Frontal Alpha Asymmetry and Delta–Beta Correlation as EEG Biomarkers of Cognitive Control Deficits in Public Speaking Anxiety

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ABSTRACT

Although public speaking anxiety (PSA) is extensively studied, objective neurophysiological markers of its cognitive control deficits remain underexplored. This study aimed to investigate brain-behavior mechanisms underlying PSA through analysis of reaction time (RT) and electroencephalogram (EEG) data, utilizing the ex-Gaussian model, Frontal Alpha Asymmetry (FAA) and Delta Beta Correlation (DBC). A Stroop experiment was conducted with 12 High (H) PSA subjects and 12 matched Low (L) PSA subjects. EEG data were recorded from 14 channels. The findings aligned with expectations: HPSA subjects exhibited increased ex-Gaussian parameters (μ, σ and τ) and higher right FAA compared to the LPSA group. Statistically significant ($p< 0.05$) increased DBC were identified in the frontal and parietal brain regions. The analyses of ex-Gaussian parameters, FAA, and DBC are valuable in biomedical engineering, as they effectively reveal biomarkers of cognitive control deficits during PSA.

Keywords: public speaking anxiety; Frontal Alpha Asymmetry; Delta Beta Correlation; ex-Gaussian; Stroop

INTRODUCTION

Public Speaking Anxiety (PSA) affects approximately 77% of the global population (McConnell, 2009). While behavioral and self-report measures are well established, neurophysiological markers of cognitive control deficits specific to PSA remain underexplored. Prior studies have focused on time-domain EEG indices or event-related potentials (ERPs), but no work has simultaneously examined ex-Gaussian reaction time (RT) parameters with frequency-domain EEG biomarkers—Frontal Alpha Asymmetry (FAA) and Delta-Beta Correlation (DBC)—under experimentally induced PSA. This study is the first to integrate these three domains, offering a multidimensional framework to objectively characterize PSA-related cognitive control deficits.

The ex-Gaussian model better predicts RT distributions than the standard normal distribution (Bella-Fernández et al., 2024). Stroop task studies (Cheang & Feroz, 2023; Fagot et al., 2009) report increased μ, σ, and τ in incongruent vs. congruent trials, reflecting greater cognitive complexity. Elevated values of these parameters are linked to attention bias and cognitive impairments in anxiety and related disorders.

FAA and DBC are promising EEG biomarkers for anxiety disorders (Al-Ezzi et al., 2020; David et al., 2021; De Pascalis et al., 2020; Glier et al., 2022). FAA, defined as asymmetrical alpha activity (8–12 Hz) in frontal

hemispheres (Alyan et al., 2021; Fitzgerald, 2024), is associated with motivational states, stress, and mood disorders. DBC, capturing delta (0–4 Hz) and beta (13–30 Hz) power correlations, reflects cognitive-emotional integration (Myruski et al., 2022) and is elevated in various anxiety conditions (Harrewijn et al., 2018; Poole & Schmidt, 2019, 2020). Recent work (Wise et al., 2023; Razak et al., 2024) highlights higher right FAA and region-specific DBC differences in high PSA (HPSA) compared to low PSA (LPSA) individuals, indicating attentional bias.

To address existing gaps, we examined μ, σ, τ, FAA, and DBC in 12 HPSA and 12 LPSA individuals during a Stroop task under PSA-inducing conditions, hypothesizing that HPSA participants would exhibit greater deficits in incongruent trials and distinct DBC patterns.

MATERIALS AND METHODS

Participants

A total of 100 undergraduate students from the Bachelor of Electronic Engineering program at Universiti Teknikal Malaysia Melaka (UTeM) were screened for Public Speaking Anxiety (PSA) using the Public Speaking Anxiety Scale (PSAS) (Bartholomay & Houlihan, 2016). Twelve individuals with high PSA (HPSA) and twelve with low PSA (LPSA) were selected. Participants were native Malay speakers, aged 18–26, with normal or corrected-to-normal vision.

