

Journal Pre-proof

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PII: S2589-0042(26)01233-2

DOI: <https://doi.org/10.1016/j.isci.2026.115858>

Reference: ISCI 115858

To appear in: *iScience*

Received Date: 9 February 2026

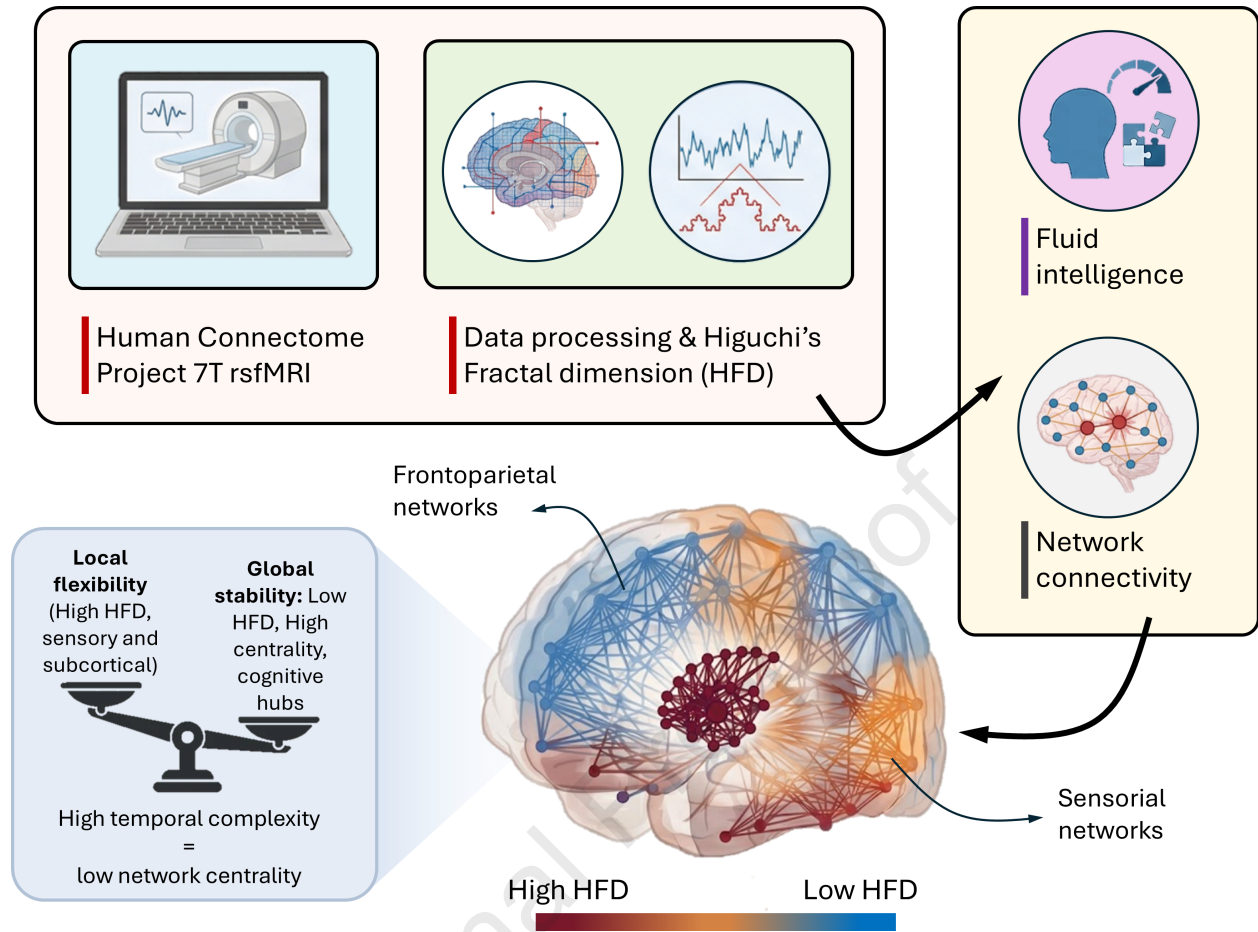
Revised Date: 11 March 2026

Accepted Date: 20 April 2026

Please cite this article as: Pini, L., Moaveninejad, S., Corbetta, M., Porcaro, C., Fractal Dynamics of the Healthy Human Brain: A Subcortical-to-Cortical Gradient of Temporal Complexity, *iScience* (2026), doi: <https://doi.org/10.1016/j.isci.2026.115858>.

