



Article

Graph Analysis of Age-Related Changes in Resting-State Functional Connectivity Measured with fNIRS

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Abstract

Resting-state functional connectivity (rsFC) provides insight into the intrinsic organization of brain networks and is increasingly recognized as a sensitive marker of age-related neural changes. Functional near-infrared spectroscopy (fNIRS) offers a portable and cost-effective approach to measuring rsFC, including in naturalistic settings. However, its sensitivity to age-related alterations in network topology remains poorly characterized. Here, we applied graph-based analysis to resting-state fNIRS data from 57 healthy participants, including 26 young adults (YA, 18–30 years) and 31 older adults (OA, 50–77 years). We observed that older adults exhibited a marked attenuation of low-frequency oscillation (LFO) power across all hemoglobin contrasts, corresponding to a 5–6-fold reduction in spectral power. In addition, network analysis revealed altered topological organization under matched sparsity conditions, characterized by reduced degree heterogeneity and increased segregation in older adults, with the strongest differences observed in the default mode (DMN), auditory, and frontoparietal control (FPC) networks. Network visualizations further indicated a shift toward more right-lateralized and posterior hub organization in older adults. Together, the coexistence of reduced oscillatory power and increased connectivity suggests that fNIRS-derived rsFC reflects combined neural and non-neural hemodynamic influences, including increased coherence arising from age-related vascular and systemic physiological processes. Overall, our findings demonstrate that fNIRS is sensitive to age-related changes in large-scale hemodynamic network organization. At the same time, sensitivity to non-neural hemodynamics highlights the need for cautious interpretation, but it may provide complementary, clinically relevant signatures of aging-related changes.

Keywords: resting state; fNIRS; functional connectivity; ageing



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1. Introduction

The worldwide aging population is expanding rapidly, with individuals aged 65 and above expected to represent a substantial proportion of the global demographic by 2050 [1]. This demographic transition is accompanied by increased prevalence of cognitive decline, with conditions ranging from normal cognitive aging to mild cognitive impairment (MCI) and dementia [2,3]. Understanding the neurovascular mechanisms underlying both normal and pathological aging has thus become a key scientific and socioeconomic priority. Aging is associated with progressive structural and functional changes that affect vascular efficiency, neural function, and large-scale brain network integration [4,5]. Conventional clinical assessments often detect impairment only after substantial neuronal and vascular deterioration, while structural brain changes typically manifest after earlier functional alterations have occurred [6].

Functional neuroimaging provides a suitable framework to investigate how aging affects brain function. In particular, functional connectivity characterizes the temporal correlation between spatially distributed brain regions, allowing the brain to be analyzed as an integrated network rather than isolated modules. When measured during rest, resting-state functional connectivity (rsFC) provides insight into the spontaneous low-frequency fluctuations that reflect the intrinsic functional architecture of the brain [7,8]. Resting-state approaches are particularly valuable for aging research because they do not rely on task performance, making them more suitable for older or cognitively impaired populations [9–12].

Previous rsFC studies using functional magnetic resonance imaging (fMRI) have shown that normal brain aging is accompanied by decreased connectivity within major brain networks and increased connectivity between networks, particularly within the frontoparietal, visual, and motor systems [13,14]. These changes are commonly interpreted as reflecting network dedifferentiation or compensatory reorganization in response to structural and metabolic decline. While fMRI has been instrumental in establishing these principles, its cost, immobility, and sensitivity to motion limit its applicability in large-scale, longitudinal, or ecologically valid aging studies.

Functional near-infrared spectroscopy (fNIRS) is an alternative approach to fMRI that employs near-infrared light to provide a portable, affordable, and non-invasive means of assessing brain function [15–18]. Its higher temporal resolution, tolerance to movement, and suitability for naturalistic environments make it particularly advantageous for studying older adults in their own environment. Importantly, however, fNIRS signals reflect a composite of cerebral oxygenation changes and extracerebral contributions arising from the scalp and skull, both of which are modulated by systemic physiological processes such as blood pressure, respiration, cardiac activity, and vascular compliance [19–21]. This is particularly critical when analyzing aging populations, as vascular remodeling, arterial stiffening, and altered cerebrovascular autoregulation are well documented [22–24]. Consequently, these systemic factors may independently influence fNIRS-derived rsFC, its correlation structure, and graph metrics, which should not be assumed to reflect purely neural mechanisms.

This mixed sensitivity presents both methodological challenges and unique opportunities for aging research, as many of the physiological processes that contribute to non-neural hemodynamic fluctuations are themselves meaningful biomarkers of aging. From this perspective, fNIRS-based connectivity measures may capture age-related alterations that reflect the integrated state of neural and vascular systems. While complete disentanglement of neural and non-neural contributions remains challenging, particularly in resting-state data, characterizing fNIRS-derived rsFC may provide further insights and relevant signatures of aging-related change.

Despite this potential, resting-state fNIRS connectivity remains comparatively under-explored, with few studies published on aging to date [25–27]. The relatively low number of channels, sensitivity to extracerebral hemodynamics, and differences in hemodynamic specificity mean that rsFC metrics derived from fNIRS may not directly mirror those from fMRI. In addition, the high temporal and low spatial resolutions of fNIRS signals introduce unique confounds, and at the same time, opportunities to characterize novel physiological aspects of aging. Therefore, establishing how fNIRS-based network measures behave in healthy young and older adults is a relevant step toward validating this modality for lifespan and clinical studies.

In this work, we evaluated the feasibility and sensitivity of fNIRS for detecting age-related alterations in resting-state functional connectivity. In particular, we characterized how standard network metrics manifest in fNIRS data and how they differ between healthy young and older adults. By explicitly considering the physiological and methodological properties of fNIRS, we sought to describe and interpret age-related differences in large-scale hemodynamic organization, reflecting both neural and non-neural components, as measured by this modality. Through this analysis, we aimed to establish a methodological foundation for future applications of fNIRS in the study of aging and age-related cognitive impairments.

2. Materials and Methods

2.1. Subjects and Experimental Protocol

A total of 57 participants with no self-reported history of neurological or psychiatric disorders were recruited for this study. Participants were divided into two groups: young adults (YA; $N = 26$; age range: 18–30 years; 3 female) and older adults (OA; $N = 31$; age range: 50–77 years; 21 female). For data collection, each participant laid on a hospital bed and was instructed to close their eyes and not focus on any specific task. Resting-state data were acquired over three runs of six minutes each. The study protocol was approved by the Research Ethics Committee of the University of Campinas, where the experiment was carried out, and all participants provided written informed consent prior to data collection.

2.2. fNIRS Acquisition and Processing

All optical measurements were acquired using a commercial continuous-wave fNIRS system (NIRScout, NIRx Medical Technologies, Berlin, Germany) with a sampling rate of 7.8 Hz. The probe configuration was designed to achieve coverage of the entire head and consisted of 16 light sources (each source contained two LEDs emitting at 760 nm and 850 nm) and 32 detectors. This setup resulted in a total of 64 source–detector pairs (channels) with an average source–detector separation of 3 cm (Figure 1). Probe placement followed the international 10–20 system and was subsequently verified using a homemade neuronavigational application [28] to confirm appropriate anatomical coverage.

The processing of the fNIRS data was performed using custom scripts based on the HOMER2 toolbox [29]. For each run, we excluded channels with a scalp-coupling index (SCI) below 0.7 from further analysis [30,31]. Importantly, channels removed due to poor quality were sporadic, and we did not observe any systematic bias across participants toward either hemisphere or age group. The remaining intensity time series were converted to optical density, and motion artifacts were corrected using a hybrid approach that combined spline interpolation followed by wavelet filtering, using 20 frames per segment for computing the moving standard deviation and 0.1 as the cutoff probability that the wavelet coefficients come from a normal distribution centered at zero [32]. Then, hemodynamic changes in the concentrations of the two main chromophores—oxy-hemoglobin (HbO) and

deoxy-hemoglobin (HbR)—were computed using the modified Beer–Lambert law with a differential pathlength factor of 6 for both wavelengths.

