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# Severity-dependent alterations in default mode network subnetwork connectivity in social anxiety disorder: an EEG study

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# Abstract

Social anxiety disorder (SAD) is associated with excessive self-focused processing and heightened sensitivity to social threat, yet how large-scale brain networks vary across symptom severity remains unclear. The Default Mode Network (DMN), central to self-referential and internally oriented cognition, represents a key system for examining severity-related neural alterations in SAD.

In this study, we investigated severity-dependent changes in DMN functional connectivity using electroencephalography (EEG). Resting-state and anxiety-loaded EEG data were collected from healthy controls and individuals with mild, moderate, and severe SAD. Functional connectivity was quantified using Phase-Locking Value (PLV) across multiple frequency bands and analyzed within anatomically defined DMN subnetworks, including frontal, fronto-parietal, posterior, and interhemispheric components.

The results revealed systematic, severity-dependent alterations in DMN connectivity. At rest, alpha-band synchronization within frontal and posterior DMN hubs was relatively preserved, whereas fronto-parietal and interhemispheric connectivity progressively declined with increasing SAD severity, particularly in higher-frequency bands. Under anxiety-loaded conditions, these alterations were amplified, with pronounced reductions in long-range connectivity while core DMN hubs remained comparatively resilient.


# Introduction

Social anxiety disorder is a common and disabling psychiatric condition, affecting approximately 7% of the population annually and a substantially larger proportion across the lifespan (Stein & Stein, 2008). It is characterized by persistent fear of negative social evaluation, avoidance of social situations, and marked impairment in interpersonal relationships, academic or occupational performance, and overall quality of life. Although SAD has traditionally been conceptualized as a disorder driven primarily by heightened emotional reactivity, growing evidence from cognitive neuroscience suggests that it is more accurately understood as a disorder of large-scale brain network dysfunction, particularly involving systems responsible for self-referential processing, emotional regulation, and cognitive control (Etkin & Wager, 2007; Menon, 2011).

Among these systems, the DMN has received increasing attention due to its central role in internally oriented cognition. The DMN is most active during rest and attenuates during externally focused, goal-directed tasks (Raichle et al., 2001; Buckner et al., 2008). It supports a broad range of mental processes, including self-referential thinking, autobiographical memory, future simulation, social cognition, emotional evaluation, and mind-wandering (Andrews-Hanna et al., 2010; Spreng et al., 2009). In the context of SAD, disruptions in DMN function have been proposed to underline core clinical features such as excessive self-focus, maladaptive rumination, anticipatory anxiety, and heightened self-criticism (Zhao et al., 2007; Liao et al., 2010).

Importantly, the DMN is not a unitary network but consists of interacting subnetworks that contribute to distinct aspects of internal cognition. The midline core, anchored in the medial prefrontal cortex (mPFC) and posterior cingulate cortex (PCC)/precuneus, is crucial for self-referential awareness and emotional monitoring (Gusnard et al., 2001; Northoff et al., 2006). Medial temporal components, including the hippocampus and parahippocampal regions, support autobiographical memory and emotional contextualization (Addis et al., 2007), while dorsal medial prefrontal and lateral parietal regions contribute to social inference, semantic processing, and self–other distinction (Cavanna & Trimble, 2006; Spreng et al., 2013). Disruptions within and between these subnetworks may therefore differentially shape symptom severity and cognitive–emotional imbalance in SAD.

Functional connectivity, defined as the statistical dependence between spatially distributed neural signals, provides a powerful framework for investigating DMN integrity. While functional magnetic resonance imaging (fMRI) has been widely used to characterize DMN abnormalities in SAD, electroencephalography (EEG) offers complementary advantages through its high temporal resolution. EEG-based FC measures, such as PLV, enable the examination of frequency-specific neural synchronization across Delta, Theta, Alpha, and Beta bands—oscillatory regimes that are differentially involved in attention, memory, emotional regulation, and executive control (Basar et al., 2001; Klimesch, 2012).

Recent EEG studies show that frequency-specific DMN connectivity alterations are related to anxiety severity. Increased frontal Alpha connectivity is associated with excessive self-focus, while reduced Beta-band connectivity reflects impaired top-down emotional regulation (Engel & Fries, 2010; Fonzo et al., 2017). Posterior DMN regions, including the PCC and precuneus, appear relatively stable under stress, whereas disrupted interhemispheric connectivity may impair affective–cognitive integration and contribute to social anxiety symptoms (Spreng et al., 2010; Davidson & Hugdahl, 1995). Resting-state EEG markers have also been shown to predict trait anxiety, supporting the utility of EEG-based approaches (Tamari Shalamberidze et al., 2025).

Despite these advances, existing studies have largely examined DMN connectivity in a global or region-specific manner, with limited attention to how interactions between distinct DMN subnetworks evolve across SAD severity levels and emotional states. In particular, the relationship between subnetwork-level DMN connectivity, frequency-specific dynamics, and graded SAD symptomatology remains insufficiently characterized, especially using EEG-based approaches.

Motivated by the complex architecture of DMN and the heterogeneous manifestations of SAD, the present study adopts a subnetwork-level EEG functional connectivity approach to characterize DMN dynamics across varying levels of SAD severity. Functional connectivity is examined across four key subnetworks—frontal (self-referential processing), fronto-parietal (attention and cognitive control), posterior (memory-based emotional regulation), and interhemispheric (bilateral integration)—under both resting-state and anxiety-loaded conditions. By identifying frequency- and subnetwork-specific connectivity patterns associated with symptom severity and stress exposure, this work aims to advance

understanding of the neurophysiological mechanisms underlying SAD and to evaluate the potential of EEG-derived DMN metrics as biomarkers for disorder progression and clinical intervention.

# Theoretical Background

Functional Connectivity

Functional connectivity reflects the temporal synchronization between spatially distributed brain regions and provides a framework for understanding large-scale neural communication underlying cognition and emotion (Friston, 2011; Fox & Raichle, 2007). Unlike structural connectivity, FC captures dynamic interactions that vary with cognitive state and emotional context, making it particularly relevant for investigating social anxiety disorder (SAD). EEG-based FC offers high temporal resolution, enabling the examination of rapid neural coordination across frequency bands associated with attention, emotional regulation, and cognitive control (Stam & van Straaten, 2012). Common EEG FC measures, including Phase-Locking Value (PLV) and coherence, quantify oscillatory synchronization across delta, theta, alpha, and beta bands, each linked to distinct functional processes (Basar et al., 2001; Nunez & Srinivasan, 2006). In this study, FC is estimated between EEG electrode pairs representing key network nodes, allowing frequency-specific assessment across resting and anxiety-loaded states and enabling identification of connectivity patterns associated with social anxiety severity and stress-related network vulnerability.