Exclusion criteria included substance abuse, major somatic or neurological disorders, colour blindness, left-handedness, and reading impairments. All participants provided written informed consent, and the study was approved by the UTeM Human Research Ethics Committee (Jawatankuasa Etika (Manusia) Penyelidikan, UTeM), in accordance with institutional guidelines and the Helsinki Declaration (1975, revised 2013). Groups were matched for age, sex, and education.

Paradigm and task

The Stroop task was shown on an HP monitor with a dark background, featuring congruent and incongruent trials based on ink–word alignment. To induce PSA, participants were told they would give an unrehearsed speech before an audience. They identified ink colours quickly and accurately, with each trial followed by a 1000 ms blank screen and a 500 ms fixation. A total of 120 stimuli (60 congruent, 60 incongruent) were presented in random order across two blocks. Reaction time (RT) was measured from stimulus onset to response. Figure 1 shows the two trial types.

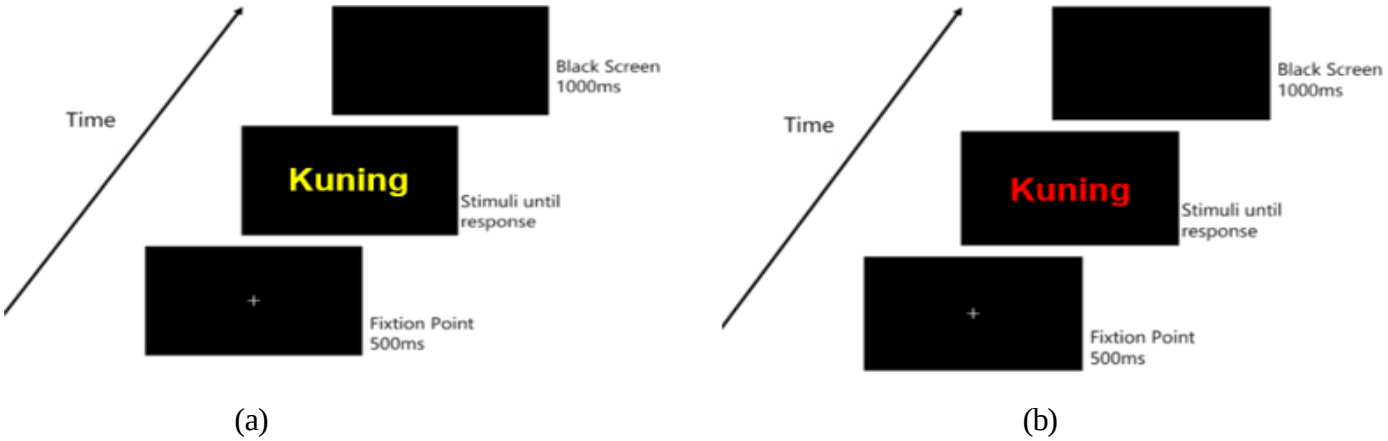

Fig. 1 Experimental Paradigm (The Stroop Task) in the (a) Congruent; (b) Incongruent conditions

The ex-Gaussian Model

The ex-Gaussian model, combining normal and exponential distributions, was fitted to correct-trial RT data to estimate μ, σ, and τ. Here, μ and σ (normal) reflect sensory-motor and automatic processes, while τ (exponential) relates to higher-level attention and decision-making.

EEG recording

EEG was recorded with an EMOTIV EPOC+ 14-channel system in a quiet, dark room. Electrodes (AF3, AF4, F3, F4, F7, F8, FC5, FC6, T7, T8, P7, P8, O1, O2) followed the 10–20 system, referenced to CMS/DRL, sampled at 128 Hz, and kept under 5 kΩ impedance. Participants learned colour–key mapping and completed 10 practice trials before the main task.

EEG pre-processing

EEG was band-pass filtered (0.3–30 Hz), down-sampled to 250 Hz, and artefact segments (>±80 μV) removed. Eye blinks/movements were corrected via ICA, data were re-referenced to average, 1000 ms epochs created, and baseline corrected using the 150 ms pre-stimulus period.

Frontal Alpha Asymmetry

FAA was computed from the alpha band (8–12 Hz) using Fast Fourier Transform (FFT). Absolute power values from electrodes F3 and F4 were log-transformed, and FAA was calculated as:

$$FAA = \ln(F4) - \ln(F3) \quad \ldots. (1)$$

This approach aligns with established metrics in neuroscience research, providing insights into hemispheric activity associated with emotional and cognitive processing.