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Fractal Dynamics of the Healthy Human Brain: A Subcortical-to-Cortical Gradient of Temporal Complexity

Lorenzo Pini,^{1,2,3} Sadaf Moaveninejad,¹ Maurizio Corbetta,^{1,2,3,6} Camillo Porcaro^{2,4,5,6,7*}

¹ Department of Neuroscience, University of Padova, Italy

² Padova Neuroscience Center, University of Padova, Italy

³ Veneto Institute of Molecular Medicine (VIMM), Padova, Italy

⁴ Institute of Cognitive Sciences and Technologies (ISTC) - National Research Council (CNR), 00185, Rome, Italy

⁵ Centre for Human Brain Health, School of Psychology, University of Birmingham, B15 2TT, Birmingham, UK

⁶ These authors contributed equally

⁷ Lead contact

* Correspondence: camillo.porcaro@unipd.it

SUMMARY

Loss of brain signal complexity can represent a sign of disease, yet standard scans often miss these patterns. Establishing a high-resolution benchmark of healthy temporal complexity is essential for developing early cerebral biomarkers. To construct a fractal normative reference map of brain complexity, we analyzed high-resolution 7T fMRI scans from 173 healthy adults. We used fractal dimension analysis to map the brain spatiotemporal organization and test its relationship with human intelligence. We discovered a robust physiological gradient, from the globus pallidus (hub of maximal 'healthy chaos') to fronto-parietal cognitive networks (which prioritize stability). This architecture predicts fluid intelligence. This study establishes the globus pallidus as the engine of neural flexibility while cortical network underlying cognitive abilities exhibit more regular dynamics, linked with fluid intelligence. These results offer insights to detect the dynamic rigidity that often precedes structural damage in neurological diseases, a hypothesis that requires validation in future clinical studies.

KEYWORDS

Fractal Dimension, Connectivity Networks, resting-state fMRI, Subcortical, Globus Pallidus, Fractal Normative map

INTRODUCTION

The temporal dynamics of brain activity contain latent diagnostic information about neural health that standard neuroimaging often overlooks. While physiological systems are characterised by their remarkable complexity, traditional diagnostic approaches often ignore the non-stationarity and nonlinearity of these signals.^{1,2} These dynamic signals carry not just noise, but specific pathophysiological signatures that reflect the system's capacity for adaptation and homeostasis. Recognising this diagnostic potential has sparked a growing interest in applying concepts from statistical physics to redefine pathological states, moving from the molecular to the organismic level.³

The human brain serves as a prime example of such complexity. However, describing the brain merely as a chaotic system is insufficient for clinical translation; we must understand how this complexity supports resilience against disease. Theoretical accounts of brain criticality propose that neural systems operate near a transition regime between order and disorder. In a medical context, this regime represents an optimal dynamic range where information processing capacity and responsiveness are maximised. This so-called 'edge of chaos' regime is functionally equivalent to

physiological homeostasis, enabling the brain to trade-off between stability (essential for reliable function) and adaptability (essential for learning and recovery after injury).^{4,5} Empirical evidence for this healthy variability comes from observations of neuronal avalanches,⁶ and scale-invariant complexity in brain signals.⁷ Consequently, a loss or alteration of this signal complexity is increasingly viewed as a prodromal biomarker for network collapse, distinct from structural atrophy, in conditions including epilepsy and neurodegeneration.^{8,9} For example, in Parkinson's disease (PD), a reduction in fractal complexity is a stronger predictor of cognitive decline than standard spectral measures, reflecting the rigidity of neural dynamics.¹⁰ Computational models of basal ganglia dynamics have demonstrated that oscillatory pathologies in PD, such as beta-band oscillations, can emerge from heterogeneous delays and bifurcation mechanisms within cortex-basal ganglia loops,^{11–13} providing a complementary mechanistic perspective on the dynamic changes captured by fractal analysis.

In this study, we use the Higuchi's fractal dimension (HFD) as a quantitative index of this neural resilience.¹⁴ The selection of HFD was based on i) robustness to relatively short time series, ii) computational efficiency, and iii) its growing application in systems neurophysiology as a tool to investigate disease progression and therapeutic.^{15,16} The brain dynamic repertoire is shaped by the balance between excitation and inhibition (E-I), which prevents both runaway excitation (as seen in seizures)¹⁷ and excessive suppression (as seen in coma or sedation).^{18,19} While HFD does not directly measure E-I balance, it captures temporal features of neural signals that are thought to reflect the underlying brain dynamic. Recent whole-brain dynamical modeling approaches have begun to provide a mechanistic perspective on E-I homeostasis,^{20,21} which could be integrated for mapping the baseline E-I balance via fractal dimension in diseased states.²²

