## **1 Abstract**

**Objective:** To quantify how strongly EEG and EOG signals couple across human sleep stages.

**Methods:** Overnight PSG from 29 adults were segmented into Wake, N1, N2, N3 and REM. Canonical correlation analysis (CCA) extracted the first two shared dimensions (ρ₁, ρ₂) in static stage-wise blocks and in 30 s sliding windows.

**Results:** Stage-wise ρ₁/ρ₂ increased monotonically from Wake to N3. Time-resolved analyses confirmed stronger and more stable coupling in deep NREM, while entropy of ρ distributions was highest in Wake and REM. Projection amplitudes did not differ by stage, indicating that correlation drives the effect.

**Conclusions:** EEG and EOG share a state-dependent low-dimensional subspace that is strongest in REM, moderate in N1, and minimal in N2/N3. These findings refine our understanding of brain–eye interactions and suggest stage-aware sensor configurations and artifact-removal strategies for sleep monitoring.

## **2 Introduction**

Sleep stages unfolds through non-rapid eye movement (NREM) sleep, comprising stages N1 to N3, and rapid-eye-movement (REM) sleep, classically scored with polysomnography that monitors electroencephalographic (EEG) activity and electrooculographic (EOG) signals related to eye movements. (Liu et al., 2021).

Growing evidence shows these two channels are not independent: ocular potentials contaminate frontal EEG, while EOG electrodes sample cortical rhythms, and exploiting this overlap improves automatic detection of REM and drowsiness (Safieddine et al., 2012; Xu et al., 2025). Such findings imply that brain and eye signals cohabit a low-dimensional “communication subspace” whose geometry may vary with different state. Yet the strength and dynamics of this coupling across all stages remain unquantified.

In this study, we seek to quantify that shared subspace across sleep stages. Using the public Apnea Positive Pressure Long-term Efficacy Study (APPLES) overnight PSG dataset (Mueller, n.d.), we applied canonical correlation analysis (CCA) to EEG and EOG signals to extract dominant joint components and assess their coupling strength during wake, N1, N2, N3, and REM stages, testing the hypothesis that coupling peaks in REM and diminishes in deeper NREM sleep.

## **3 Methods**

**3.1 Dataset**

Data for this study were drawn from 29 adult participants in the APPLES dataset. For each subject, we analyzed four bipolar EEG channels (C3–M2, C4–M1, O1–M2, O2–M1) alongside two EOG channels (LOC and ROC), with all recordings segmented according to the five sleep stages: Wake (W), N1, N2, N3 and REM (R).

**3.2 Preprocessing**

Preprocessing was carried out in MNE-Python by first loading the EEG and EOG channels from EDF recordings and then importing the corresponding sleep stage annotations. We converted annotation timestamps to seconds relative to each recording’s start time to ensure precise alignment with the signal data and excluded or corrected any segments with alignment issues. Finally, we extracted continuous EEG and EOG epochs for each labeled stage (W, N1, N2, N3, REM) to serve as inputs for our canonical correlation analyses.

**3.3 Dimensionality Reduction and Method Selection**

We first compared EEG and EOG subspaces with two subspace comparison methods, namely the Principal Component Analysis (PCA) and Independent Component Analysis (ICA) on EEG and EOG independently, followed by subspace angle computation. However, both returned angles near-zero or ill-defined subspace angles (e.g., 0 or 1e-14 radians), offering little physiological insight.

We therefore switched to Canonical Correlation Analysis (CCA), as it provides a more robust and interpretable measure of cross-modality dependence.

**3.4 Static CCA Analysis (Per-Stage Aggregation)**

After mean-cantering, a two-component CCA was applied. The first and second canonical correlations (ρ₁ and ρ₂) were obtained by correlating the paired canonical variates. To reduce serial dependence, the canonical time series were resampled at 1 Hz before computing stage-wise summary statistics.

**3.5 Time-Resolved CCA Analysis**

To characterize the temporal evolution of EEG–EOG coupling across sleep stages, the same two-component CCA was run in sliding windows of 30 s with a 15 s step (50 % overlap). These values were analysed in three ways:

* Stage-wise aggregation: mean and dispersion of ρ₁ and ρ₂ across all windows within each stage.
* Overnight trajectories:10-min bin averages to visualise stage-specific coupling trends across the recording.
* Distributional complexity: per subject and stage, Shannon entropy, skewness, and kurtosis of the ρ₁/ρ₂ distributions to assess variability and deviation from normality.

## **4. Results**

**4.1 Static Canonical Correlation Analysis (CCA)**

Stage-wise CCA on full-length segments showed a clear depth effect. The first canonical correlation (ρ₁) rose from 0.55 ± 0.14 in Wake to 0.86 ± 0.06 in N3; the second (ρ₂) increased from 0.32 ± 0.15 to 0.61 ± 0.09 (Fig. 1). One-way ANOVAs confirmed significance (ρ₁: F(4, 140)=24.4, p<0.001; ρ₂: F(4, 140)=16.7, p<0.001), indicating stronger EEG–EOG synchrony in deeper NREM sleep.