PLV as a Measure of Functional Connectivity

In this study, we employed PLV as the estimator of FC. PLV is a widely used phase-based synchronization measure that captures the consistency of phase differences between two EEG signals over time (Lachaux et al., 1999). It reflects how stably the oscillatory phases of two signals are locked together, independent of their amplitude, thus making it particularly suitable for assessing non-linear and transient interactions within brain networks.

Mathematically, PLV is defined as the absolute value of the average of the unit phase differences across trials or time points. The value ranges from 0 to 1, where 0 indicates completely random phase relationships and 1 represents perfect phase synchronization. This measure is particularly robust to volume conduction and signal amplitude fluctuations, which are common concerns in EEG analyses. The formula for PLV between two signals x(t) and y(t) is given by:

$$PLV = \left| \frac{1}{N} \sum {}_{n} \exp \left( j \left[ \Phi_{xn}(t) - \Phi_{Yn}(t) \right] \right) \right|$$

where $\Phi_{xn}(t)$ and $\Phi_{Yn}(t)$ represent the instantaneous phases of signals x and y at time t and over N samples or trials. The exponential term represents the phase difference at each point, and the average over time quantifies the stability of phase locking.

The choice of PLV in our study allows for a fine-grained and temporally sensitive analysis of the synchrony among key regions of the DMN. By applying PLV across multiple frequency bands—delta, theta, alpha, and beta—we capture distinct functional contributions of each oscillatory regime to cognitive and emotional processing in both control and SAD participants.

Classification of Functional Connectivity Values

To facilitate a structured interpretation of functional connectivity (FC) patterns, FC values were categorized according to a standardized classification scheme summarized in Table 1. This scheme distinguishes four levels of connectivity strength—very low, low, medium, and high—based on correlation magnitude and is intended to reflect the degree of neural synchronization and functional integration between Default Mode Network (DMN) nodes (Friston, 1994; Rubinov & Sporns, 2010). High connectivity typically characterizes interactions among core DMN regions, such as the medial prefrontal cortex and posterior cingulate cortex, supporting coherent self-referential processing, whereas progressively lower connectivity levels indicate reduced integration and emerging dysconnectivity (Cohen, 1988; Mukaka, 2012; Zalesky et al., 2012). Consistent with prior neuropsychiatric research, markedly reduced or near-absent FC is interpreted as clinically meaningful network fragmentation (Whitfield-Gabrieli & Ford, 2012). This classification framework enables systematic tracking of connectivity degradation across SAD severity levels. As noted in Table 1, these thresholds are adopted for descriptive and visualization purposes only and are not used for inferential statistical testing, following common practice in network neuroscience (Stam et al., 2007; Rubinov & Sporns, 2010).

Table 1
FC Strength Classification Scheme

| **Connectivity Strength** | **($r$)-value Range** | **Functional Implication** |
|---|---|---|
| High Connectivity | $r \geq 0.5$ | This level is usually observed between core DMN regions (PCC and mPFC) indicate robust functional integration supporting coherent self-referential processing. However, abnormally elevated or rigid DMN coupling may promote maladaptive rumination in anxiety and mood disorders as discussed by Whitfield-Gabrieli & Ford (2012). |
| Medium Connectivity | $0.3 \leq r < 0.5$ | Falls within the “medium effect” range proposed by Cohen (1988) and considered moderate correlation in medical statistics guidelines by Mukaka (2012). This level is typically observed between principal DMN hubs and secondary DMN regions in healthy individuals. Balanced correlations around 0.3–0.5 are described as the conventional baseline for meaningful neuroimaging connectivity, reflecting flexible information exchange and normal network function (Zalesky et al., 2012). |
| Low Connectivity | $0.1 \leq r < 0.3$ | Aligns with the “small effect” category of Cohen (1988) and interpreted as weak association in clinical research frameworks (Mukaka, 2012). Such reduced correlations suggest early-stage compromise in coherence among interacting DMN regions. Low DMN region coupling is commonly linked with diminished processing efficiency, attentional instability, or developmental network vulnerabilities (Zalesky et al., 2012). |
| Very Low / Dysconnectivity | $r < 0.1$ | Values approaching zero are methodologically regarded as negligible relationships (Cohen, 1988). Near-absent correlations between distributed DMN regions represent a breakdown of network coherence. Whitfield-Gabrieli & Ford (2012) emphasize that profound DMN fragmentation is clinically meaningful in severee neuropsychiatric disorders such such as schizophrenia or ASD, where unified self-representation and social cognition are markedly impaired. |

Functional Connectivity Mapping of DMN Subnetworks

The DMN comprises several interconnected subnetworks that support diverse cognitive and emotional functions, including self-referential thought, emotional regulation, autobiographical memory, and social cognition (Buckner et al., 2008; Andrews-Hanna et al., 2010). In this study, FC among EEG electrodes were categorized into four principal DMN domains, each reflecting a distinct aspect of self-awareness and social information processing relevant to SAD. This classification allows for a more granular investigation of how anxiety-related dysregulation manifests within specific DMN circuits.

# Frontal Connectivity (Self-Referential Processing & Executive Control)

Frontal connectivity reflects synchronized activity within the frontal DMN, primarily involving the medial prefrontal cortex (mPFC) and bilateral dorsal superior frontal gyri (dSFG). This subnetwork supports self-referential processing and executive monitoring, with the mPFC acting as a central hub for introspection, emotional self-awareness, and social valuation (Gusnard et al., 2001; Northoff et al., 2006; Buckner et al., 2008). Functional coupling between the mPFC and dSFG integrates self-focused cognition with executive control, enabling adaptive regulation of behavior in social contexts. In healthy individuals, this connectivity supports flexible control of internal thoughts and emotions, whereas altered or inefficient regulation within this circuit has been associated with excessive rumination, heightened self-consciousness, and biased social threat evaluation in SAD (Zhao et al., 2007; Liao et al., 2010). Figure 1-a shows the connection within the fronto subnetwork.