Despite its potential, a normative '*Fractal Map*' of the human brain remains poorly mapped at high resolution.^{23,24} Resting-state fMRI (rs-fMRI) provides a valuable window into these dynamics.^{10,16} However, most studies focus solely on functional connectivity (FC), the spatial coordination between regions. Computational models predict that healthy brain function requires a specific synergy between local signal complexity (HFD) and global network integration (FC).²⁵ Thus, in pathological states, static FC maps alone may be insufficient to capture the full spectrum of early changes,²⁶ and that concurrent assessment of local signal complexity could reveal complementary signatures of network dysfunction.¹⁰

Here, we construct, for the first time, a healthy normative reference map of intrinsic brain dynamics by integrating indices of temporal complexity with FC. Our primary goal is to define a reference against which pathological deviations can be measured. Using a large public dataset (Human Connectome Project [HCP], 7T rs-fMRI), we aimed to map the hierarchical gradient of complexity and determine its relationship with cognitive reserve (fluid intelligence). While 7T imaging provides superior resolution, HFD is a scale-free metric that has been shown to be robust in prior studies using lower resolutions, supporting its potential translational utility in standard 3T clinical settings,^{10,23} although a formal cross-field-strength validation remains to be conducted.

We hypothesised that the brain follows a specific stability-plasticity gradient varying systematically along the subcortical-to-cortical axis.²⁷ We anticipated that evolutionarily older subcortical structures might exhibit distinct dynamical signatures compared to the cortex. In particular, the basal ganglia represent a key candidate because they regulate action selection and motor gating through cortico–basal ganglia–thalamo–cortical loops.^{28,29} Beyond their classical role in motor control, basal ganglia circuits have been shown to generate context-dependent neural variability, supporting adaptive motor behavior and flexible neural dynamics.³⁰ These properties suggest that basal ganglia networks may function as reservoirs of physiological complexity, influencing large-scale brain dynamics within

distributed cortical–subcortical networks.^{31,32} In contrast, the thalamus primarily serves as a sensory relay and coordination hub,³³ while the amygdala processes affective signals.³⁴ Therefore, the basal ganglia are uniquely suited for investigating the non-linear, fractal signatures of motor gating. We further hypothesised a functional trade-off between HFD and connectivity centrality. We posit that brain regions acting as global hubs must maintain lower complexity to ensure signal stability, whereas regions with higher local complexity support flexible processing.^{35,36,37} Finally, we examined whether individual variations in this ‘*fractal reserve*’ predict cognition. If verified, this could establish HFD as a potential clinically proxy for assessing brain functional integrity in neurological and psychiatric diseases.^{4,5,6} To ensure this metric is robust enough, we conducted a comprehensive sensitivity analysis of the temporal window parameters (K-Values) for the HFD and validated findings using Detrended Fluctuation Analysis (DFA). The workflow of the analysis is depicted in **Fig. 1**.

RESULTS

Study Population and Analytical Framework

The study included $n=173$ individuals from the HCP dataset with available rs-fMRI data (female 61%; age range: 22-36; range 22-25 years, $n=20$; range 26-30 years, $n=84$; range 31-35 years, $n=67$; +36 years, $n=2$). **Fig. 1** provides an overview of the analytical framework, while **Supplementary Figure S1** provides a representation of the data across different K-Values, showing the robustness of the results at the different temporal windows that could be taken into consideration.

Hierarchical Gradients of Brain Complexity

To define the fractal architecture of the healthy brain, we first assessed the multivariate patterns of HFD at the parcel-level. The analysis revealed three biologically distinct patterns from HFD maps considering an exemplative $K=50$ ($KMO=0.924$) (See **Fig. 2**). The first component explained 27% of the variance and mapped on frontoparietal parcels. The second explained 21% of the variance and mapped mainly over the motor and lateral occipital parcels (**Fig. 2**). Crucially, the third component isolated a distinct subcortical dynamic signature (14% variance) (**Fig. 2**). Overlapping and stable latent factorial HFD patterns were reported for different K values (10, 60, and 80). A post-hoc analysis was run to assess the correlation of the loading matrices (with fixed $n=3$) using the whole K span (from 10 to 180 with range of 5). We reported a very high correlated pattern, suggesting that the pattern reported in the main analysis and confined to a single K value was stable among multiple Ks (**Supplementary Figure S2**).