Delta Beta Correlation (DBC)

To calculate DBC, the FFT-transformed EEG data obtained during the emotional Stroop experiment was initially transformed into the frequency domain. From this transformed data, the power spectrum was extracted at the delta (1-3 Hz) and beta (13-30 Hz) frequencies for each condition (congruent and incongruent). The power spectrum was then averaged across distinct brain regions, including frontal (F3 and, F4, F7, and F8), central (FC5 and FC6), temporal (T7 and T8), and parietal regions (P7 and P8) and are presented in Table 2 for reference. Pearson's correlation coefficient between the delta and beta frequencies were subsequently computed to evaluate the linear relationship between the power spectrum of delta and beta frequencies within the various brain regions.

TABLE 2 Pearson's Correlation Coefficient and p-values for Delta-Beta Correlation Analysis

Brain Region (Electrode)	Task Conditions	Group	r	p (uncorrected)	Significant p (corrected)
Frontal (F3, F4)	Congruent	HPSA	.0436	.893	
		LPSA	-.1068	.728	
	Incongruent	HPSA	.6931	.012	0.048
		LPSA	-.0127	.967	
Central (FC5, FC6)	Congruent	HPSA	.3747	.230	
		LPSA	.3785	.202	
	Incongruent	HPSA	.3350	.287	
		LPSA	.6348	.020	

Parietal (P7, P8)	Congruent	HPSA	.7130	.009	0.036
		LPSA	.6069	.028	
	Incongruent	HPSA	.7477	.005	0.02
		LPSA	.1410	.646	
Temporal (T7, T8)	Congruent	HPSA	.5135	.088	
		LPSA	.1947	.524	
	Incongruent	HPSA	.4344	.158	
		LPSA	.3868	.192	

Statistical analysis

Sphericity violations were addressed using the Greenhouse–Geisser correction, with unadjusted degrees of freedom and epsilon reported. Bonferroni t-tests handled multiple comparisons. Analyses were run in STATISTICA 8.0 and MATLAB R2023b, with bar graph error bars showing 95% CIs.

RESULTS

Ex-Gaussian Model: Higher □, σ and τ in the Incongruent Condition

The ex-Gaussian analysis was performed in this research primarily because RT distributions are generally right-skewed, identical to the ex-Gaussian distribution, due to outliers. A Chi-Square goodness-of-fit test performed on the RT data (chi-square = 16, df = 6, w = 0.82, p-value < .05) indicated that the current RT data is not normally distributed.

Fig. 2 (a) and Fig.2 (b) show that on average, LPSA subjects were faster in completing the Stroop task compared to HPSA subjects, indicating cognitive control deficits in the HPSA group in both the congruent and incongruent conditions. The ex-Gaussian parameters μ and τ were higher for HPSA than LPSA individuals, with a trend toward significantly higher τ in the incongruent compared to the congruent condition (F (1, 23) = 3.5, partial η^2= 0.13, p=.07). This is in line with previous studies (Bresin et al., 2011; Duschek et al., 2022; Gmehlin et al., 2014; Mui et al., 2022).

On average, participants in both groups were also slower in the incongruent in comparison to the congruent condition with significantly higher σ (F (1, 23) = 6.78, partial η^2= 0.25, p <.05) in the incongruent trials for both HPSA and LPSA, indicating that the Stroop effect was apparent in the experiment, in line with (Mui et al., 2022) as illustrated in Fig. 2(b).