A graph of a diagram

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Figure 1. Static canonical correlation analysis (CCA) across sleep stages. Panel A shows the first canonical correlation (ρ₁), and Panel B shows the second (ρ₂), both increasing from Wake to N3.

**4.2 Time-Resolved CCA Analysis**

Sliding windows (30 s, 50 % overlap) reproduced the pattern with finer resolution. Aggregated ρ₁ ranged from **0.73 ± 0.13** (Wake) to **0.87 ± 0.07** (N3); ρ₂ from **0.45 ± 0.17** to **0.65 ± 0.11** (Fig. 2). Variability diminished with depth, and 10-min bins revealed stable plateaus in N2/N3 but fluctuating profiles in REM and Wake. Transition phases (e.g., N1) showed inconsistent coupling patterns.

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Figure 2. Time-resolved CCA reveals stronger and more stable EEG–EOG coupling in deeper sleep stages.

**4.3 Distributional Complexity of Coupling Dynamics**

Entropy was highest in Wake and REM and lowest in N3 (Fig. 3). Skewness remained near zero, but kurtosis was elevated in Wake/REM, suggesting occasional extreme coupling events.

A graph with blue and white lines

AI-generated content may be incorrect.Figure 3. Coupling entropy is highest in Wake and REM, and lowest in N3.

**4.4 Projection Statistics and Downsampled Components**

Mean and variance of the individual canonical projections showed no stage effect (all ANOVA p > 0.17; Fig. 4). Thus, stage differences stem from correlation structure rather than projection amplitude.

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Figure 4: Mean canonical projections remain stable across stages

## **Discussion**

Using static and time-resolved CCA, we confirm a state-dependent low-dimensional “communication subspace” between EEG and EOG. REM sleep shows the strongest coupling (ρ₁, ρ₂ highest), reflecting synchronized rapid eye movements and “sawtooth” EEG bursts from the PGO circuit (McCarley, 1994), which explains why REM can be robustly identified from EOG alone (DelRosso et al., 2018).

In contrast to REM, EEG–EOG coupling was significantly reduced in NREM sleep, especially in Stage N3. CCA revealed only a single weak canonical component, indicating minimal shared variance. EEG in N3 was dominated by large, synchronous delta waves, while EOG remained nearly flat—suggesting a functional decoupling between cortex and the oculomotor system during this restorative state. Methodologically, this decoupling means N3 EEG is relatively free of ocular artifacts, contributing to the clarity of delta activity.

Stage N2 showed slightly more coupling, though still limited. Sleep spindles and K-complexes typically lacked associated eye movements, with rare exceptions likely linked to brief arousals or blink-like responses. These transient events were not representative of ongoing coupling, reinforcing that EEG and EOG in N2 are largely modality-specific.

Stage N1, however, displayed modest but consistent EEG–EOG coupling. Canonical modes linked low-frequency EEG (e.g., waning alpha or theta) with slow eye excursions, consistent with drowsy transitions. This suggests a coordinated disengagement from wakefulness and supports the use of EEG–EOG features to improve N1 detection, a classification often challenged by subtle EEG changes (Xu et al., 2025).

Wakefulness yields context-dependent coupling: eyes-open epochs are artifact-dominated, while eyes-closed epochs show minimal correlation, emphasizing that the communication subspace is context-dependent.

Across stages, EEG–EOG coupling is strongest in REM (and partially in N1) and largely absent in N2/N3. From a systems neuroscience perspective, this selective sharing is analogous to cortical areas communicating only along certain subspace dimensions (Semedo et al., 2019).

**Limitation**

Two main limitations temper these conclusions. First, the APPLES cohort is enriched for obstructive-sleep-apnoea patients; frequent arousals and body movements could inflate coupling metrics, especially in lighter stages. Stratifying by arousal index or replicating the analysis in healthy sleepers will clarify generalisability. Second, we relied on linear, zero-lag CCA. Non-linear or time-shifted interactions—e.g. cortical bursts that precede eye movements—may have been missed. Future work could apply time-lagged CCA or mutual-information approaches and map the scalp distribution of shared components to identify which cortical regions communicate most with the eyes.

**Future directions**

The strong REM coupling provides indirect evidence for human PGO-like waves (Hobson, 2010) and invites future studies to test whether shared components are time-locked to individual REMs or sawtooth EEG bursts. The moderate N1 coupling also merits follow-up: multimodal recordings (e.g. EEG–EOG–fMRI) could determine whether slow eye rolls at sleep onset are neurally generated. Practically, these results support stage-aware monitoring: EOG alone may suffice for REM detection (DelRosso et al., 2018; Van Gorp et al., 2024), but accurate NREM staging still requires EEG. CCA-based artifact removal could be adapted by stage—aggressive in REM, minimal in N3—similar to sensor-fusion pipelines (Liu et al., 2021).

**Conclusion**

EEG and EOG signals share a low-dimensional communication subspace whose strength varies by sleep stage—strongest in REM, weakest in deep NREM, and moderate in N1. This stage-dependent coupling reframes EOG not as artifact but as a dynamic partner to EEG, enabling new perspectives on sleep physiology, sensor design, and artifact-aware analysis.

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