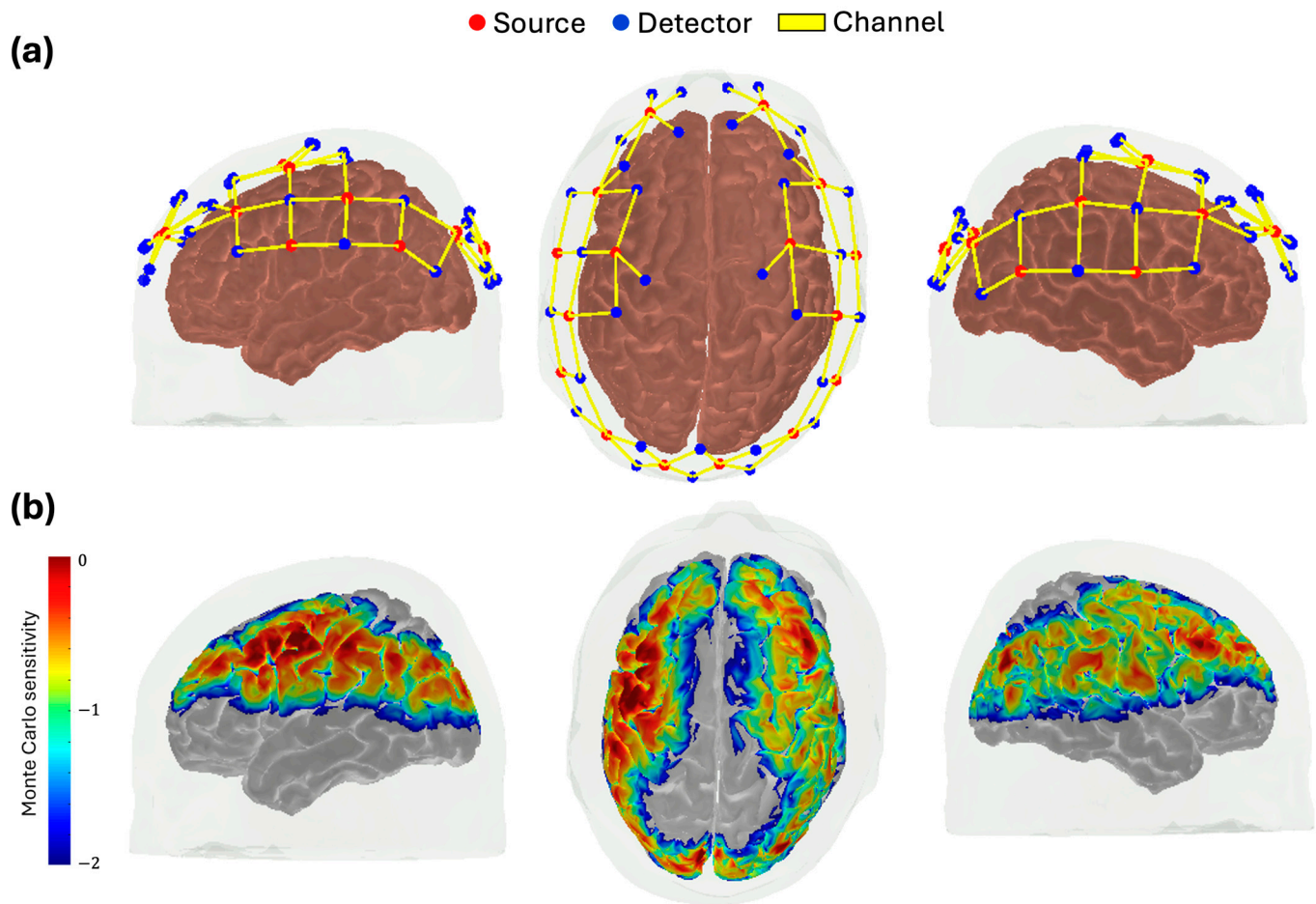


Figure 1. Topographical layout of the fNIRS probe configuration. (a) The source–detector configuration included 16 sources (red) and 32 detectors (blue), resulting in a total of 64 source–detector pairs (i.e., channels, yellow lines) with a source–detector separation of approximately 3 cm. (b) The sensitivity profile of the regions measured by the optical probe layout, which was obtained through Monte Carlo simulation of photon transport using the software tMCimg [33], available on the AtlasViewer package [34].

To minimize extra-cerebral contributions and isolate neural low-frequency oscillations (LFOs), each hemoglobin time series was band-pass filtered between 0.009 and 0.08 Hz using a third-order Butterworth filter [35–37]. Then, the first component of Principal Component Analysis (PCA) filter was removed to reduce systemic interference [38]. For analyses focusing on specific sub-bands of LFOs (Section 3.1), the data was band-pass filtered into two narrower frequency ranges: band V (0.010–0.027 Hz), associated with endothelial function and autoregulatory oscillations, and; band IV (0.027–0.073 Hz), associated with neurogenic vasomotion and local smooth muscle activity [39,40]. In all cases, the HbO and HbR time series were pre-whitened to minimize serially correlated errors [41], and the third and last chromophore (total hemoglobin, HbT) was calculated as the sum of HbO and HbR.

2.3. Functional Connectivity Analysis

Functional connectivity was quantified by computing pairwise Pearson correlation coefficients between all fNIRS channel time series for each hemoglobin contrast (HbO, HbR, and HbT). To reproduce the resting-state functional connectivity (rsFC) networks typically reported in the literature, we applied the Fisher z-transformation to the correlation matrix

measured for each run. Subject-level matrices were averaged across the runs to obtain individual functional connectivity maps, which were then averaged across subjects within each group. The resulting group matrices were subsequently standardized using Z-scores for visualization purposes. A seed-based approach was utilized to identify rsFC networks corresponding to cortical regions covered by our fNIRS probe: sensorimotor, auditory, visual, fronto-parietal control (FPC), and default mode network (DMN). For each of these networks, a representative seed channel was selected based on anatomical localization derived from the AtlasViewer v2.44.0 software [34]. Specifically, the sensorimotor network was localized to the left precentral gyrus, the auditory network to the left superior temporal gyrus, the visual network to the left cuneus gyrus, the FPC network to the left middle frontal gyrus, and the DMN to the left superior frontal gyrus.

For graph-based analysis, adjacency matrices were constructed from pairwise correlation matrices by retaining a fixed proportion of the strongest connections, such that edges below the corresponding correlation value were set to zero [42]. Graph properties were then evaluated as a function of network sparsity, defined as the proportion of retained edges relative to all possible connections. To assess the robustness of network properties across a physiologically plausible range of densities, we systematically varied sparsity from 0.05 to 0.50 in increments of 0.05, which allowed us to analyze how the pattern varies as a function of the threshold [36,43]. At each sparsity level, we quantified several graph properties characterizing network topology [44–46]. Network density was assessed using degree density, defined as the ratio of observed edges to the maximum possible edges in the network, while degree heterogeneity was quantified as the standard deviation of the nodal degree distribution. Functional segregation was evaluated using the average clustering coefficient, which measures the tendency of nodes to form locally interconnected groups. Functional integration was assessed through normalized global efficiency, calculated as the average inverse of the shortest path length across all node pairs and reflects the efficiency of information transfer across the network, while modularity was computed using the Newman–Girvan algorithm [47]. At the nodal level, hub regions were identified as nodes with the highest degree centrality [48]. All graph-theoretical analyses were performed using the Brain Connectivity Toolbox [42].

2.4. Statistical Analysis

Given the relatively low number of participants and deviations from normality observed in several measures, particularly within the older adult group, we opted to report results as median and interquartile range (IQR). When appropriate, between-group comparisons (younger vs. older adults) were conducted using the Mann–Whitney U test to compare the overall distributions between the two groups, and effect sizes were computed as the rank-biserial correlation (r_{rb}) to account for non-normality, with standard interpretation (~ 0.1 small, 0.3 medium, and 0.5 large). In all cases, we applied the Benjamini–Hochberg false discovery rate (FDR) separately for spectral and graph analyses to adjust p -values for multiple comparisons (p_{fdr}). Statistical significance was assessed based on corrected p -values and defined as $p_{fdr} < 0.05$ (two-tailed), and all statistical analyses were performed in Python (version 3.11) using the `scipy.stats` and `statsmodels` libraries.