# Fronto-Parietal Connectivity (Cognitive Control Over the DMN & Attention Regulation)

The fronto-parietal subnetwork links prefrontal control regions with posterior DMN hubs, particularly the posterior cingulate cortex (PCC) and precuneus, supporting flexible shifts between internally focused and goal-directed cognition. This circuitry underlies attentional control, emotion regulation, and disengagement from ruminative thought patterns in social contexts (Andrews-Hanna et al., 2010; Leech & Sharp, 2014; Menon, 2011). Key connections between dorsal superior frontal gyri (dSFG), medial prefrontal cortex, PCC, and bilateral angular gyri enable top-down modulation of self-referential activity and multimodal attentional integration. Disruptions in this network may reduce attentional flexibility and promote persistent negative self-focus and social-cognitive dysregulation. Figure 1-b shows the connection within the fronto-parietal subnetwork.

# Posterior Connectivity (Memory-Based Emotional Regulation & Social Identity Formation)

The posterior DMN, centered on the posterior cingulate cortex (PCC) and precuneus, supports autobiographical memory retrieval, affective valuation, and context-dependent emotional regulation (Greicius et al., 2003; Cavanna & Trimble, 2006). By integrating self-relevant memories into ongoing emotional and social processing, this subsystem contributes to emotional learning, resilience, and social identity formation (Zhang et al., 2019). In this study, posterior DMN connectivity was indexed using Pz–POz and P3–P4 electrode pairs, representing posterior midline integration and bilateral parietal interactions, respectively. These scalp-level measures are interpreted as functional proxies of posterior DMN activity rather than direct markers of deep cortical sources. Disruptions in posterior connectivity have been linked to heightened negative autobiographical memory salience, impaired affective

contextualization, and reduced emotional resilience, which are central to maladaptive self-referential processing in social anxiety disorder (Zhang et al., 2019).

## Interhemispheric Connectivity (Coordination Between Hemispheres & Emotional Processing)

Effective emotional regulation relies on coordinated interaction between the two cerebral hemispheres, enabling integration of executive control, linguistic processing, and affective evaluation (Davidson & Hugdahl, 1995). Interhemispheric functional connectivity supports balanced analytic–emotional processing and flexible modulation of emotional responses in socially demanding contexts.

In this study, interhemispheric FC was assessed using cross-hemispheric frontal and parietal electrode pairs (Fp1–P4, Fp1–P7, and P3–P4), capturing long-range integration between prefrontal regulatory regions and posterior associative areas. These connections index large-scale bilateral coordination rather than localized cortical activity (Fig. 1-c). Disruptions in interhemispheric connectivity may impair the integration of regulatory and affective processes, contributing to emotional dysregulation and heightened social threat sensitivity in social anxiety disorder. Additionally, bilateral frontal connectivity (Fp1–Fp2) reflects interhemispheric prefrontal communication essential for higher-order cognitive control and reappraisal; its attenuation may limit effective top-down regulation of anxiety.

## Methods

# Participants

Participants were recruited through large-scale screening using the Social Interaction Anxiety Scale (SIAS) Social Interaction Anxiety Scale. From an initial pool of 502 respondents, 84 participants were selected and classified into four groups based on SIAS scores: healthy controls (HC; <20), mild (< 40), moderate (< 60), and severe (≥ 60) social anxiety disorder (SAD). All participants were right-handed, medication-free, in good physical and mental health, and had normal or corrected-to-normal vision. Four participants were excluded due to data quality issues.

Age did not differ significantly across groups ($F(1,87) = 2.664$, $p = 0.054$, $\eta^2 = 0.093$). An a priori power analysis confirmed that the sample size provided 80% statistical power at $\alpha = 0.05$. The study was approved by the Medical Research Ethics Committee of the Royal College of Medicine Perak, Malaysia, and written informed consent was obtained from all participants in accordance with the Declaration of Helsinki.

## Experimental Procedure

EEG data were collected under two conditions: resting state and anxiety-loaded. During the resting-state conditions, participants sat comfortably in a dim, quiet, sound-attenuated room with their eyes closed.

Anxiety was induced using a standardized public speaking task, a well-established paradigm for eliciting social-evaluative stress in SAD research (Stein & Stein, 2008; Etkin & Wager, 2007). Following a five-minute anticipation period, participants delivered a speech lasting three to five minutes in front of a neutral panel. During the speech, participants were unexpectedly interrupted and instructed to sit silently for two minutes, a manipulation designed to enhance social-evaluative stress. EEG was recorded continuously throughout anticipation, performance, and post-interruption phases.

## EEG Acquisition and Preprocessing

EEG signals were recorded using a 32-channel EEG system (ANT Neuro, Netherlands) positioned according to the international 10–20 system (Nunez & Srinivasan, 2006). Signals were referenced to CPz and grounded at AFz during acquisition. Data were sampled at 2048 Hz, down-sampled to 256 Hz, and band-pass filtered between 0.4 and 50 Hz. Electrode impedance was maintained below 10 kΩ.

EEG signals were re-referenced to the common average. Artifacts related to eye blinks, muscle activity, or movement were identified using automated procedures and visual inspection and removed prior to analysis (Stam & van Straaten, 2012). Only artifact-free data segments were retained.

Although EEG does not permit direct measurement of subcortical activity, key Default Mode Network (DMN) hubs, including the posterior cingulate cortex (PCC) and medial prefrontal cortex (mPFC), are known to project reliably to cortical generators detectable at the scalp level, supporting their use as functional proxies (Das et al., 2022).

# Functional Connectivity Analysis

Functional connectivity was estimated using Phase-Locking Value (PLV), which quantifies the temporal consistency of phase differences between EEG signals (Lachaux et al., 1999). PLV values range from 0 to 1, with higher values indicating stronger phase synchronization. Compared with amplitude-based measures, PLV provides robustness against volume conduction and transient amplitude fluctuations (Vinck et al., 2011).

PLV was computed between electrode pairs across delta, theta, alpha, and beta frequency bands, which are associated with distinct cognitive and emotional processes (Basar et al., 2001; Klimesch, 2012). Connectivity was evaluated separately for resting-state and anxiety-loaded conditions to assess baseline DMN organization and stress-related modulation.

# DMN Subnetwork Definition

To investigate subnetwork-specific alterations in Default Mode Network (DMN) functional connectivity, EEG electrode pairs were organized into four anatomically and functionally motivated DMN subnetworks: frontal, fronto-parietal, posterior, and interhemispheric. This subdivision follows established DMN topology and electrophysiological and neuroimaging evidence linking scalp-level activity to cortical DMN

hubs, including the medial prefrontal cortex (mPFC) and posterior cingulate cortex (PCC) (Buckner et al., 2008; Andrews-Hanna et al., 2010; Das et al., 2022).