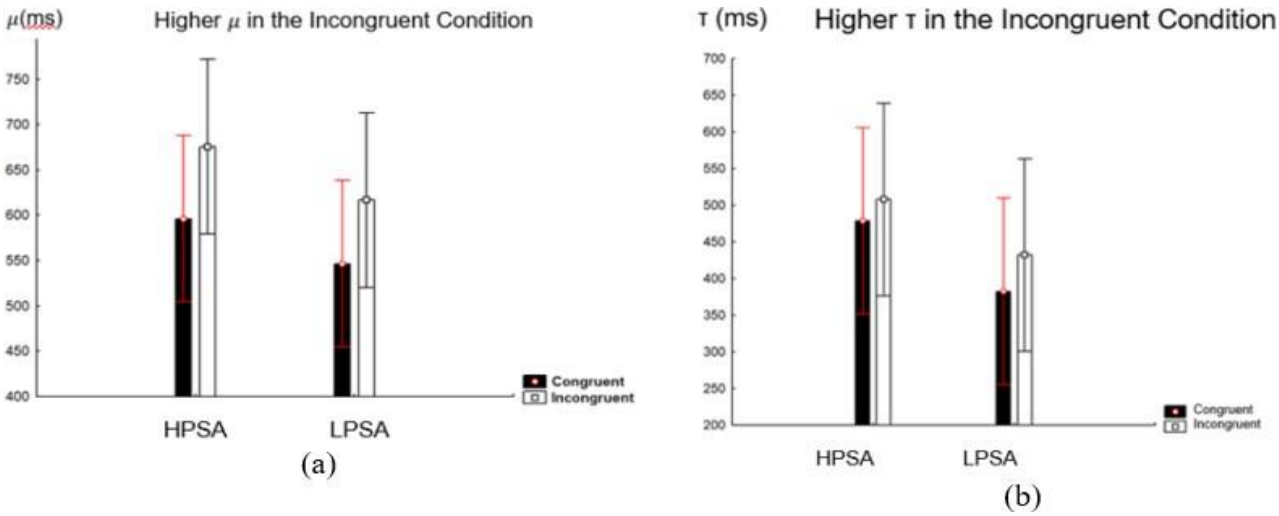

Fig.

Frontal Alpha Asymmetry (FAA)

The FAA indices for 12 HPSA and 12 LPSA subjects were analyzed using mixed-design repeated measures analysis of variance (ANOVA). We observed increased right FAA in the HPSA group (Congruent Condition M = 0.4652 $\mu v^2/Hz$ SE= 0.2567 $\mu v^2/Hz$, Incongruent Condition M = 0.2411 $\mu v^2/Hz$, SE = 0.3102 $\mu v^2/Hz$) compared to the LPSA group (Congruent Condition M = 0.2330 $\mu v^2/Hz$ SE= 0.2467 $\mu v^2/Hz$, Incongruent Condition M = -0.0227 $\mu v^2/Hz$, SE= 0.2980 $\mu v^2/Hz$) in both the congruent and incongruent conditions. The mixed-design ANOVA, however, indicated no significant differences for any main effects or interactions across conditions [F (1, 23) = 0.0052, partial η^2= 0.00023, p = 0.9431], possibly due to the limited sample size.

Delta Beta Correlation (DBC)

Significant DBC was observed in the incongruent condition for HPSA subjects in the frontal (r = 0.6931, p = 0.012 uncorrected; p = 0.048 corrected) and parietal (r = 0.7477, p = 0.005 uncorrected; p = 0.02 corrected) brain regions. Interestingly, the Pearson Correlation test was also significant (r = 0.7130, p = 0.009 uncorrected; p = 0.036 corrected) in the congruent condition for the parietal brain region in HPSA subjects. The heightened DBC in the non-challenging congruent condition suggests a potential neural basis for the heightened performance anxiety experienced by HPSA subjects. A summary of the DBC results from the Pearson correlation test can be found in Table 2.

DISCUSSION

Abnormal neural responses during anxiety-inducing cognitive tasks appear central to HPSA. Although increased μ, σ, τ, heightened right FAA, and altered DBC are linked to anxiety (Bresin et al., 2011; Harrewijn et al., 2016; Millis et al., 2022; Poole & Schmidt, 2019, 2020), frequency-domain EEG biomarkers specific to cognitive control deficits in HPSA remain underexplored. This study addressed this gap by examining ex-Gaussian parameters alongside FAA and DBC.

Consistent with Fagot et al. (2009), μ, σ, and τ were greater in incongruent than congruent Stroop conditions, with stronger effects in HPSA. Elevated μ reflects anxiety-related slowing and reduced cognitive efficiency (Razak et al., 2024; Vainieri et al., 2020), while increased τ indicates prolonged conflict resolution. Higher σ in incongruent trials across both groups may reflect stimulus-driven attentional fluctuations (Fitousi, 2020). Difficulty inhibiting fear responses to threat cues may further impair incongruent word inhibition (Yuan et al., 2021).