Given the sex imbalance between the two groups, any quantitative analysis would inherently confound age group and sex (YA: 88% male; OA: 68% female). For this reason, all statistical comparisons were repeated using only the male participants to control for sex and provide a check for age-related effects. In addition, to assess the magnitude of potential sex variations within the aging brain, we compared male and female participants within the OA cohort. This dual-validation approach was chosen because of the low number of female participants in the young group, which would not provide enough

power to estimate age/sex interactions and could introduce instability and potential bias in covariance-based models.

3. Results

3.1. Global Attenuation of Low-Frequency Oscillatory Power in Older Adults

Across all frequency bands and chromophores, we observed a pronounced attenuation of low-frequency oscillation (LFO) power in older adults compared to young adults (Figure 2). This reduction was large in magnitude and consistent across the whole resting-state spectrum as well as Band V and Band IV. For example, HbO power was reduced by approximately 80% in the resting-state spectrum and 70% in Band IV in OA compared to YA. A comparable pattern was observed for HbR, with reductions of 85% and 77% in the resting-state and Band IV ranges, respectively. All bands and chromophores showed reductions of at least 68% in the older cohort, and across chromophores, the biggest absolute differences were observed for HbO and HbT.

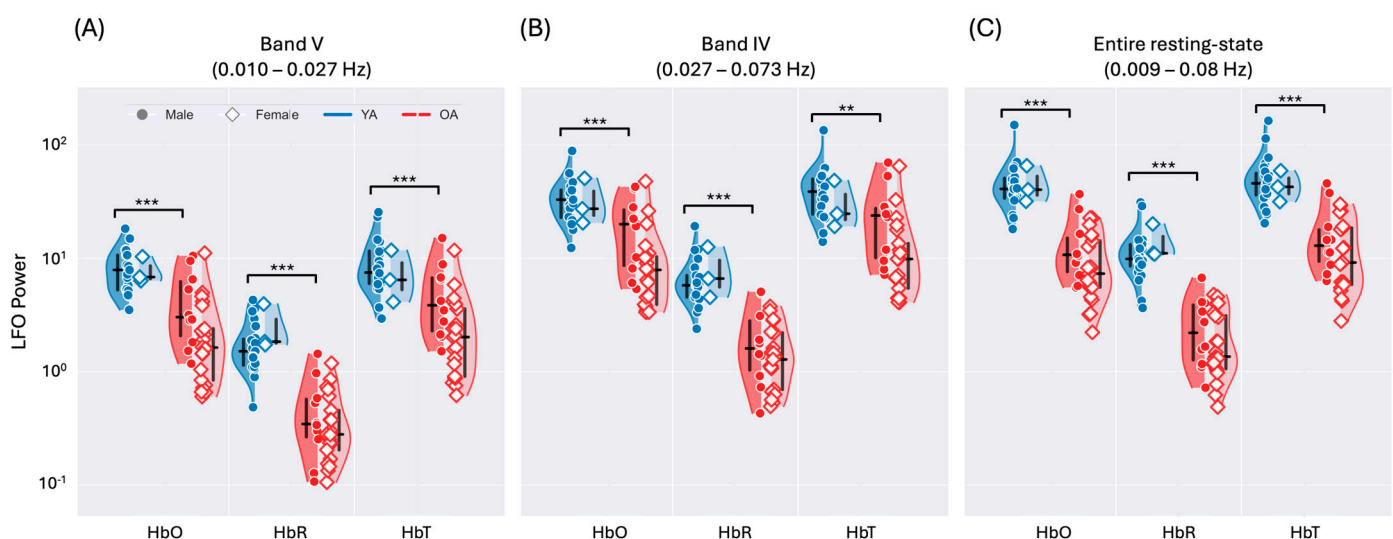


Figure 2. Low-frequency oscillation (LFO) power by group and contrast for young adults (YA, blue) and older adults (OA, red) across three frequency bands: (A) Band V (0.010–0.027 Hz), (B) Band IV (0.027–0.073 Hz), and (C) full resting-state LFO range (0.009–0.08 Hz). Results are shown separately for oxy-hemoglobin (HbO), deoxy-hemoglobin (HbR), and total hemoglobin (HbT). Individual data points and distribution estimations are marked by sex (male: full circles, darker shade, female: empty diamonds, lighter shade). The black vertical bars represent the inter-quartile range (IQR), while the black horizontal tick represents the average of the distribution. (**: $p < 0.05$, $0.3 < r_{rb} \leq 0.5$; ***: $p < 0.05$, $r_{rb} > 0.5$).

Results separated by age group and sex are presented in Table 1. To exclude potential confounding by sex imbalance, we performed sensitive analysis restricted to male participants. This analysis confirmed a robust age effect: all three LFO bands showed significant, large-magnitude decreases in older males compared with younger males. The strongest effects were observed for the resting-state spectrum (HbO: $p_{\text{fdr}} = 0.0002$, $r_{\text{rb}} = 0.88$; HbR: $p_{\text{fdr}} = 0.0002$, $r_{\text{rb}} = 0.96$; HbT: $p_{\text{fdr}} = 0.0003$, $r_{\text{rb}} = 0.84$), followed by Band V (HbO: $p_{\text{fdr}} = 0.01$, $r_{\text{rb}} = 0.59$; HbR: $p_{\text{fdr}} = 0.0002$, $r_{\text{rb}} = 0.88$; HbT: $p_{\text{fdr}} = 0.03$, $r_{\text{rb}} = 0.50$). The smallest effect sizes were observed in Band IV (HbO: $p_{\text{fdr}} = 0.02$, $r_{\text{rb}} = 0.53$; HbR: $p_{\text{fdr}} = 0.0003$, $r_{\text{rb}} = 0.85$; HbT: $p_{\text{fdr}} = 0.05$, $r_{\text{rb}} = 0.44$). Across bands, the largest age-related differences were consistently observed for HbR, whereas HbT showed the smallest effect sizes.

Table 1. Low-frequency oscillation (LFO) power across different spectral ranges in young (YA) and older adults (OA), including pooled estimates across all participants and stratified by sex, and shown separately for each hemoglobin contrast. Values represent the median across participants, with the 25th and 75th percentiles in parentheses. (F: Female, M: Male, all: pooled across sexes).

		Resting State (0.009–0.08 Hz)	Band V (0.010–0.027 Hz)	Band IV (0.027–0.073 Hz)
Oxy-hemoglobin concentration (HbO)				
OA	all	8.3 (5.6, 15.1)	1.9 (1.3, 4.0)	9.1 (5.0, 19.7)
	M	10.8 (7.6, 15.1)	3.0 (2.1, 6.2)	20.1 (8.7, 26.8)
	F	7.3 (5.6, 14.3)	1.6 (0.8, 2.4)	7.9 (3.9, 10.3)
YA	all	40.7 (33.6, 51.5)	7.6 (5.4, 10.6)	31.8 (22.9, 40.1)
	M	41.1 (34.0, 51.0)	7.9 (5.3, 10.7)	33.0 (23.0, 40.0)
	F	40.4 (36.1, 53.0)	6.9 (6.6, 8.6)	27.4 (24.0, 39.2)
Deoxy-hemoglobin concentration (HbR)				
OA	all	1.6 (1.1, 3.3)	0.30 (0.22, 0.56)	1.4 (0.80, 2.2)
	M	2.2 (1.3, 3.9)	0.35 (0.27, 0.57)	1.6 (1.0, 2.8)
	F	1.4 (1.1, 3.1)	0.28 (0.20, 0.46)	1.3 (0.70, 2.2)
YA	all	10.7 (8.9, 13.3)	1.6 (1.2, 2.0)	5.9 (4.6, 7.1)
	M	9.9 (8.6, 13.2)	1.5 (1.1, 1.9)	5.8 (4.6, 7.1)
	F	11.1 (11.0, 15.6)	1.8 (1.8, 2.9)	6.6 (5.6, 9.6)
Total-hemoglobin concentration (HbT)				
OA	all	11.3 (6.2, 18.8)	2.4 (1.4, 4.2)	10.0 (6.7, 23.1)
	M	12.9 (9.4, 17.9)	3.9 (2.3, 6.7)	23.9 (10.1, 27.6)
	F	9.2 (5.9, 18.6)	2.0 (0.9, 3.6)	9.9 (5.5, 13.5)
YA	all	45.4 (35.9, 57.3)	7.5 (6.0, 11.6)	37.6 (24.4, 49.3)
	M	46.0 (36.4, 56.4)	7.5 (6.0, 11.6)	38.8 (24.6, 50.2)
	F	42.8 (37.2, 51.1)	6.5 (5.3, 9.1)	24.8 (22.0, 36.7)

Numbers in bold represent the pooled estimates across all participants within each age group.