The Globus Pallidus: A Hub of Maximal Complexity

A key finding of this study is the identification of the Globus Pallidus (GP) as a core brain complexity hub. We assessed HFD values at brain parcels identifying parcels with the top and lowest values at different percentile-based thresholds (top/bottom 10% vs 90%, 15% vs 85%, and 20% vs 80% of the group-averaged HFD distribution across all 116 parcels), fixing $K=50$. The analysis showed that the highest HFD values mapped in the GP (**Fig. 3, Panel A**), while the lowest HFD values mapped mainly in posterior cingulate cortex, lateral parietal, and dorsolateral prefrontal parcels, although with more variability compared to the subcortical parcels (**Fig. 3, Panel B**). Further characterizing this via a winner-take-all approach, the GP represented the parcel with the highest complexity at a chosen $K=50$ (number of individuals ($n=94$ left globus pallidus; $n=77$ right globus pallidus; $n=2$ right accumbens)). These results were stable both below (e.g., $K=10$; $n=87$ left globus pallidus; $n=86$ right

globus pallidus) or above (e.g., $K=60$; $n=90$ left globus pallidus; $n=81$ right globus pallidus; $n=2$ right nucleus accumbens) this K threshold (see **Supplementary Figure S3**). The low inter-individual variability for the highest-HFD parcels likely reflects the stable neurophysiological properties of the GP (e.g., high tonic firing rates and complex inhibitory-excitatory microcircuit interactions) that generate high temporal irregularity as an intrinsic property of GP.

In contrast, the parcel with the lowest complexity showed more heterogeneity, mapping to cortical parcels. This heterogeneity was evident both at subject and threshold levels, mainly mapping to posterior cingulate, lateral parietal, and dorsolateral prefrontal regions (see **Supplementary Figure S4**). Due to the apparent heterogeneous results of the parcels exhibiting the lowest HFD values, a post-hoc comparison for cortical parcels was performed to explore whole-brain FC maps associated with those parcels. Details are reported in the **Supplementary Materials**. We identified two main seed-FC PCs (the first explaining more than 30% of the variance; the second around 11% of the variance). While the first PC showed a widespread distribution (and indistinguishable) likely capturing the “randomness” associated with the variability of parcels showing the lowest HFD, the second PC mapped specifically on the lateral parietal cortices, the posterior cingulate cortex, superior temporal gyrus, the dorsolateral prefrontal cortex and the medial prefrontal cortex (**Supplementary Figure S5**). This analysis showed that the apparent heterogeneous results for the lowest HFD seem to converge to a common high-cognitive cortical network. The FC seed-(lowest) ROI analysis was repeated for K values equal to 10, 60 and 80, yielding to identical results (**Supplementary Fig. S5**).

Fractality Follows a Functional Hierarchy

We confirmed that temporal complexity is not uniform but follows a specific functional axis. A significant pattern between network and HFD for different thresholds was observed at the ANOVA ($F[7,49]=635$, $p<0.0001$; **Fig. 3, Panel C**). Specifically, when all the 7 resting-state networks from Yeo et al.³⁸ and subcortical regions grouped together were analyzed, subcortical parcels showed the highest complexity value ($p<0.001$ for all the post-hoc comparisons), followed by the limbic network (LIM) ($p<0.001$ for all the post-hoc comparisons except the subcortical).

A significant post-hoc difference was reported also between the other networks (**Supplementary Table S1**). A clearer characterization of the differences among cortical networks was evident when grouping networks according to their domain of function, specifically sensorimotor (visual – VIS – and sensorimotor – SMN – networks), cognitive (default mode – DMN – and frontoparietal – FPN – networks), and attention (dorsal-attention – DAN – and ventral-attention – VAN). A significant result was assessed through the ANOVA ($F[2,4]=43$, $p=0.002$). Post-hoc analysis showed a pattern from higher to lower complexity following the sensorimotor-cognitive networks axis (attentional vs sensorimotor; $T=-6.86$, $p=0.021$; cognitive vs sensorimotor; $T=-8.72$, $p=0.013$), while no differences were reported between attentional and cognitive ($T=2.13$; $p=0.17$). These clusters were qualitatively confirmed in the UMAP 3D space (**Fig 3, panel D**).