# Frontal DMN Subnetwork

The frontal DMN subnetwork represents cortical projections of the medial prefrontal cortex and adjacent superior frontal regions implicated in self-referential processing, emotional appraisal, and executive monitoring (Gusnard et al., 2001; Northoff et al., 2006; Buckner et al., 2008). This subnetwork was defined using electrode pairs among Fp1, Fp2, F3, F4, Fz, FC1, and FC2, capturing both medial and bilateral frontal integration. Functional coupling within this subnetwork reflects internal self-focused cognition and regulatory control, which are frequently altered in social anxiety disorder (Zhao et al., 2007; Liao et al., 2010).

# Posterior DMN Subnetwork

The posterior DMN subnetwork corresponds to the PCC and precuneus, which play a central role in autobiographical memory, emotional contextualization, and internally directed cognition (Greicius et al., 2003; Cavanna & Trimble, 2006). Posterior connectivity was indexed using electrode pairs among Pz, P3, P4, POz, O1, and O2, representing midline and bilateral parietal–occipital integration. These scalp-level measures serve as functional proxies for posterior DMN hubs rather than direct measurements of deep cortical generators (Vogt & Laureys, 2005; Das et al., 2022).

# Fronto-Parietal DMN Subnetwork

The fronto-parietal DMN subnetwork captures long-range interactions between anterior self-referential regions and posterior representational hubs, supporting cognitive control over internally focused processes (Andrews-Hanna et al., 2010; Spreng et al., 2010; Menon, 2011). This subnetwork was defined by electrode pairs connecting frontal sites (Fp1, Fp2, F3, F4, Fz) with posterior sites (Pz, P3, P4, POz). Fronto-parietal connectivity reflects integrative regulation within the DMN and is particularly sensitive to emotional load and attentional demands in anxiety-related conditions (Leech & Sharp, 2014; Seeley et al., 2007).

# Interhemispheric DMN Subnetwork

Interhemispheric connectivity reflects bilateral coordination across DMN-related cortical regions and is essential for balanced emotional and cognitive integration (Davidson & Hugdahl, 1995; Gazzaniga, 2000). This subnetwork was defined using homologous cross-hemispheric electrode pairs, including Fp1–Fp2, F3–F4, FC1–FC2, P3–P4, and O1–O2, as well as long-range frontal–parietal pairs such as Fp1–P4 and Fp1–P7. Reduced interhemispheric synchronization has been associated with impaired emotional regulation and heightened social threat sensitivity in anxiety disorders (Davidson, 2002; Paul et al., 2007).

# Subnetwork-Level Functional Connectivity Quantification

Functional connectivity within each DMN subnetwork was quantified by averaging Phase-Locking Value (PLV) measures across all electrode pairs belonging to that subnetwork. This approach reduces sensitivity to single-channel variability while preserving interpretability at the network level, consistent with established practices in EEG network neuroscience (Stam et al., 2007; Rubinov & Sporns, 2010).

Although EEG does not allow direct measurement of subcortical structures, converging intracranial and source-level evidence supports the reliability of scalp EEG signals as functional correlates of cortical DMN hubs, particularly the mPFC and PCC, justifying the use of electrode-based subnetworks as proxies for DMN organization (Das et al., 2022).

# Statistical Analysis

Statistical analyses were performed to evaluate the effects of social anxiety severity and experimental condition on Default Mode Network (DMN) functional connectivity. For each DMN subnetwork and EEG frequency band, Phase-Locking Value (PLV) measures were examined using two-way mixed-design analyses of variance (ANOVA), with Group (healthy control, mild, moderate, severe) specified as a between-subject factor and Condition (resting state, anticipation, performance, post-interruption) specified as a within-subject factor.

The assumption of sphericity for within-subject effects was assessed using Mauchly's test, and Greenhouse–Geisser corrections were applied when violations were identified (Greenhouse & Geisser, 1959). Significant main effects and interaction effects were further explored using Bonferroni-adjusted post hoc comparisons to control multiple testing. Statistical significance was evaluated using a two-tailed threshold of $\alpha = 0.05$, and effect sizes were reported using partial eta-squared ($\eta^2$), in accordance with recommended practice for ANOVA-based designs (Cohen, 1988). Bonferroni correction was applied across pairwise group comparisons within each DMN subnetwork and frequency band to control for multiple comparisons. All statistical procedures were applied consistently across DMN subnetworks and frequency bands.

Statistical analyses were conducted using MATLAB version R2024a.

# Data Availability

The datasets generated and analyzed during this study are available from the corresponding author upon reasonable request.

## Results

This section presents a comprehensive analysis of FC across DMN subnetworks in control and SAD participants categorized as mild, moderate, or severe. The evaluation includes both resting-state and anxiety-loaded conditions, assessing FC values derived from PLV across Delta, Theta, Alpha, and Beta bands. Electrode pairs were grouped into four subnetworks: frontal, fronto-parietal, posterior, and

interhemispheric connections. The following subsections describe the key electrodes and roles of each DMN subnetwork, followed by comparative results across subject groups and conditions.

# Statistical Inference of PLV Differences

Group- and condition-related differences in Phase-Locking Value (PLV) were examined using two-way mixed-design analyses of variance (ANOVA) for each Default Mode Network (DMN) subnetwork and EEG frequency band. Where appropriate, Greenhouse–Geisser corrections were applied, and Bonferroni-adjusted post hoc comparisons were used.

As summarized in Table 2, significant main effects of Group were observed predominantly in the beta band across multiple DMN subnetworks, with post hoc contrasts surviving Bonferroni correction for multiple comparisons. In the fronto-parietal subnetwork, a significant group effect was detected ($F(3,76) = 5.812$, $p = 0.001$). Post hoc comparisons indicated significantly lower PLV values in the severe group compared with the control ($p = 0.001$) and mild ($p = 0.014$) groups.

Similarly, the interhemispheric subnetwork exhibited a significant group effect in the beta band ($F(3,76) = 4.951$, $p = 0.003$), with reduced PLV values observed in the moderate ($p = 0.004$) and severe ($p = 0.012$) groups relative to controls. The frontal subnetwork also showed a significant group effect ($F(3,76) = 3.477$, $p = 0.020$), with post hoc analysis revealing lower PLV values in the severe group compared with controls ($p = 0.018$).