Within-group comparisons of sex differences in the OA cohort were not statistically significant in any LFO band, particularly for HbR (Band V: $p_{\text{fdr}} = 0.49$, $r_{\text{tb}} = 0.16$; Band IV: $p_{\text{fdr}} = 0.41$, $r_{\text{tb}} = 0.21$; resting state: $p_{\text{fdr}} = 0.41$, $r_{\text{tb}} = 0.22$). Sex differences in HbO (Band V: $p_{\text{fdr}} = 0.07$, $r_{\text{tb}} = 0.49$; Band IV: $p_{\text{fdr}} = 0.07$, $r_{\text{tb}} = 0.51$; resting state: $p_{\text{fdr}} = 0.28$, $r_{\text{tb}} = 0.32$) and HbT (Band V: $p_{\text{fdr}} = 0.07$, $r_{\text{tb}} = 0.52$; Band IV: $p_{\text{fdr}} = 0.07$, $r_{\text{tb}} = 0.54$; resting state: $p_{\text{fdr}} = 0.27$, $r_{\text{tb}} = 0.31$) were modest in magnitude and did not reach statistical significance, although trends were more apparent in Bands V and IV and may warrant further investigation in larger samples.

3.2. Age-Related Global Network Properties in fNIRS-Derived Signals

Across both age groups and all hemoglobin contrasts, the overall spatial organization of rsFC patterns showed a consistent and reproducible large-scale structure. We identified canonical resting-state networks (sensorimotor, auditory, visual, frontoparietal control, and default mode) in both YA and OA groups for HbO, HbR, and HbT signals (Figure 3), reinforcing the capability of fNIRS to provide insights into rsFC across age.

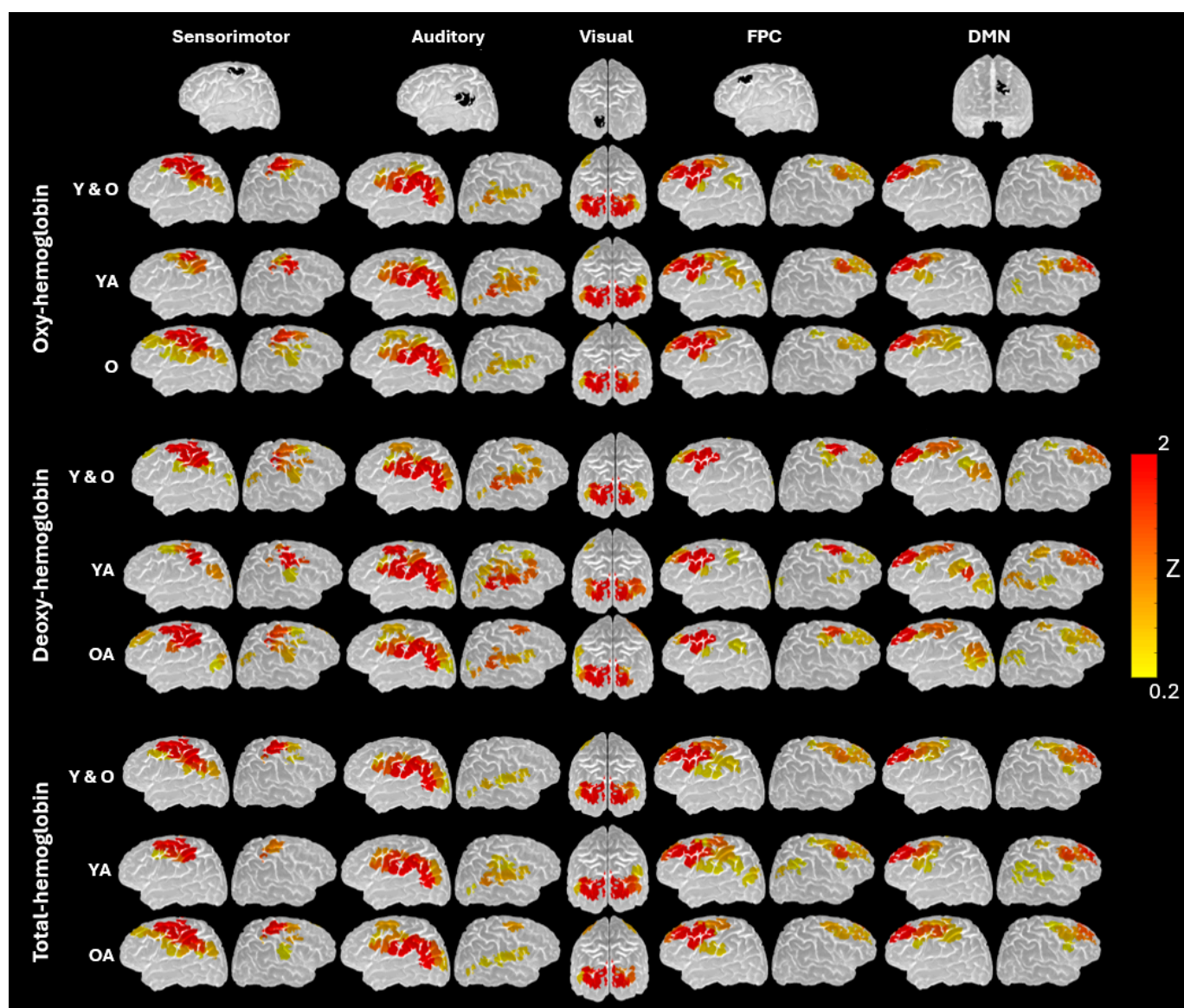


Figure 3. Resting-state functional connectivity (rsFC) networks obtained with fNIRS for both young adults (YA) and older adults (OA), as well as the combined cohort (Y & O), across all hemoglobin contrasts. The black region in the top row shows the cortical projection of the fNIRS channel used as a reference (seed). (FPC: fronto-parietal control network; DMN: default-mode network).

To quantify age-related differences in network organization, we examined the dependence of global graph properties on network sparsity (Figure 4). As expected, decreasing the sparsity led to monotonic reductions in overall connectivity, degree heterogeneity, clustering coefficient, and global efficiency in both groups, reflecting progressive edge removal as the networks became sparser. While these trends were qualitatively similar across age groups and hemoglobin contrasts, we observed systematic differences in the magnitude and rate of change of several metrics.