The "Stability-Plasticity" Trade-off: Connectivity vs. Complexity

We identified a fundamental trade-off between a region's temporal complexity and its role in the global network. The partial least square on HFD and Eigenvector Centrality EC measure yielded to two significant components (compared to a null model; $n=1000$ permutations). The first component ($p<0.0001$) mapped mainly on sensorimotor (motor and visual) regions for HFD values and frontoparietal cortices for the graph measure. Notably the weights between the two sources of information were strongly negatively correlated ($\rho=-0.786$; $p<0.00001$), highlighting that higher

values in global connectivity were associated with lower HFD measurements. The second significant component ($p=0.001$) mapped on temporo-parietal lobe on HFD and insular-frontal cortices for EC, and the weights were again strongly anticorrelated ($\rho=-0.654$; $p<0.00001$). A very similar pattern was observed when we considered the Participation Coefficient (PC). The first significant component ($p<0.0001$ compared to a null model) mapped on sensorimotor cortices on one side (HFD) and frontoparietal parcels on the other (PC). The weights from the two measures were strongly anticorrelated ($\rho=-0.773$; $p<0.00001$), in line with the EC results. The second component ($p<0.0001$) mapped mainly on the frontal region (HFD) and occipitotemporal (PC) cortices, with a strong negative correlation ($r=-0.590$; $p<0.00001$) (see **Fig. 4**).

Secondly, we assessed the latent relationship between HFD and local connectivity measures, clustering coefficient (CL) and local efficiency (LE), which capture key aspects of local network organization. LE and HFD showed a single component surviving statistical threshold against a null model ($p<0.0001$). The weights between HFD and CL for the first latent component showed a slight significant positive correlation ($\rho=0.328$; $p=0.0008$), in contrast with the strong negative association with global connectivity measures. On the contrary, the two main components significant against the null model for LE (first component: $p<0.0001$; second component: $p=0.005$) showed no significant relation between HFD and LE weights (first component: $\rho=-0.010$; $p>0.05$; second component: $\rho=-0.194$; $p>0.05$). Results are shown in **Supplementary Figure S6**.

Statistical Causal Flow of Brain Dynamics

To understand how these complexity domains, interact, we applied a Linear Non-Gaussian Acyclic Model (LiNGAM). The inferred causal statistical ordering was subcortical, sensorial (visual-motor networks), attentional, and cognitive, suggesting hierarchical progression from subcortical-unimodal-transmodal systems. The estimated adjacency matrix showed that, cross-sectionally, the visual-motor systems received strong input from the subcortical regions (coefficient = 1.742), while the attentional system was influenced both by the subcortical (0.496) and sensorial (0.617) systems. Finally, the cognitive system received substantial input from the attentional system (0.888), as well as a (modest) negative input from the visual-motor systems (-0.146), potentially reflecting functional competition between these two domains (see **Fig. 5**). Due to the nature of LiNGAM model, the results of this analysis should be interpreted as statistical directionality rather than evidence of true causal mechanisms.

Fractal Dynamics Predict Cognitive Reserve

Finally, we tested whether these "fractal biomarkers" hold clinical relevance for cognitive function. We identified two main components of intelligence, explaining 30% of the variance. The first loaded on crystallized intelligence (18% variance) and the second on fluid intelligence (12% variance). While the first component did not show a significant relationship with HFD ($p>0.05$ FDR-corrected), the second component (fluid intelligence) showed a significant negative relationship with the DAN ($r=-0.215$; $p=0.005$), DMN ($r=-0.219$; $p<0.004$), and FPN ($r=-0.200$; $p<0.008$) HFD values, surviving FDR-corrected threshold. Of note, subcortical HFD values, analyzed at the aggregate level, did not show a significant association with either cognitive component. This suggests that neural efficiency in

individuals with higher fluid intelligence is characterized by more regular dynamics in higher-order networks (see **Fig. 6**).

Within and between K values

In addition to the multiple controls of the K value described above, we conducted two specific analyses to assess the stability of the observed patterns as K was systematically varied. We found high within-subject correlations across brain systems (aggregated cortical networks and subcortical regions), with Pearson's r values between different K values and the reference $K = 50$ consistently exceeding 0.88 and averaging above 0.95. Moreover, the complexity patterns at the level of brain systems were highly consistent across individuals as K varied from 10 to 90 in steps of 10. Results are presented in **Supplementary Figure S7**.

Detrended Fluctuation Analysis

To ensure these findings represent a universal property of brain signals, we validated the gradient using DFA. While HFD primarily reflects the degree of temporal irregularity and self-similarity of the rs-fMRI signal, DFA complement HFD, characterizing the persistence of long-range temporal correlations.