In addition to main effects, a significant Group × Condition interaction was observed in the fronto-parietal subnetwork in the beta band ($F(9,228) = 2.432$, $p = 0.012$, Greenhouse–Geisser corrected), indicating that PLV varied across experimental conditions as a function of SAD severity.

No significant main effects or interaction effects were observed in the alpha band for the fronto-parietal or interhemispheric subnetworks.

Table 2
Summary of Two-Way Mixed ANOVA Results for PLV Values Across DMN Subnetworks and Frequency Bands.

| DMN Subnetwork | EEG Band | Group Effect (F, p) | Condition Effect (F, p) | Group × Condition Interaction (F, p) | Significant Post-hoc Comparisons (Bonferroni-corrected) |
|---|---|---|---|---|---|
| Fronto-Parietal | Beta | F (3,76) = 5.812, p = 0.001 | F (3,228) = 1.823, p = 0.144 | F (9,228) = 2.432, p = 0.012 | Severee < Control ($p$ = 0.001), Severee < Mild ($p$ = 0.014) |
| Interhemispheric | Beta | F (3,76) = 4.951, p = 0.003 | F (3,228) = 2.001, p = 0.115 | F (9,228) = 1.678, p = 0.094 | Moderate < Control ($p$ = 0.004), Severe < Control ($p$ = 0.012) |
| Frontal | Beta | F (3,76) = 3.477, p = 0.020 | F (3,228) = 1.142, p = 0.330 | F (9,228) = 1.285, p = 0.249 | Severe < Control ($p$ = 0.018) |
| Fronto-Parietal | Alpha | F (3,76) = 2.031, p = 0.116 | F (3,228) = 2.679, p = 0.085 | F (9,228) = 1.216, p = 0.271 | — |
| Interhemispheric | Beta | F (3,76) = 2.547, p = 0.063 | F (3,228) = 2.134, p = 0.102 | F (9,228) = 1.389, p = 0.211 | — |

Resting-State DMN Connectivity Across Control, Mild, Moderate, and Severe SAD

Resting-state Default Mode Network (DMN) functional connectivity, quantified using Phase-Locking Value (PLV), is illustrated in Fig. 2a–d for control, mild, moderate, and severe social anxiety disorder (SAD) groups.

In healthy controls (Fig. 2a), resting-state DMN connectivity exhibited a coherent and well-integrated organization. High PLV values were observed within frontal and posterior DMN hubs, particularly in the alpha band, indicating strong intra-regional synchronization at rest. Fronto-parietal and interhemispheric connections showed moderate PLV values, consistent with balanced long-range and bilateral integration typically reported in normative DMN organization (Greicius et al., 2003; Fox et al., 2005; Buckner et al., 2008).

In mild SAD (Fig. 2b), frontal alpha-band connectivity remained largely comparable to controls, whereas reductions became evident in long-range connectivity. Fronto-parietal connections, particularly along lateral pathways, showed lower PLV values relative to controls, while midline fronto-parietal links remained relatively preserved. Interhemispheric connectivity also showed early attenuation, suggesting

initial weakening of bilateral coordination at rest (Davidson & Hugdahl, 1995; Zhao et al., 2007; Liao et al., 2010).

In moderate SAD (Fig. 2c), frontal alpha-band synchronization continued to be preserved; however, reductions in higher-frequency (beta-band) connectivity became more apparent. Fronto-parietal connectivity showed further attenuation across both lateral and midline pathways, while posterior DMN connectivity remained relatively stable. Interhemispheric PLV values continued to decline, indicating progressive disruption of long-range integration (Etkin et al., 2009; Blair et al., 2010; Sylvester et al., 2012).

In severe SAD (Fig. 2d), resting-state DMN connectivity demonstrated the most pronounced alterations. While alpha-band synchronization within frontal and posterior hubs persisted, beta-band connectivity was further reduced. Fronto-parietal connectivity showed marked attenuation, particularly in lateral connections, and interhemispheric synchronization reached its lowest levels across severity groups. Posterior connectivity exhibited modest reductions relative to controls (Stein et al., 2007; Spreng et al., 2009; Fonzo et al., 2017; Zhang et al., 2019).

Overall, resting-state DMN functional connectivity displayed a graded pattern of reduction with increasing SAD severity, characterized by early alterations in long-range fronto-parietal and interhemispheric connectivity and progressively broader reductions involving higher-frequency synchronization in more severe stages. Across all groups, midline frontal and posterior DMN hubs showed relative preservation of alpha-band connectivity.

DMN Functional Connectivity During the Anxiety-Loaded Condition Across Control, Mild, Moderate, and Severe SAD

Default Mode Network (DMN) functional connectivity during the anxiety-loaded condition, quantified using Phase-Locking Value (PLV), is illustrated in Fig. 3a–d for control, mild, moderate, and severe social anxiety disorder (SAD) groups.

In healthy controls (Fig. 3a), anxiety loading resulted in mild-to-moderate reductions in DMN connectivity relative to resting state. Frontal and posterior DMN hubs retained moderate alpha-band synchronization, whereas fronto-parietal and interhemispheric connections showed more pronounced attenuation. This pattern is consistent with previously reported stress-related modulation of long-range DMN connectivity, with relative preservation of core network hubs (Greicius et al., 2003; Seeley et al., 2007; Qin et al., 2014; Davidson, 2002).

In mild SAD (Fig. 3b), frontal and posterior alpha-band connectivity remained largely comparable to controls under anxiety. However, reductions were more evident in fronto-parietal and interhemispheric connections, particularly along midline pathways. Decreases in higher-frequency connectivity were also observed, suggesting early stress-related alterations in long-range integration (Zhao et al., 2007; Liao et al., 2010; Etkin & Wager, 2007; Davidson, 2002).

In moderate SAD (Fig. 3c), anxiety loading produced broader DMN connectivity reductions. Frontal connectivity showed attenuation across both alpha and beta bands, and fronto-parietal coherence was further reduced. Posterior connectivity remained relatively stable, while interhemispheric synchronization declined markedly, indicating increased sensitivity to anxiety-related modulation (Etkin et al., 2009; Menon, 2011; Leech & Sharp, 2014; Ochsner et al., 2004).

In severe SAD (Fig. 3d), anxiety-related DMN dysconnectivity was most pronounced. Frontal alpha-band synchronization persisted at reduced levels, whereas beta-band connectivity showed substantial attenuation. Fronto-parietal connectivity exhibited widespread reductions, particularly in lateral pathways, and interhemispheric synchronization reached its lowest levels across all groups. Posterior DMN connectivity remained comparatively preserved (Etkin & Wager, 2007; Sylvester et al., 2012; Davidson & Hugdahl, 1995).