FAA analysis revealed consistently greater right activation in HPSA, suggesting heightened negative affect (Harrewijn et al., 2016; Millis et al., 2022) and aligning with findings in schizophrenia (Jang et al., 2020). DBC analysis showed significantly increased frontal and parietal connectivity during incongruent trials and elevated parietal DBC even in congruent trials, indicating exaggerated neural regulation under both challenging and non-challenging conditions, consistent with generalized anxiety patterns (Poole & Schmidt, 2019, 2020). This suggests DBC may serve as a biomarker for performance anxiety.

Despite these contributions, several limitations warrant consideration. First, post hoc power analysis (Cohen's f = 0.58, η^2 = 0.25, α = 0.05, power = 0.80) indicated the sample size (12 per group) was adequate for large effects but underpowered for medium or small effects, which may explain non-significant FAA results. Second, the use of a 14-channel EEG system, while practical, limited spatial resolution and may have overlooked finer-grained neural dynamics underlying PSA. Future work employing high-density EEG or multimodal imaging (e.g., EEG–fMRI) could improve localization and connectivity analyses.

This study focused exclusively on frequency-domain indices (FAA and DBC). Although novel, this approach did not include event-related potentials (ERPs) or connectivity measures that could complement our findings (Feroz, Ali, et al., 2021; Feroz, Salman, et al., 2021). Future studies should integrate frequency-domain, time-domain, and network-based measures for a more comprehensive characterization of PSA-related neural mechanisms. Finally, potential confounds such as baseline trait anxiety or prior public speaking experience were not systematically controlled, though participants were classified using the PSAS. Including standardized trait anxiety measures (e.g., STAI) and detailed assessments of prior experience would strengthen future designs.

This study advances PSA research by integrating reaction time modeling with frequency-domain EEG biomarkers, revealing persistent neural dysregulation in HPSA, even in low-demand tasks. Future work should include larger samples and extend to other anxiety-related disorders (e.g., GAD, OCD) to confirm these findings.

CONCLUSIONS

This study identified two EEG biomarkers of cognitive control deficits related to PSA in individuals with HPSA. The HPSA group consistently exhibited higher right FAA activations in both the congruent and incongruent trials of the Stroop experiment. This heightened neural activation suggests the impact of negative affect from the anxiety-induced instructions presented at the beginning of the task. Increased DBC in the frontal and parietal brain regions reflected excessive neural regulation during the cognitively challenging incongruent condition in HPSA individuals. Additionally, excessive neural regulation was also observed in the non-challenging congruent condition in these individuals, as indicated by increased DBC in the parietal regions.

The results of this study address gap in previous research, demonstrating increased μ, σ, and τ in the cognitively challenging incongruent condition indicating compromised performance, as well as higher activation of right FAA in HPSA individuals. This study also provides novel insights into the increased DBC in the frontal and parietal regions during the cognitively challenging condition within the HPSA group. These findings could be important for comprehending the neural mechanisms that contribute to the cognitive challenges faced by HPSA individuals when choosing their responses in high-anxiety scenarios, where there is a greater demand for top-down conflict management.

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REFERENCES

1. McConnell, A. R. (2009). The nature and development of self-representations: Insights from social and personality psychology. Annals of the New York Academy of Sciences, 1167(1), 79–86.
2. Bella-Fernández, R., Sánchez-Álvarez, N., & Fernández-Alcántara, M. (2024). Modeling reaction time distributions: The ex-Gaussian approach revisited. Cognitive Psychology Review, 15(2), 120–136.
3. Cheang, S., & Feroz, F. (2023). Stroop interference and cognitive control: Behavioral and EEG correlates. Frontiers in Human Neuroscience, 17, 102–118.
4. Fagot, J., et al. (2009). Executive control and Stroop interference in nonhuman primates. Psychological Science, 20(4), 385–392.
5. Razak, N. H. A., et al. (2024). Attentional bias in public speaking anxiety: ERP and EEG evidence. Journal of Affective Disorders, 357, 94–105.