Results showed a structured gradient of complexity across the brain, with the lowest values observed in the aggregated subcortical network ($F[7,1204]=161$; $p<0.00001$; $\eta^2=0.198$). At the cortical level, reduced complexity was found in unimodal networks (VIS and SMN), whereas the highest DFA values characterized polymodal associative networks (VAN, DAN, and DMN). This pattern was in line with the subcortical–unimodal–polymodal gradient reported using HFD. Results are showed in the **Supplementary Figure S8** and post-hoc comparisons are reported in the Supplementary Materials. At subcortical levels the repeated measure ANOVA showed significant differences ($F[7,1204]=161$; $p<0.00001$; $\eta^2=0.198$), with the globus pallidus, a critical node in the basal ganglia, displaying the lowest DFA values. Subcortical post-hoc analysis and results are reported as **Supplementary Materials** and in the **Supplementary Figure S8**.

Further, we computed the Pearson correlation matrix of DFA values, using the HCP-averaged pattern as a reference. This matrix served as the input for clustering via the K-means algorithm, which revealed three distinct clusters ($K=3$ was identified as the most stable solution applying the consensus clustering approach): the first primarily encompassing sensorimotor-attentional networks (VIS, SMN, DAN, VAN); the second capturing higher-order cognitive networks (FPN, DMN); and the third corresponding to the limbic-subcortical axis (**Supplementary Figure S8**), again confirming the patterns reported by HFD.

DISCUSSION

In this study, we explored the relationship between fractality, brain connectivity, and cognition across distinct neural networks to construct a high-resolution '*Fractal Atlas*' of the healthy human brain. By integrating fractal analysis with network-based connectivity measures, we provide a multiscale description of intrinsic brain dynamics and their cognitive relevance. Rather than focusing solely on theoretical physics, we interpret our findings within a biomedical framework. We propose that this spatiotemporal atlas represents a fundamental marker of the brain's adaptive capacity. Our results are interpreted within a criticality framework, where scale-free temporal dynamics and network organisation reflect adaptive principles of brain function. Analogous to how stereotactic atlases guide

neurosurgery, this Fractal reference map establishes a normative baseline for detecting subtle, dynamic deviations in pathology.

The first major feature of this atlas is a systematic variation of HFD values along the subcortical-unimodal-transmodal axis. Specifically, lower HFD values were found in frontoparietal cognitive networks, including the frontoparietal, default mode, and dorsal-attentional networks, suggesting comparatively more regular and temporally structured dynamics. In contrast, higher HFD values were observed in sensorimotor networks, including visual and motor systems, which are continuously engaged in processing rapidly changing external inputs. These findings are consistent with previous work showing elevated fractal complexity in sensory resting-state networks, likely reflecting the need for flexible temporal dynamics to support continuous environmental monitoring and rapid adaptation.²³ Crucially, the robustness of this map was confirmed by the convergence of HFD with DFA. The network-level gradients were independently reproduced using DFA, a complementary scale-free metric that quantifies long-range temporal correlations. The convergence of HFD and DFA patterns strengthens the robustness of the observed organisational axis and indicates that it reflects a general property of temporal organisation rather than a metric-specific artifact. Specifically, HFD quantifies the degree of waveform irregularity and self-similarity, whereas DFA characterizes the persistence of temporal correlations across scales. The convergence of both metrics on the same organizational gradient strengthens the confidence that it reflects a property of brain temporal organization rather than a metric-specific result. Moreover, the unimodal-transmodal complexity axis was confirmed by the statistical causal directionality pattern (inferred via LiNGAM), showing a primarily bottom-up flow of causal statistical influence, originating in subcortical regions and following sensorimotor and attentional trajectories to shape cognitive processes.

At the deep end of our fractal normative map, very high HFD values were observed in subcortical regions, underscoring their complex roles in the brain functional architecture. Subcortical structures are known to participate in motor, cognitive, and affective processing and to modulate information flow between cortical systems.³⁹ From a connectivity perspective, subcortical nuclei have been described as integration zones linking multiple functional networks. Greene et al.⁴⁰ proposed the existence of motor, visual-attentional, and cognitive subcortical integration zones with distinct connectivity profiles. Our results suggest that elevated temporal irregularity, as indexed by HFD, may be a characteristic feature of these integrative roles, following a gradient from sensory to higher-order systems. Among subcortical structures, the GP consistently exhibited the highest HFD values across thresholds and individuals, serving as a '*landmark*' of maximal complexity. This region plays a central role in motor control, cognitive modulation, and affective regulation by shaping information transfer within cortico-basal ganglia loops.⁴¹ The GP comprises functionally distinct segments; the external segment, which serves as a hub within the indirect pathway exerting tonic inhibition on the subthalamic nucleus, and the internal segment, which acts as the primary output nucleus providing tonic inhibitory control over thalamocortical circuits. The parcellation employed in this study treats the GP as a single parcel. Future studies using finer-grained parcellations could examine whether these segments differ in their fractal properties.