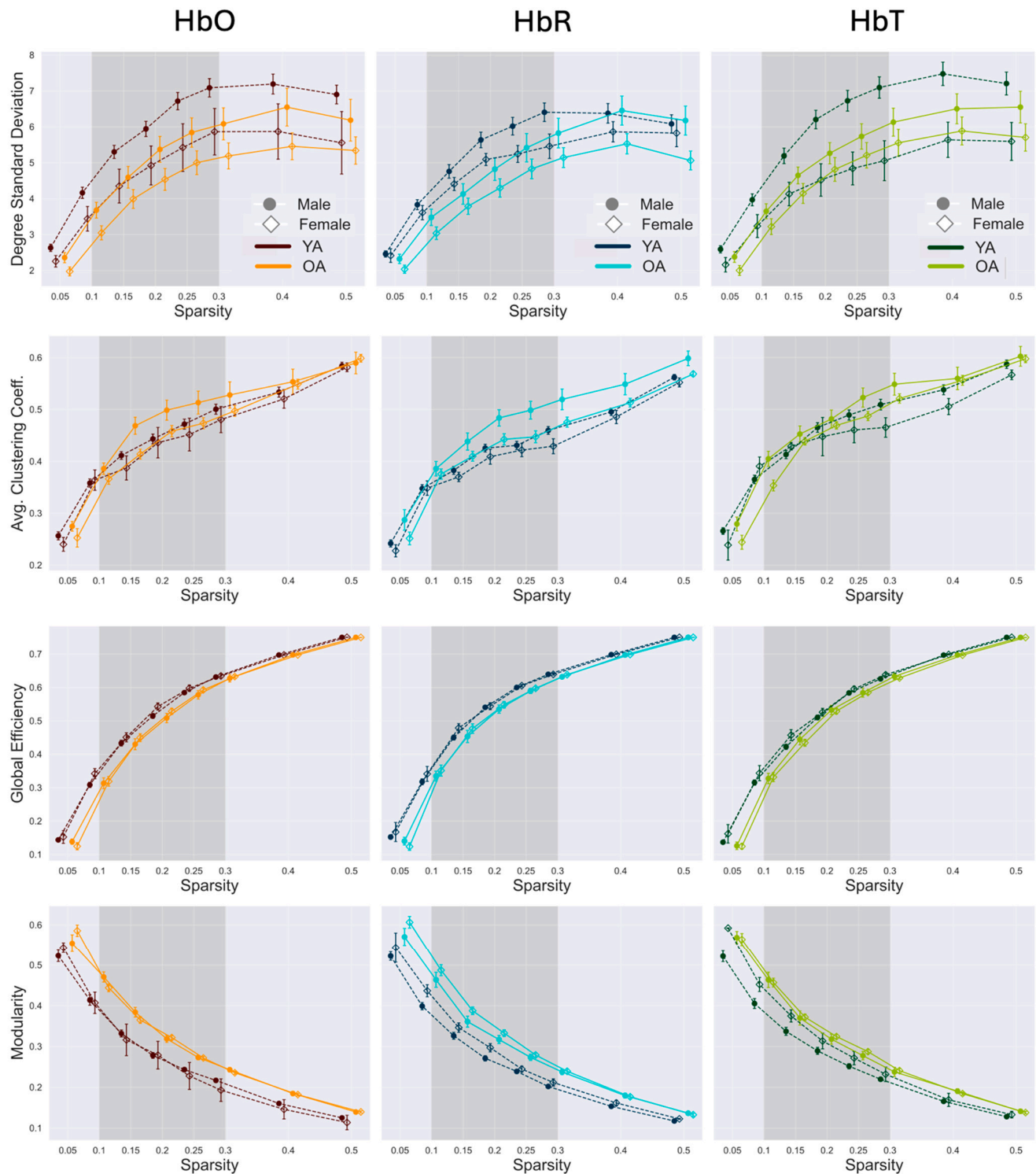


Figure 4. Global network topological properties as a function of network sparsity. Macroscopic graph properties were derived for each age group and sparsity value, stratified by sex. To improve visual clarity, data points for different groups are shown with small horizontal offsets at each sparsity level. Error bars represent the standard error across subjects, and the shaded area indicates the sparsity range of biological interest.

Across the full sparsity range, YA consistently exhibited higher degree standard deviation than OA, indicating greater network heterogeneity in younger participants. In contrast, OA tended to show higher clustering and modestly higher modularity relative to

YA, suggesting a more locally segregated network organization with age. These patterns were consistent across hemoglobin contrasts but were most pronounced for HbO and HbR.

We quantified these effects using subject-level averages on the sparsity range of biological interest (0.1–0.25), commonly adopted in the literature to balance network sparsity against fragmentation (Table 2). Considering only male participants, OA exhibited significantly higher modularity than YA for both HbO ($p_{\text{fdr}} = 0.03$, $r_{\text{rb}} = 0.64$) and HbR ($p_{\text{fdr}} = 0.04$, $r_{\text{rb}} = 0.60$). Clustering coefficient was also higher in OA relative to YA, reaching significance for HbR ($p_{\text{fdr}} = 0.03$, $r_{\text{rb}} = 0.68$). These effects were directionally consistent across sparsity values within the selected range.

Table 2. Global topological properties averaged across the 0.1–0.25 sparsity range in young (YA) and older adults (OA), including pooled estimates across all participants and stratified by sex, and shown separately for each hemoglobin contrast. Values represent the median across participants, with the 25th and 75th percentiles in parentheses. (F: Female, M: Male, all: pooled across sexes).

		Degree	Standard Deviation	Average Clust. Coeff.	Global Efficiency	Modularity
HbO						
OA	all	4.3	(3.5, 5.4)	0.44 (0.41, 0.47)	0.48 (0.43, 0.51)	0.35 (0.33, 0.38)
	M	5.0	(4.1, 5.6)	0.46 (0.43, 0.50)	0.46 (0.44, 0.50)	0.36 (0.34, 0.37)
	F	4.2	(3.3, 5.3)	0.43 (0.41, 0.46)	0.48 (0.43, 0.51)	0.34 (0.33, 0.38)
YA	all	5.5	(4.6, 6.0)	0.42 (0.40, 0.45)	0.48 (0.43, 0.49)	0.31 (0.29, 0.33)
	M	5.5	(4.6, 6.1)	0.42 (0.40, 0.46)	0.47 (0.43, 0.49)	0.32 (0.29, 0.33)
	F	4.5	(4.5, 5.2)	0.42 (0.39, 0.44)	0.49 (0.48, 0.50)	0.30 (0.30, 0.35)
HbR						
OA	all	4.2	(3.5, 5.3)	0.43 (0.41, 0.46)	0.49 (0.46, 0.51)	0.36 (0.33, 0.38)
	M	4.4	(4.2, 5.6)	0.46 (0.43, 0.47)	0.49 (0.45, 0.49)	0.35 (0.33, 0.37)
	F	4.0	(3.1, 4.7)	0.42 (0.41, 0.45)	0.50 (0.47, 0.51)	0.36 (0.33, 0.38)
YA	all	5.1	(4.6, 5.6)	0.40 (0.37, 0.42)	0.49 (0.47, 0.50)	0.31 (0.29, 0.33)
	M	5.1	(4.6, 5.7)	0.41 (0.37, 0.42)	0.49 (0.47, 0.50)	0.31 (0.29, 0.32)
	F	4.7	(4.6, 4.9)	0.38 (0.38, 0.39)	0.50 (0.49, 0.51)	0.33 (0.31, 0.33)
HbT						
OA	all	4.7	(3.5, 5.5)	0.45 (0.43, 0.47)	0.48 (0.44, 0.49)	0.36 (0.32, 0.37)
	M	4.9	(4.4, 5.2)	0.46 (0.44, 0.51)	0.48 (0.44, 0.49)	0.35 (0.33, 0.38)
	F	4.5	(3.5, 5.6)	0.44 (0.43, 0.46)	0.48 (0.44, 0.49)	0.36 (0.32, 0.37)
YA	all	5.5	(4.2, 5.9)	0.44 (0.41, 0.46)	0.47 (0.45, 0.49)	0.33 (0.30, 0.35)
	M	5.7	(4.3, 6.0)	0.44 (0.42, 0.46)	0.46 (0.45, 0.49)	0.32 (0.30, 0.35)
	F	4.3	(4.2, 4.8)	0.44 (0.42, 0.45)	0.48 (0.48, 0.50)	0.35 (0.34, 0.37)

Numbers in bold represent the pooled estimates across all participants within each age group.

To further assess potential sex-related influences, we compared males and females within the OA group only. Males tended to show slightly higher degree heterogeneity and clustering than females; however, none of these differences reached statistical significance after correction for multiple comparisons ($p_{\text{fdr}} > 0.58$ in all cases).

3.3. Network-Level Topology Differences Within rsFC Networks

At the mesoscopic scale, specific rsFC networks followed the same pattern observed in global graph organization metrics, particularly the DMN, auditory network, and FPC network. Figure 5 presents the main differences for HbR, while the same plot for HbO and HbT are shown, respectively, in Figures S1 and S2 in the Supplementary Materials. Consistent with the global analyses, OA generally exhibited reduced network heterogeneity alongside increased segregation relative to YA, although the magnitude and direction of these effects depended on network, metric, and hemoglobin contrast.

