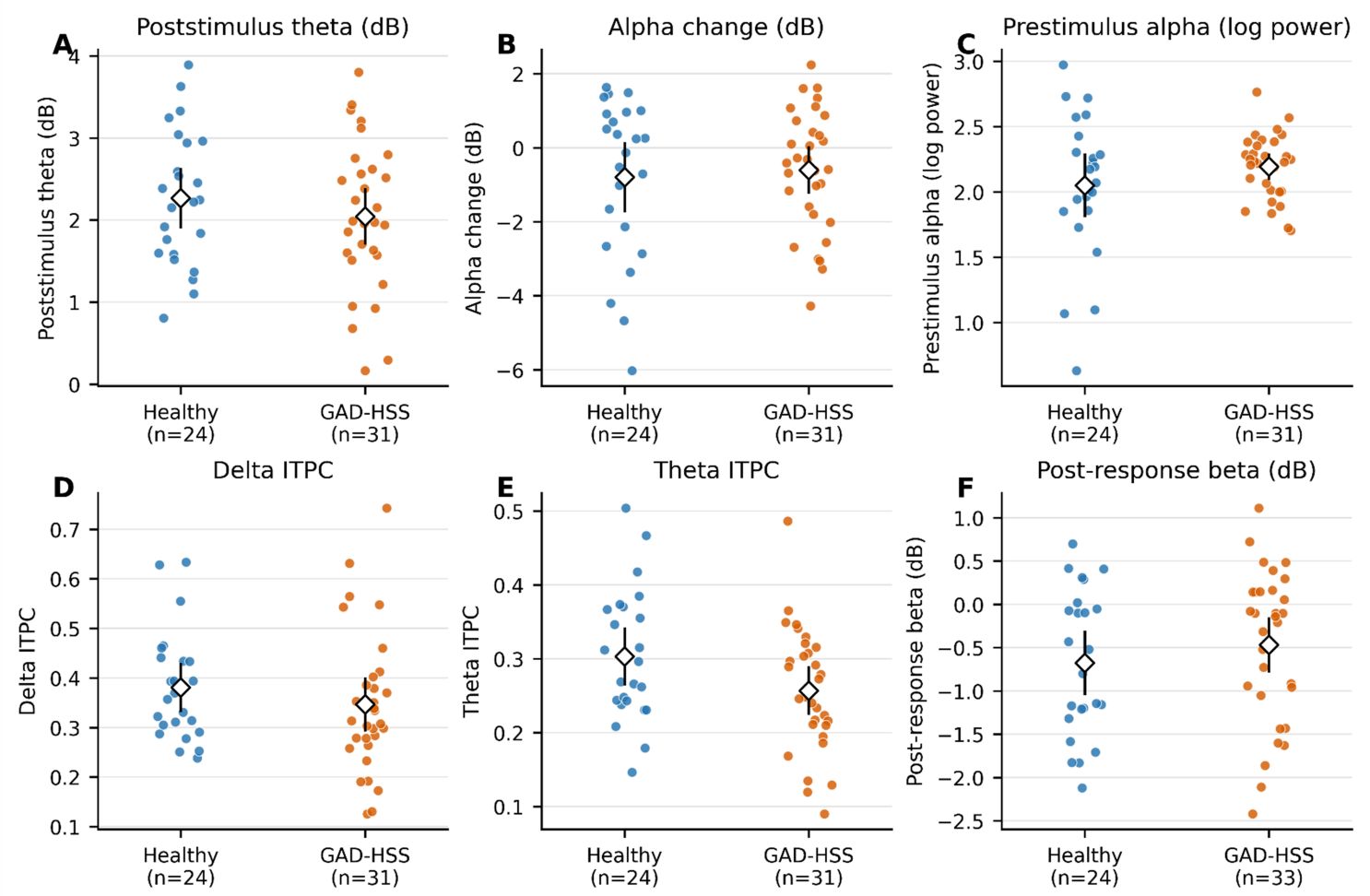


Figure 3

Time-frequency region-of-interest summaries. Raw participant values, group means, and 95% confidence intervals are shown for poststimulus theta, alpha suppression, prestimulus alpha, delta and theta inter-trial phase coherence, and all valid post-response beta. Stimulus-locked measures include 24 healthy controls and 31 GAD-HSS participants. Post-response beta includes 24 healthy controls and 33 GAD-HSS participants because response-locked retention criteria differed.