Experimental evidence indicates that pallidal neurons can maintain high spike-time irregularity despite restoring mean firing rates following inhibitory perturbations, reflecting complex inhibitory-excitatory interactions at the microcircuit level.^{42,43} In the context of our results, high HFD reflects the need for "healthy chaos", temporally irregular dynamics that support flexible modulation rather than fixed or repetitive signaling patterns. This offers a potential mechanistic explanation for findings in PD, where cognitive decline is associated with a specific loss of fractal complexity in these basal

ganglia loops.¹⁰ It is important to highlight that the interpretation of HFD in relation to E-I balance should be considered within the constraints of fMRI. The signal from rs-fMRI is an indirect neural measure (hemodynamical), and inferences about neuronal E-I dynamics remain hypothetical rather than mechanistically conclusive.

Notably, DFA results confirmed this pattern, showing weak long-range temporal correlations in the globus pallidus, consistent with its high HFD and reinforcing the complementary interpretation of the two metrics. Taken together, these findings are compatible with theoretical accounts proposing that subcortical systems play a key role in dynamically modulating large-scale brain organization. Our results suggest that they contribute to maintaining a balance between temporal flexibility and network stability, a property often associated with criticality-inspired models of brain dynamics.^{35,44}

Our result suggests an inverse correlation between HFD and measures of connectivity centrality. Regions exhibiting high local temporal complexity tended to show lower centrality, whereas highly connected hubs displayed more regular temporal dynamics. This trade-off aligns with integration–segregation frameworks of brain organisation, which posit that efficient brain function arises from balancing globally integrated communication with locally specialised processing.^{36,45} Within a criticality perspective, this relationship can be interpreted as reflecting complementary roles of flexible local dynamics and stabilising hub regions, without implying that either directly indexes criticality.^{6,7} Consistent with this view, sensory networks displayed higher HFD values, indicative of greater temporal irregularity, whereas transmodal cognitive networks exhibited lower HFD and higher centrality, reflecting more structured dynamics that support sustained integration. Similar observations have been reported by Shine et al.,⁴⁶ who demonstrated that hub regions prioritise stability to facilitate global coordination. These findings suggest a division of labour in which temporal complexity and network centrality jointly support adaptive brain function.

At the cognitive level, we evaluated the clinical relevance of our atlas by linking these dynamics to fluid intelligence. We observed a statistically significant relationship between HFD and fluid intelligence, whereas crystallised intelligence showed no association. Fluid intelligence reflects flexible problem-solving and adaptive cognition and may therefore be more sensitive to intrinsic network dynamics. However, the magnitude of this association indicates that HFD explains only a small proportion of cognitive variance, consistent with multifactorial models of intelligence. Prior studies have reported inverse relationships between fractal measures and attentional or cognitive performance,^{47,48,49–51} supporting the notion that more regular, temporally structured dynamics in association networks may facilitate efficient cognitive processing. Our findings align with reports showing lower resting-state HFD in the default mode network in individuals with better cognitive performance, including in clinical populations.¹⁰

Importantly, these results should not be interpreted as indicating that higher or lower fractal complexity is universally beneficial. Rather, optimal cognitive function likely depends on context-dependent modulation of temporal complexity across networks, balancing flexibility and stability according to task demands. Overall, the results linking lower HFD with high connectivity centrality and better fluid intelligent performance could suggest that hubs prioritize stability to support global communication and cognitive flexibility. This highlights the cost of specialization. Regions with high centrality facilitate global integration but sacrifice local flexibility for network-wide efficiency.^{23,24} E/I balance may shift toward stronger inhibition to maintain the organised, less complex activity required for efficient task performance. Thus, our results, consistent with Porcaro et al.,²³ provide additional evidence that lower HFD in cognitive networks could support efficient, hierarchical network

organization. These results should be validated by future studies to confirm the observed associations and to elucidate the underlying neurobiological mechanisms.

In conclusion, this study establishes a high-resolution 'fractal atlas' of the healthy human brain. It demonstrates that temporal signal complexity is not noise, but rather a fundamental physiological property organized along a precise subcortical-to-cortical axis. Integrating fractal dimension analysis with network connectivity allowed us to identify a governing principle of neural organization: a trade-off between local dynamic flexibility (maximal in the GP and sensory systems) and global network stability (maximal in high-order cortical hubs). This framework challenges the cortico-centric view of brain dynamics by highlighting the basal ganglia as a critical engine of 'healthy chaos', which is essential for adaptive behaviour. In terms of potential clinical relevance, these findings provide a normative benchmark, and future studies could test whether deviations from this reference map, such as a specific loss of complexity in subcortical structures or destabilization of cortical networks, could serve as novel biomarkers for neurodegenerative and neuropsychiatric disorders. Translation to individual-level diagnostic utility will require prospective, longitudinal validation in clinical populations.