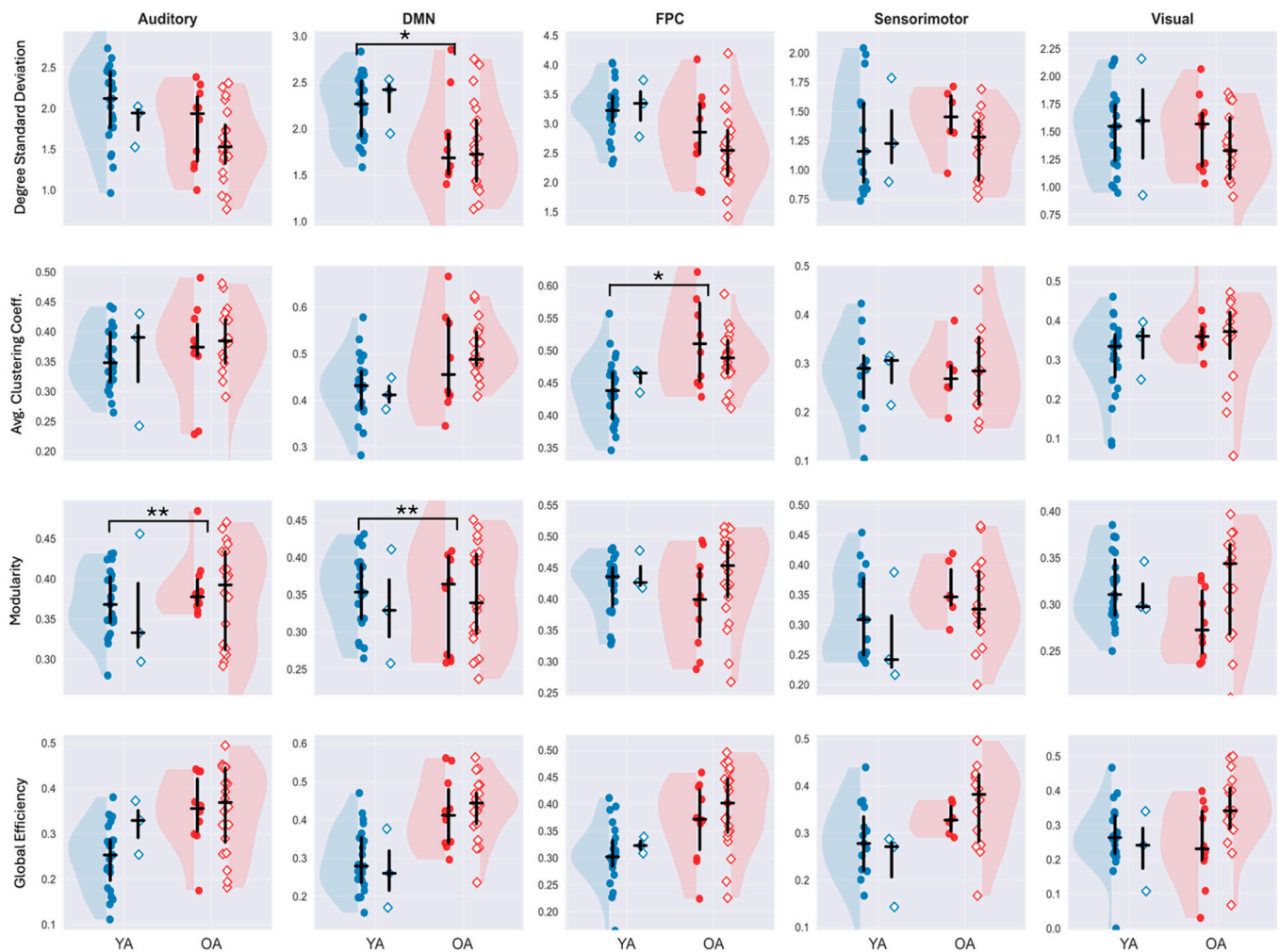


Figure 5. Network-specific topology obtained for deoxy-hemoglobin (HbR) across canonical resting-state networks. All global metrics comparing young adults (YA, blue) and older adults (OA, red), stratified by sex, across five major rsFC networks: sensorimotor, auditory, visual, frontoparietal control (FPC), and default mode network (DMN). (*: $p < 0.1$, **: $p < 0.05$). Individual points represent subject-level values, and shaded regions indicate the group-level density distributions. (filled circles: males; empty circles: females).

Focusing on male participants only, the DMN emerged as the network most sensitive to age across all hemoglobin contrasts. In HbO, OA showed significantly higher average clustering compared to YA (median [IQR]: OA = 0.54 [0.45–0.57], YA = 0.42 [0.39–0.46], $p_{\text{fdr}} = 0.048$, $r_{\text{rb}} = 0.64$), alongside higher modularity (OA = 0.44 [0.40–0.46], YA = 0.33 [0.25–0.39], $p_{\text{fdr}} = 0.056$, $r_{\text{rb}} = 0.62$). A similar pattern was observed for HbR and HbT, with OA exhibiting higher DMN modularity in both contrasts (HbR: OA = 0.41 [0.34–0.48], YA = 0.28 [0.24–0.35], $p_{\text{fdr}} = 0.048$, $r_{\text{rb}} = 0.66$); HbT: OA = 0.39 [0.36–0.49], YA = 0.33 [0.28–0.36], $p_{\text{fdr}} = 0.048$, $r_{\text{rb}} = 0.64$). In contrast, degree heterogeneity within the DMN was lower in OA than YA, reaching trend-level significance in HbR (OA = 1.7 [1.5–1.9], YA = 2.3 [1.9–2.5], $p_{\text{fdr}} = 0.097$, $r_{\text{rb}} = 0.53$) and HbT (OA = 1.9 [1.7–2.0], YA = 2.2 [2.0–2.6], $p_{\text{fdr}} = 0.056$, $r_{\text{rb}} = 0.61$).

Beyond the DMN, the auditory network also demonstrated robust age-related increases in segregation. Modularity was significantly higher in OA than YA for both HbO (OA = 0.38 [0.37–0.44], YA = 0.30 [0.25–0.35], $p_{\text{fdr}} = 0.048$, $r_{\text{rb}} = 0.64$) and HbR (OA = 0.36 [0.31–0.42], YA = 0.25 [0.20–0.29], $p_{\text{fdr}} = 0.048$, $r_{\text{rb}} = 0.70$), indicating a more segregated auditory rsFC organization with aging.

In the FPC network, OA showed higher average clustering than YA in HbR (OA = 0.51 [0.45–0.57], YA = 0.44 [0.40–0.47]), although this effect reached trend-level significance only ($p_{\text{fdr}} = 0.077$, $r_{\text{tb}} = 0.57$). Age-related effects were also observed in the visual network for HbT, where global efficiency was lower in OA compared to YA (OA = 0.26 [0.25–0.28], YA = 0.30 [0.27–0.32], $p_{\text{fdr}} = 0.077$, $r_{\text{tb}} = 0.57$), consistent with reduced integration at the network level.

By contrast, the sensorimotor network was comparatively insensitive to age, showing no significant differences across metrics or hemoglobin contrasts. Finally, within-group comparisons between males and females in the OA cohort did not reveal significant sex effects in any network-level metric (all $p_{\text{fdr}} > 0.89$), supporting the interpretation that the observed network-specific differences are primarily driven by age rather than sex.

3.4. Age-Dependent Differences in Spatial Organization

To illustrate the spatial organization of the fNIRS-derived graph and identify highly connected nodes, we visualized group-level networks at a fixed sparsity of 0.2 (Figure 6). This sparsity lies within the biologically plausible range commonly used in rsFC studies and ensures equivalent edge density across age groups and hemoglobin contrasts.