Overall, anxiety loading revealed a severity-dependent amplification of DMN connectivity reductions. Across groups, frontal and posterior alpha-band synchronization demonstrated relative resilience, whereas fronto-parietal, interhemispheric, and higher-frequency connectivity showed progressively greater attenuation from mild to severe SAD.

state for the control, mild, moderate and severe SAD subjects.

## Discussion

The present findings reveal a coherent, severity-dependent pattern of Default Mode Network (DMN) functional connectivity alteration in social anxiety disorder (SAD), evident across both resting-state and anxiety-loaded conditions. Rather than reflecting a global disruption of DMN organization, the results indicate a progressive weakening of integrative and regulatory pathways, with relative preservation of core DMN hubs and increasing inefficiency in fronto-parietal and interhemispheric connectivity as symptom severity increases (Buckner et al., 2008; Raichle, 2015).

# Resting-State DMN Organization Across SAD Severity

During resting state, frontal DMN hubs associated with medial prefrontal cortex (mPFC) activity exhibited consistently preserved alpha-band synchronization across all severity groups. This pattern suggests that core self-referential and internally oriented processes remain largely intact even in moderate and severe SAD, in line with the established role of the mPFC within the DMN (Andrews-Hanna et al., 2010; Denny et al., 2012). In contrast, frequency-specific alterations emerged with increasing severity, particularly within the beta band. Reductions in beta-band connectivity were observed in mild and moderate SAD, followed by relative increases in severe SAD. Given the involvement of beta oscillations in sustained control and regulatory processes (Engel & Fries, 2010), this non-linear pattern may reflect a shift from reduced regulatory engagement to inefficient or maladaptive recruitment of control-related networks in more severe stages (Etkin et al., 2006; Roy et al., 2012).

Fronto-parietal connectivity emerged as the earliest and most vulnerable DMN pathway at rest. Progressive reductions in alpha-band synchronization along lateral fronto-parietal connections indicate compromised long-range integration between anterior self-referential regions and posterior representational hubs. Such alterations may limit the flexible modulation of internally directed cognition and disengagement from maladaptive self-focus (Spreng et al., 2010; Klumpp et al., 2012). Midline fronto-parietal connections demonstrated comparatively greater resilience but weakened gradually with increasing severity, suggesting partial compensation via central integrative circuits implicated in attentional and salience processing (Seeley et al., 2007; Menon, 2011). These observations are supported by inferential analyses showing significant severity effects, particularly in the beta band, underscoring the sensitivity of PLV-based connectivity metrics to SAD progression.

Posterior DMN connectivity showed the greatest stability at rest, with only modest alpha-band reductions across severity levels. This relative preservation is consistent with the role of posterior cingulate cortex and precuneus in maintaining autobiographical and contextual representations (Vogt & Laureys, 2005; Cavanna & Trimble, 2006). Nonetheless, subtle weakening in moderate and severe SAD may reflect increasing susceptibility to negatively biased internal mentation, potentially linked to altered interactions between posterior DMN regions and memory-related structures (Vincent et al., 2006; Addis et al., 2007).

Interhemispheric connectivity exhibited the most consistent and progressive decline at rest, particularly in the alpha band, indicating reduced bilateral integration with increasing SAD severity (Gazzaniga, 2000). Elevated beta-band interhemispheric synchronization observed in severe SAD may reflect inefficient or maladaptive coupling under heightened internal cognitive load (Chu et al., 2025). Together, these patterns suggest disrupted coordination between cognitive appraisal and affective processing systems (Davidson, 2002; Paul et al., 2007).

# Anxiety-Loaded Modulation of DMN Connectivity

Transitioning from rest to anxiety-loaded conditions amplified many of the connectivity alterations observed at baseline. While frontal alpha-band synchronization remained relatively preserved across groups—indicating sustained self-focused engagement under stress—beta-band frontal connectivity declined systematically with increasing SAD severity. This dissociation suggests preserved introspective processing alongside reduced efficiency of higher-frequency regulatory mechanisms during social threat (Engel & Fries, 2010; Etkin & Wager, 2007). In severe SAD, such imbalance may contribute to heightened vigilance and difficulty suppressing internally generated negative salience (Goldin et al., 2009).

Fronto-parietal pathways showed pronounced stress-related degradation, particularly along midline connections, reflecting reduced integration between self-referential processing and contextual regulation. Posterior DMN hubs remained comparatively resilient during anxiety, maintaining alpha-band synchronization and internal narrative continuity. However, this preservation may also facilitate persistent self-referential thought when frontal inhibitory control is compromised (Spreng et al., 2009; Fransson & Marrelec, 2008). Interhemispheric connectivity was the most stress-sensitive subnetwork,

exhibiting marked alpha-band desynchronization across all groups and maximal impairment in severe SAD, consistent with disrupted bilateral integration during emotional challenge (Davidson, 2002; Ochsner et al., 2004).

# Frequency-Specific Considerations and Network-Level Implications

Across conditions, delta- and theta-band connectivity remained largely preserved, indicating intact slow-wave network organization. In contrast, alpha-band alterations preferentially affected integrative pathways, while beta-band disruptions were most prominent under anxiety, highlighting impaired top-down modulation of emotionally salient information (Klimesch, 2012; Engel & Fries, 2010). This frequency-specific profile aligns with transdiagnostic findings across affective disorders, suggesting shared mechanisms of DMN dysregulation (Tozzi et al., 2021).

# Summary Remarks

Taken together, the present findings conceptualize SAD as a condition characterized by preserved core DMN architecture alongside progressively compromised regulatory and integrative connectivity, particularly under stress. Fronto-parietal and interhemispheric pathways emerge as primary loci of vulnerability, whereas posterior DMN hubs provide relative stability. This network-level imbalance offers a neurophysiological framework for key SAD features, including excessive self-focus, emotional reactivity, and rumination, and supports models that frame SAD as a disorder of maladaptive self-referential processing rather than global network breakdown (Northoff et al., 2006; Zhao et al., 2007).

# Conclusion

This study provides converging evidence that social anxiety disorder (SAD) is associated with systematic and severity-dependent alterations in Default Mode Network (DMN) functional connectivity. Using EEG-based phase synchronization quantified by Phase-Locking Value (PLV), we show that changes in DMN connectivity scale with symptom severity and are modulated by task context.