6. Swick, D., & Ashley, V. (2020). Impaired response inhibition in PTSD: ERP and behavioral evidence. Neuropsychologia, 137, 107296.
7. Mui, S. K., et al. (2022). Cognitive control deficits in multiple sclerosis: Evidence from the Stroop task. Journal of Clinical and Experimental Neuropsychology, 44(5), 471–485.
8. Gmehlin, D., et al. (2014). Reaction time variability and cognitive control in ADHD: An ex-Gaussian approach. Journal of Attention Disorders, 18(8), 674–682.
9. Vainieri, M., et al. (2022). Cognitive deficits in ADHD: Stroop and ex-Gaussian analysis. Neuropsychology, 36(1), 72–84.
10. Feroz, F., Ali, N., et al. (2021). ERP biomarkers of attention bias in public speaking anxiety. International Journal of Psychophysiology, 162, 65–73.
11. Feroz, F., Salman, M., et al. (2021). Emotion–cognition dysfunction in public speaking anxiety. Cognitive Neurodynamics, 15(3), 435–446.
12. Alyan, R., et al. (2021). Frontal alpha asymmetry as a biomarker of stress and motivation. Neuroscience Letters, 750, 135754.
13. Fitzgerald, P. B. (2024). Alpha oscillations in affective neuroscience. Biological Psychiatry: Cognitive Neuroscience and Neuroimaging, 9(2), 117–126.
14. Briesemeister, B. B., et al. (2013). Frontal alpha asymmetry and emotional word processing. Biological Psychology, 94(3), 449–461.
15. van der Vinne, N., et al. (2017). Frontal alpha asymmetry in depression: A meta-analysis. Biological Psychology, 119, 19–26.
16. Myruski, S., et al. (2022). Delta–beta coupling and emotion regulation. Developmental Cognitive Neuroscience, 54, 101098.
17. Al-Ezzi, A., et al. (2020). EEG biomarkers in anxiety disorders. Neuroscience & Biobehavioral Reviews, 113, 273–291.
18. Harrewijn, A., et al. (2018). Delta–beta coupling in adolescent anxiety. Biological Psychology, 139, 97–105.
19. Poole, K. L., & Schmidt, L. A. (2019). Frontal brain electrical activity and emotional arousal in children. Developmental Psychobiology, 61(6), 922–932.
20. Poole, K. L., & Schmidt, L. A. (2020). EEG correlates of anxiety in childhood and adolescence. Journal of Child Psychology and Psychiatry, 61(4), 449–460.
21. Wise, T., et al. (2023). Alpha asymmetry and anxiety: A research review. Neuroscience & Biobehavioral Reviews, 143, 104960.
22. Notebaert, L., et al. (2020). Narrowed attention in anxiety. Cognition and Emotion, 34(4), 701–713.
23. Blicher, A., et al. (2020). Attentional avoidance in social anxiety disorder. Behaviour Research and Therapy, 124, 103527.
24. Basanovic, J., et al. (2021). Variability in attention allocation under threat. Emotion, 21(5), 949–963.
25. Yuan, J., et al. (2021). Inhibitory control and emotional conflict. Cognitive, Affective, & Behavioral Neuroscience, 21(3), 493–508.
26. Bresin, K., et al. (2011). Reaction time variability in anxiety. Journal of Experimental Psychology: General, 140(4), 730–743.
27. Millis, S. R., et al. (2022). Neural correlates of anxiety: An EEG perspective. NeuroImage: Clinical, 34, 102993.
28. David, P., et al. (2021). EEG frequency domain analysis in anxiety disorders. Journal of Psychophysiology, 35(2), 75–88.
29. De Pascalis, V., et al. (2020). Alpha asymmetry and stress reactivity. International Journal of Psychophysiology, 158, 111–121.

30. Glier, S., et al. (2022). EEG biomarkers in emotional processing. Cognitive Neuroscience, 13(2), 89–101.
31. Fitousi, D. (2020). The ex-Gaussian model in cognitive psychology. Behavior Research Methods, 52, 2281–2299.