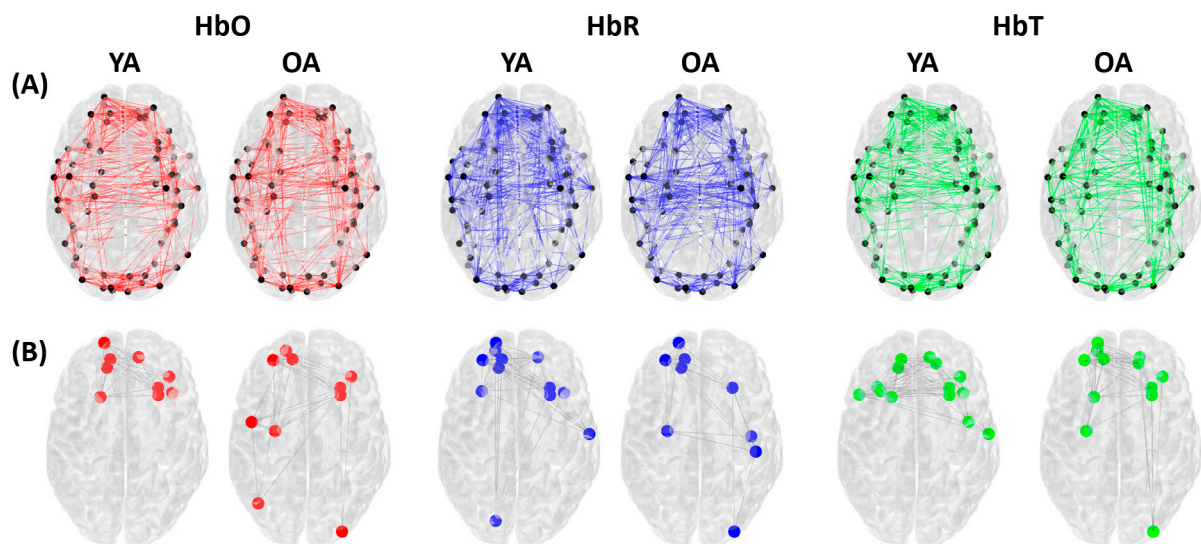


Figure 6. Group-level visualization of rsFC networks and hub distributions at fixed sparsity (0.2). (A) Network representations for young adults (YA) and older adults (OA) across hemoglobin contrasts: HbO (red), HbR (blue), and HbT (green). (B) Spatial distribution of hub nodes for each contrast and age group.

Across all contrasts, both YA and OA exhibited coherent large-scale connectivity patterns, with dense interconnections spanning frontal, parietal, and occipital regions. Despite matched sparsity, qualitative differences in network organization were apparent between groups. In particular, older adults showed more spatially widespread and visually homogeneous connectivity patterns.

Hub regions (defined as nodes with degrees at least one standard deviation above the network mean) were consistently observed in the frontal and occipital cortices across age groups and hemoglobin contrasts. The overall number of hubs was comparable across groups, but YA tended to show a more left-lateralized hub organization, while OA showed a shift toward greater right hemisphere involvement, particularly in posterior and occipital regions.

3.5. Influence of Global Variance Suppression to Age-Related Network Differences

To evaluate whether global systemic factors explained the enhanced connectivity observed in older adults, we examined the proportion of variance removed by the first principal component during preprocessing (Figure 7A). The variance accounted for by this component did not differ significantly between groups (HbO: YA = 29.8 [24.7–33.9], OA = 26.2 [22.3–28.3], $p_{\text{fdr}} = 0.054$, $r_{\text{rb}} = 0.31$; HbR: YA = 29.9 [22.9–39.0], OA = 25.1 [19.4–30.3], $p_{\text{fdr}} = 0.054$, $r_{\text{rb}} = 0.36$; HbT: YA = 27.1 [24.2–33.9], OA = 26.3 [19.9–27.8], $p_{\text{fdr}} = 0.054$, $r_{\text{rb}} = 0.30$).

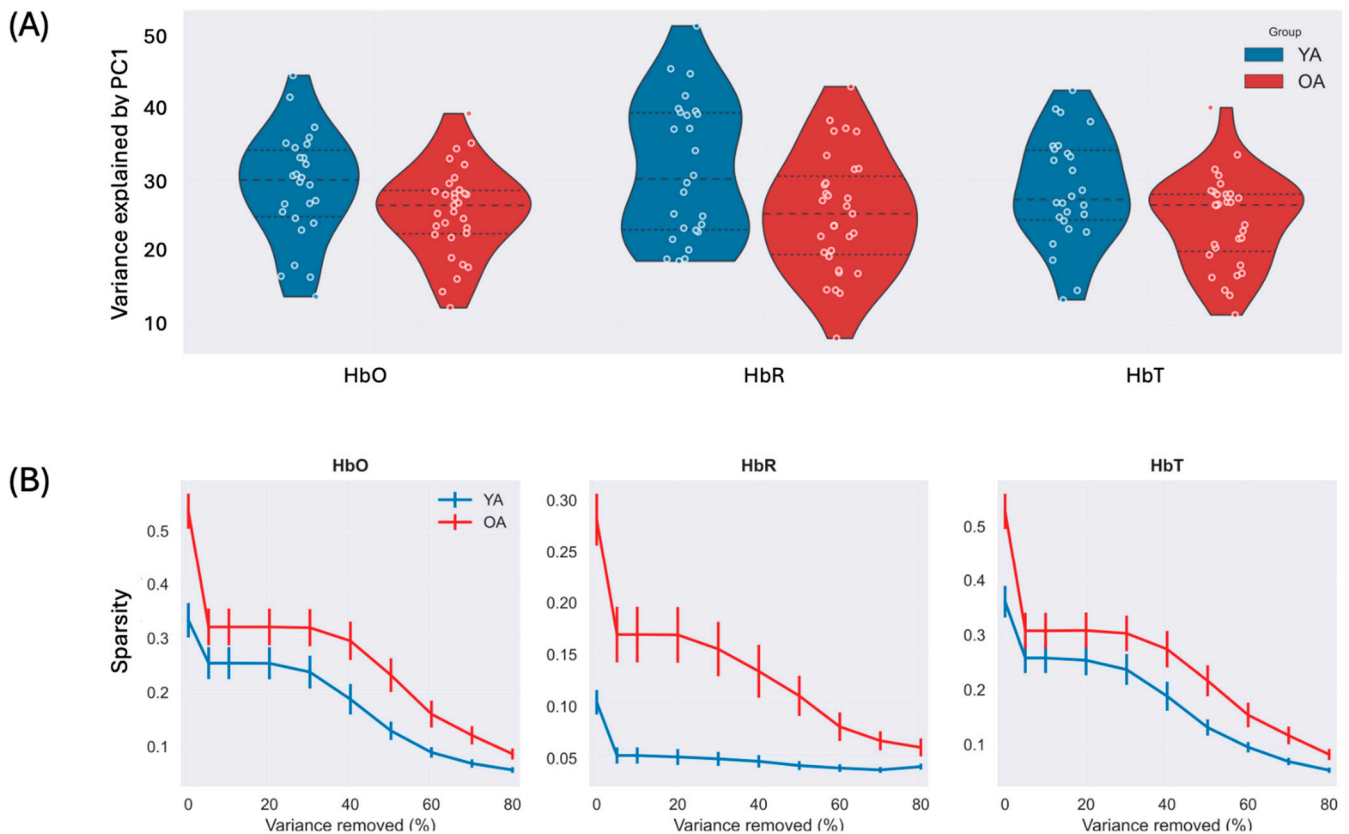


Figure 7. Influence of global variance removal on fNIRS-derived rsFC. (A) Percentage of total signal variance explained by the first principal component (PC1) for each hemoglobin contrast in young adults (YA) and older adults (OA). PC1 was computed separately for each run and averaged across runs to obtain one value per participant. (B) Median network sparsity as a function of the proportion of variance removed using PCA, independently of the number of components removed. Error bars indicate standard error across participants.

Furthermore, we performed an additional control analysis in which a fixed proportion of variance was removed from each participant's data using PCA, independent of the number of components required to reach that threshold (Figure 7B). This approach avoids arbitrary selection of principal components and ensures equivalent suppression of global variance across age groups. Across variance removal levels ranging from 0% to 80%, network sparsity decreased in both YA and OA groups. At every level of variance removed, OA consistently exhibited higher sparsity than YA across all hemoglobin contrasts. Notably these group differences persisted even after aggressive variance suppression (>60%), where substantial portions of global variance were removed.

Together, these findings indicate that the enhanced connectivity observed in OA cannot be attributed solely to differences in the amount of global variance removed during preprocessing. While vascular and extracerebral contributions remain intrinsically coupled to fNIRS signals and cannot be fully eliminated, the robustness of age-related differences

across a wide range of variance removal levels suggests that they reflect stable differences in large-scale hemodynamic network organization rather than preprocessing artifacts.

4. Discussion

In this work, we investigated the feasibility and sensitivity of fNIRS to capturing age-related alterations in resting-state functional connectivity (rsFC). Establishing standardized methods for quantifying network topology from fNIRS data, given its combined sensitivity to neural, vascular, and systemic physiological processes, is an essential first step toward enabling its use in longitudinal and clinical contexts. Because fNIRS is portable, affordable, and tolerant to movement, it offers a unique opportunity to extend rsFC research into naturalistic and less controlled environments such as patients' homes, day-care centers, or hospices, which can expand ageing research and increase information beyond clinical targets. Developing reference ranges and characteristic patterns of fNIRS-based graph metrics across the lifespan will be critical for future studies of neurodegenerative conditions associated with aging, including mild cognitive impairment (MCI), Alzheimer's disease, and vascular dementia.