At rest, core DMN hubs located along frontal and posterior midline regions exhibit relatively preserved alpha-band synchronization across all severity groups. In contrast, fronto-parietal and interhemispheric connectivity shows progressive attenuation with increasing SAD severity, indicating selective vulnerability of long-range integrative pathways. Frequency-specific effects further differentiate severity levels, with beta-band connectivity demonstrating greater disruption in moderate and severe SAD.

Under anxiety-loaded conditions, these connectivity alterations are amplified. While alpha-band synchronization within frontal and posterior hubs remains relatively stable, fronto-parietal and interhemispheric connectivity exhibits marked reductions, particularly in higher-frequency bands. Interhemispheric pathways emerge as especially sensitive to anxiety-related modulation, distinguishing severe SAD from milder symptom levels.

Collectively, the findings support a network-level characterization of SAD marked by preserved core DMN architecture alongside progressively compromised regulatory and integrative connectivity, most evident under social stress. These results highlight the utility of EEG-based functional connectivity measures, such as PLV, for capturing context- and severity-dependent network alterations in SAD and provide a foundation for future work examining their potential relevance for longitudinal monitoring and intervention evaluation.

## Declarations

# Funding Declaration

No funding was received for this work.

# Author Contribution

N.K. conceived and designed the study, supervised the research, guided the analytical framework, and contributed to manuscript drafting and revision.C.H.L. performed EEG data preprocessing, functional connectivity analysis, statistical analyses, and figure and table preparation, and drafted the initial version of the manuscript.S.S. provided methodological guidance in signal processing and network analysis and contributed to the interpretation of the results and manuscript revision.T.T.K.T. contributed to manuscript editing and refinement and assisted in the interpretation of the results in the context of existing literature.All authors reviewed and approved the final version of the manuscript.

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# Data Availability

The datasets generated and analyzed during this study are available from the corresponding author upon reasonable request.

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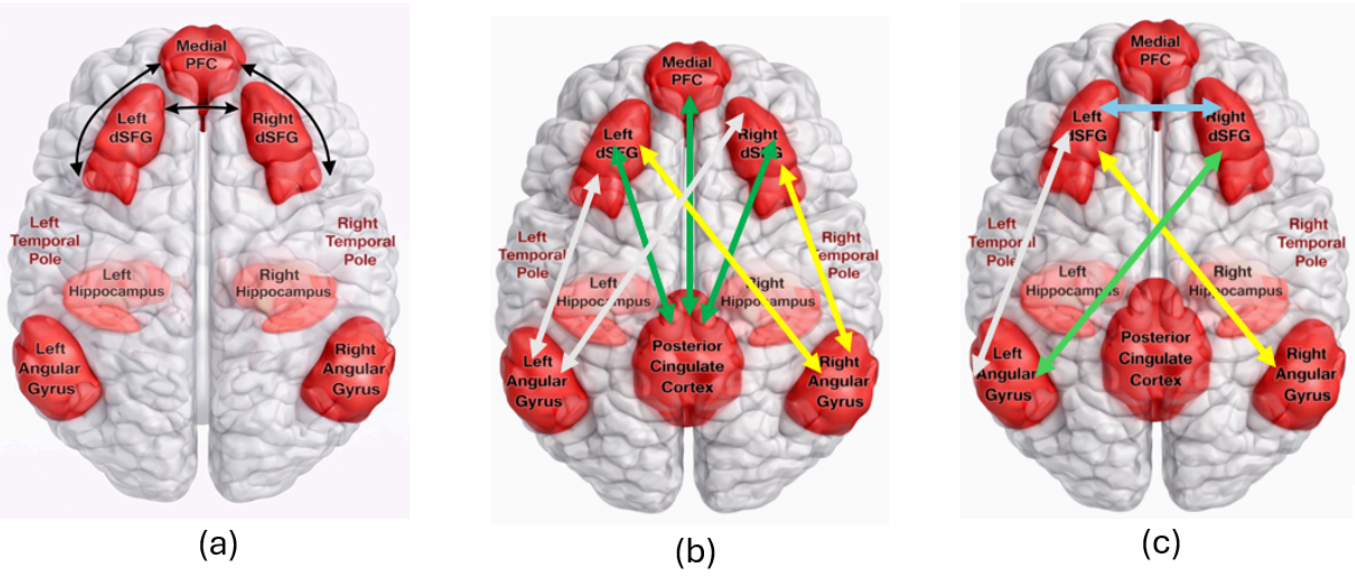
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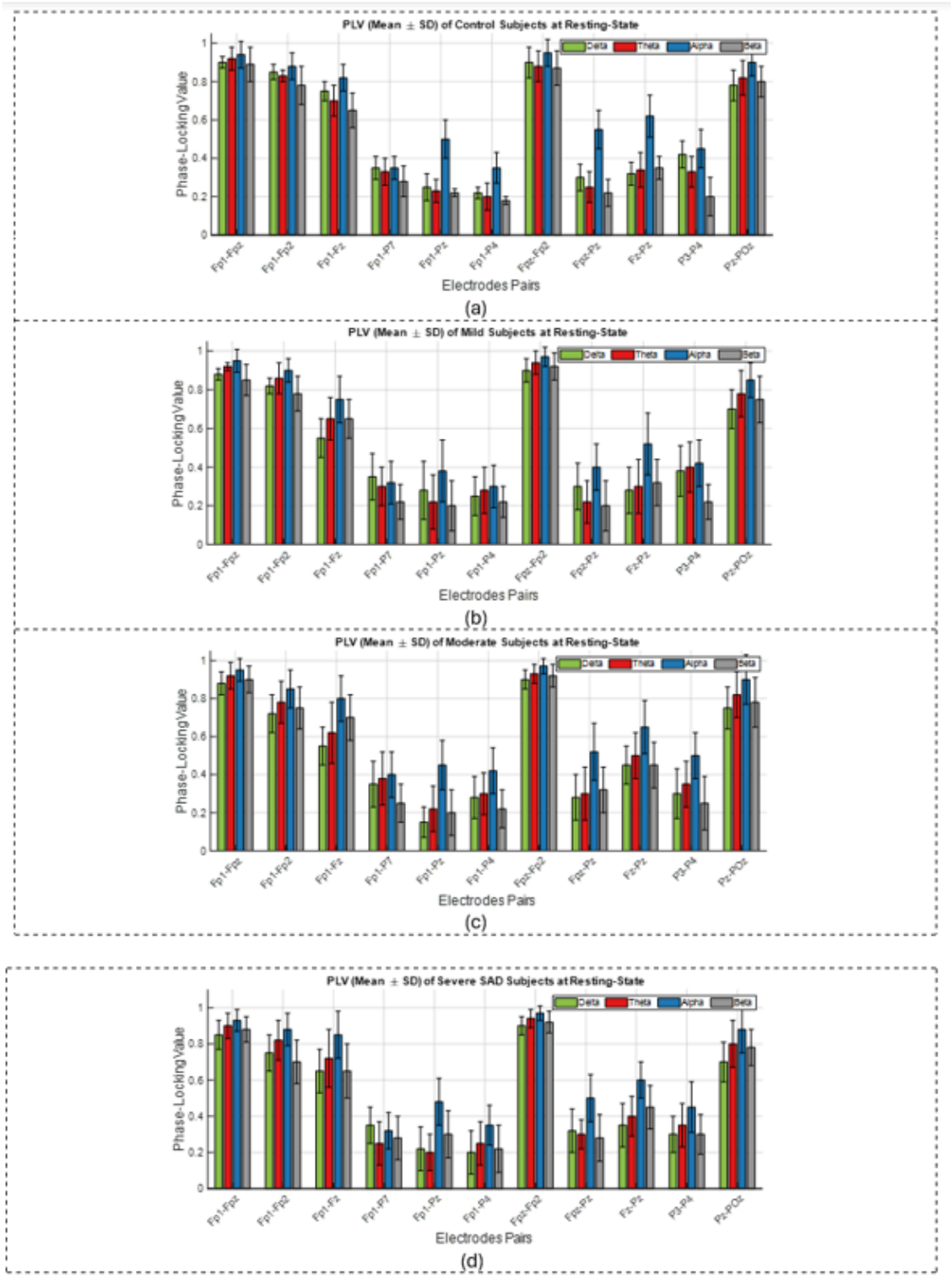
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# Figures