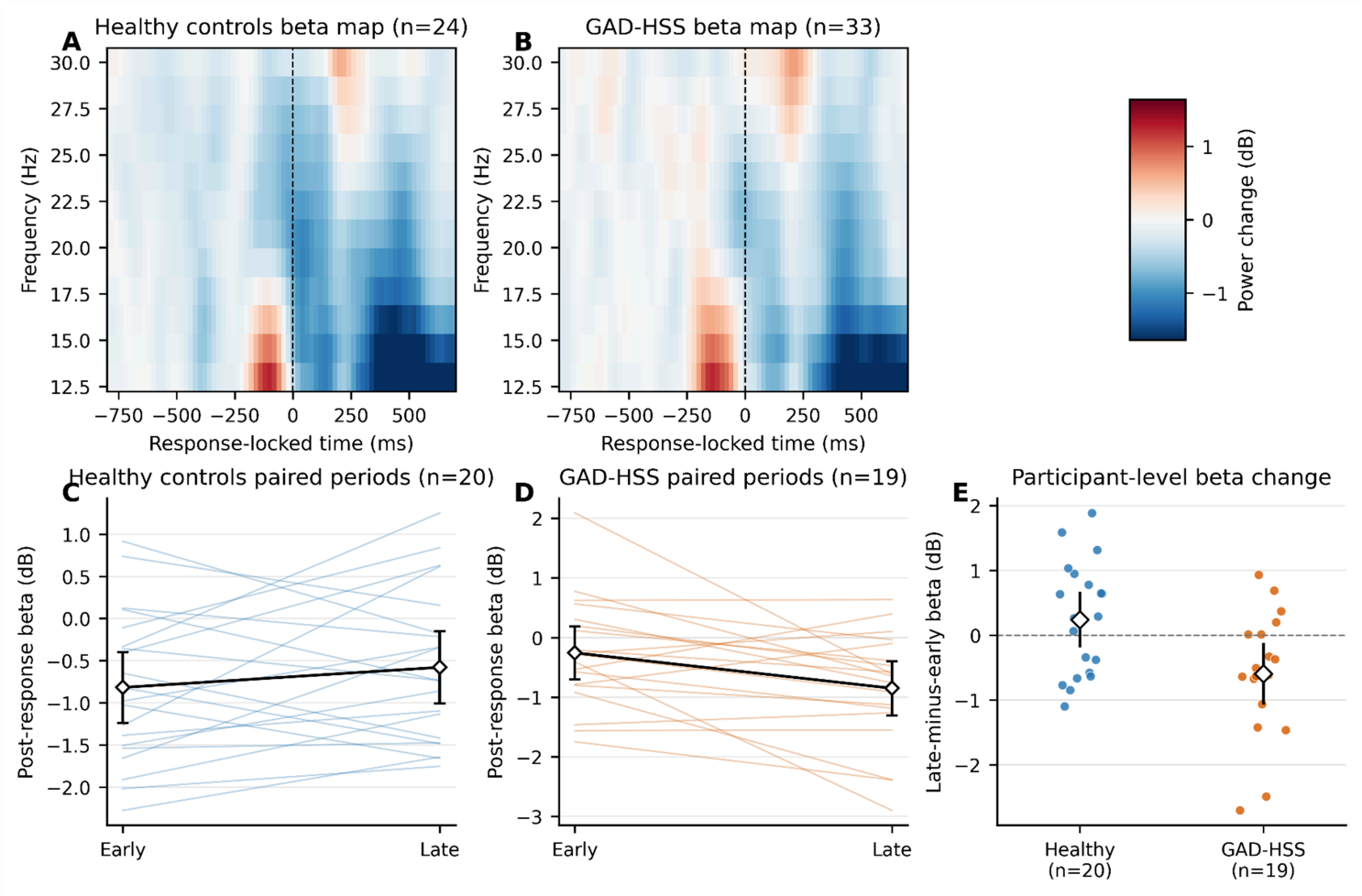


Figure 4

Response-related beta dynamics. Panels A and B show response-locked beta maps for records meeting all valid response-locked quality and trial criteria (healthy controls n = 24; GAD-HSS n = 33). Panels C and D show paired early and late beta values (healthy controls n = 20; GAD-HSS n = 19). Panel E shows participant-level late-versus-early beta changes with group summaries and 95% confidence intervals. The contrast was significant within the primary unadjusted response-beta family but was reference-sensitive, did not survive global EEG correction, and was not family-FDR significant after age and sex adjustment.

Supplementary Files

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