A central methodological consideration in fNIRS rsFC analysis is the attenuation of non-neural hemodynamic variance. While short-separation channel regression is the most direct approach to address these contributions, our probe configuration did not include short-separation detectors. We therefore employed principal component analysis (PCA) as a spatial filtering strategy to reduce dominant global variance associated with systemic physiology. PCA identifies spatially coherent components that account for large proportions of shared variance across channels, and removing the first component has been shown to yield rsFC and graph metrics comparable to short-channel regression at the group level [38,49]. Nevertheless, systemic physiological processes can span multiple spatial components, and removal of only the first principal component, although widely adopted, may not fully eliminate non-neural variance.

To assess whether age-related differences in our results were driven primarily by differential variance suppression, we examined the proportion of variance removed by PCA in both age groups. The variance captured by the first principal component was slightly lower in older adults than in young adults, although this difference was not statistically significant. Notably, across participants, the first component accounted for approximately 20% to 40% of total variance, suggesting that non-neural contributions likely remained in the retained signal. Importantly, sensitivity analysis controlling for total variance removed demonstrated that older adults consistently exhibited higher network sparsity across a wide range of variance suppression levels (Figure 7B). This suggests that the observed group differences are not trivially explained by the magnitude of global signal removal alone. At the same time, these analyses cannot exclude qualitative differences in residual systemic variance between age groups, which are known to accompany vascular aging. More generally, no linear correction method—whether PCA or short-channel regression—can fully disentangle neural and non-neural contributions; as a result, residual vascular and systemic contributions are likely to persist to some extent, as indicated in previous work [38,50].

Within this methodological framework, our results revealed two specific fNIRS patterns in older adults compared to young adults: (1) a marked attenuation of low-frequency oscillatory power across all spectral bands and hemoglobin contrasts, and (2) systematic differences in network topology under matched sparsity conditions, characterized by reduced degree heterogeneity and increased segregation. These topological effects were network-specific, with the strongest age-related differences observed in the default mode, auditory, and frontoparietal control networks, and were accompanied by a shift toward

a more right-lateralized, posterior-hub organization in older adults. Together, these findings indicate that fNIRS captures reliable age-related differences in resting-state dynamics and hemodynamic network organization, reflecting combined changes in neural activity, vascular function, and systemic regulation that accompany healthy aging.

The substantial reduction in LFO power in older adults is consistent with prior fNIRS and fMRI studies and is commonly attributed to diminished neurovascular coupling efficiency, hemodynamic responsiveness, and impaired vascular reactivity [51–55]. In our data, LFO power was reduced by 70–85% across all frequency bands and chromophores, indicating a global suppression of spontaneous hemodynamic fluctuations rather than a band- or contrast-specific effect. This pattern likely reflects the combined influence of cerebral hemodynamic oscillations and age-related damping of systemic physiological rhythms associated with vascular remodeling and increased arterial stiffness [56,57]. In line with this interpretation, fMRI studies have demonstrated that age-related reductions in BOLD signal amplitude are strongly mediated by cardiovascular factors rather than neural activity alone [58], supporting an integrated neurovascular and systemic account of LFO attenuation measured with fNIRS.

These physiological changes may also explain the coexistence of reduced oscillatory power and altered network topology in older adults. Reduced oscillatory amplitude, coupled with increased vascular stiffness and altered cerebrovascular compliance, are expected to reduce the amplitude of spontaneous hemodynamic fluctuations and result in greater spatial coherence of global hemodynamic processes across the brain. From a methodological standpoint, two factors are particularly relevant when interpreting the measures derived from such signals. First, differences in signal-to-noise ratio (SNR) across age groups could influence signal variance and bias connectivity estimates. However, SNR did not differ systematically across participants and age groups. Second, reduced signal variance can itself influence correlation estimates when it disproportionately affects local variance while shared global structure is preserved [59]. Under these conditions, correlations become more sensitive to residual global variance, leading to inflated similarity between channels despite lower overall signal amplitude. Consistent with this mechanism, older adults exhibited lower global variance captured by the first principal component, and higher network sparsity persisted when the amount of variance removed was matched across groups. Together, these findings suggest that the apparent increase in spatial coherence in older adults likely reflects an interaction between age-related physiological damping of hemodynamic oscillations and statistical properties of correlation-based network metrics.

Interestingly, similar patterns have been reported in fNIRS studies of pathological aging, including Alzheimer's disease and MCI [60,61], as well as in recent work employing multi-distance separations [60,62]. While such findings have often been interpreted in terms of neural compensation or dedifferentiation, our results suggest that such interpretations should be made cautiously in fNIRS studies, as vascular and systemic factors may play a substantial role. From this perspective, fNIRS may be especially sensitive to early or subclinical alterations in neurovascular and systemic coupling that accompany aging, rather than providing a direct analogue of neural connectivity measured with fMRI.

Despite interpretative challenges, our findings demonstrate that fNIRS is sensitive to age-related changes in hemodynamic network organization. Still, several design considerations limit generalizability. Most notably, the substantial sex imbalance represents a significant confound. Although male-only analyses confirmed robustness of age-related effects, sex influences rsFC topology, vascular physiology, and hemodynamic responses are well documented [51,63,64]. Modest and non-significant sex differences within the older cohort may reflect insufficient statistical power rather than true absence of effects. The strong association between age and sex implies that some proportion of observed

age effects could reflect sex differences in hemodynamic or vascular function. Future studies should prioritize sex-balanced recruitment or employ formal sex stratification with adequate power.

In terms of fNIRS technology, sparse channel coverage (64 channels) precluded assessment of fine-grained cortical parcellation. In addition, the inherently sparse and subject-specific spatial sampling of fNIRS imposes limitations on seed-based connectivity analyses, as channel exclusion during quality control can introduce variability in the spatial distance between predefined seed locations and retained channels across participants. Moving forward, future studies combining fNIRS with other neuroimaging modalities and/or physiological recordings could better isolate neuronal from systemic signals and extend coverage to subcortical regions. Finally, the cross-sectional design cannot distinguish developmental trajectories from cohort effects or determine whether observed patterns predict cognitive outcomes. Longitudinal designs are essential to establish whether fNIRS-derived metrics reflect stable reorganization, progressive decline, or early markers of pathology.

5. Conclusions

Overall, our results provide evidence of robust age-related differences in fNIRS-derived signals. Older adults showed denser and more integrated fNIRS-based networks despite reduced oscillatory power, suggesting a complex interplay between neural and vascular factors. By characterizing how graph-theoretical metrics manifest in fNIRS data, we believe that this work contributes to laying the groundwork for using portable optical neuroimaging to monitor functional brain organization throughout the aging process and in neurodegenerative disease. Going forward, understanding the effects of fNIRS setups (e.g., channel density and location) on the variability of the network metrics will be key to ensuring reproducibility and comparability across fNIRS studies. In addition, larger normative datasets spanning different age groups can help define reference ranges for network density, clustering, and efficiency, serving as benchmarks for clinical research.

Supplementary Materials: The following supporting information can be downloaded at: <https://www.mdpi.com/article/10.3390/jal6010011/s1>. Figure S1: Network-specific topology obtained for oxy-hemoglobin (HbO) across canonical resting-state networks; Figure S2: Network-specific topology obtained for total-hemoglobin (HbT) across canonical resting-state networks.

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Abbreviations

The following abbreviations are used in this manuscript:

DMN	Default-mode network
fMRI	Functional magnetic resonance imaging
fNIRS	Functional Near-Infrared Spectroscopy
FPC	Frontoparietal control
HbO	Oxy-hemoglobin
HbR	Deoxy-hemoglobin
HbT	Total hemoglobin
IQR	Interquartile range
LFO	Low-frequency oscillation
MCI	Mild cognitive impairment
OA	Older adults
PCA	Principal component analysis
rsFC	Resting state functional connectivity
SCI	Scalp-coupling index
SNR	Signal-to-noise ratio
YA	Young adults

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