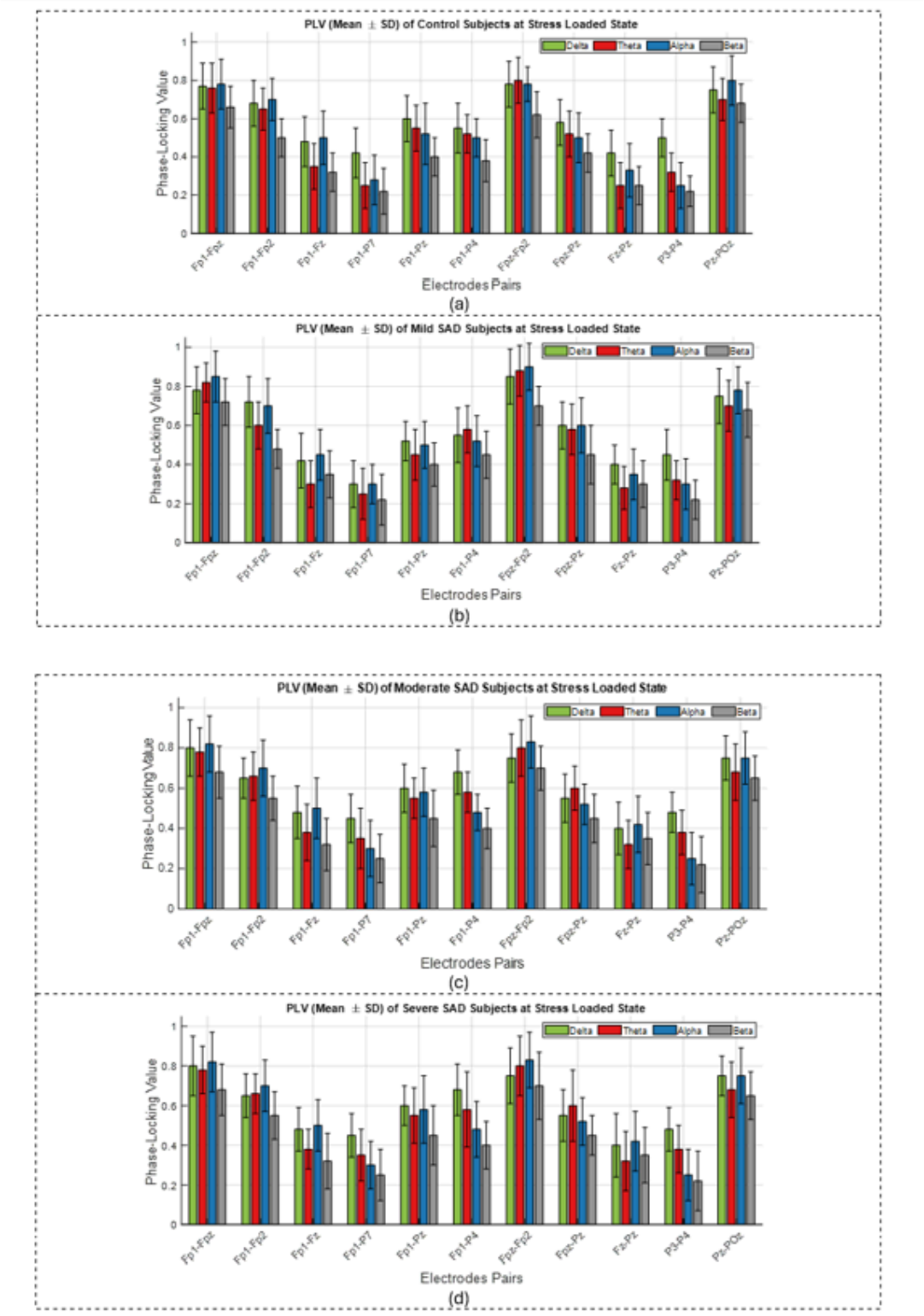
## Figure 1

The DMN subnetworks; (a) The DMN regions involved in the Frontal subnetwork, (b) The DMN regions involved in the fronto-parietal subnetwork, and (c) The DMN regions involved in the interhemispheric subnetwork.




## Figure 2

The mean values and standard deviation of FC with DMN electrodes during the resting-state for the control, mild, moderate and severe SAD subjects.




## Figure 3

The mean values and standard deviation of FC with DMN electrodes during the stress loaded

state for the control, mild, moderate and severe SAD subjects.