Limitations of the study

Some limitations should be acknowledged. First, although correlations with behaviour were statistically robust, effect sizes were modest and may have been influenced by range restriction and 'ceiling effects' in a healthy cohort.⁵² Consequently, the cognitive tests employed may have lacked sufficient sensitivity to capture the full variance in this population. Second, regarding the known sensitivity of HFD to parameter selection, we conducted the analysis using multiple K-values to ensure robustness. The reference value of K=50 was selected based on the curve stability criterion, corresponding to the inflection point where additional increases in K no longer produce meaningful changes in the estimated HFD, and was further supported by within-subject correlations exceeding $r = 0.88$ across K values. While this approach mitigates the risk of findings being driven by arbitrary parameter settings, fully data-driven optimization strategy (e.g., cross-validation) could complement this approach in future work. Third, all analyses were cross-sectional. The statistical directionality inferred from causal modelling should not be interpreted as evidence of true causal mechanisms, and generalizing these relationships across different tasks remains an open question. Finally, while this study establishes a framework in healthy individuals using high-field 7T MRI, its clinical utility depends on translation to standard 3T protocols. Future work should investigate HFD as a biomarker in neuropsychiatric and neurodegenerative conditions, where altered fractality may reflect shifts away from the brain critical state.¹⁰ Fourth, the neurovascular coupling underlying the rs-fMRI signal acts as a low-pass filter on neural activity. However, our analysis focuses on relative HFD differences across regions, and the convergence with DFA argues against metric-specific artifacts. Regional variability in neurovascular coupling, particularly in subcortical structures, deserves attention in future studies. Indeed, these brain structures exhibit substantial internal heterogeneity (e.g., the thalamus contains nuclei with different connectivity profiles; the striatum shows dorsal-ventral functional gradients). Our grouping of these regions reflects the resolution of the Tian et al.⁵³ parcellation rather than an assumption of homogeneity.

Despite these limitations, the present work provides a comprehensive description of how temporal complexity and FC jointly vary across brain systems. By framing HFD and DFA as complementary descriptors of scale-free temporal organisation rather than direct markers of criticality, this study advances a cautious but integrative framework for understanding intrinsic brain dynamics. Future longitudinal and computational modelling studies will be necessary to determine how these properties

evolve across cognitive states and pathological conditions and to assess their potential clinical relevance. In parallel, replication of these findings in multi-centre cohorts will be important to consolidate the generalizability of the proposed fractal normative brain map.

RESOURCE AVAILABILITY

Lead contact.

Requests for further information and resources should be directed to and will be fulfilled by the lead contact, Camillo Porcaro (camillo.porcaro@unipd.it).

Data and code availability

This paper analyzes existing, publicly available data, accessible at [DOI: <https://doi.org/10.1016/j.neuroimage.2013.05.033>]. Any additional information required to reanalyze the data reported in this paper is available from the lead contact upon request.

ACKNOWLEDGMENTS

This work was partially supported by PRIN Codice MUR 20228ARNXS (to CP and SM). LP is supported by the Associazione Italiana Ricerca Alzheimer Onlus (Airalzh-Grants-for-Young-Researchers - AGYR 2025). MC was supported by Visionary Nature Based Actions For Health, Wellbeing & Resilience in Cities (VARCITIES), Horizon 2020-SC5-2019-2 (Grant Agreement number 869505); Italian Ministero della Salute: Eye-movement dynamics during free viewing as biomarker for assessment of visuospatial functions and for closed-loop rehabilitation in stroke (EYEMOVINSTROKE; RF-2019-12369300), HORIZONERC- SyG (Grant No.101071900), HORIZON- INFRA- 2022 SERV (Grant No. 101147319) "EBRAINS 2.0: A Research Infrastructure to Advance Neuroscience and Brain Health". LP and MC are supported by Cariparo Foundation Excellence grant 2023-2024 (Agreement No. 68076; AWARENET project).

AUTHOR CONTRIBUTIONS

LP: former analysis, data processing, methodology, data visualization, interpretation, writing the original draft, SM: methodology, reviewing the draft; MC: methodology, interpretation, reviewing the draft, supervision, CP: former analysis, conceptualization, methodology, interpretation, supervision, writing the original draft, supervision.

DECLARATION OF INTERESTS

The authors declare no conflict of interest.

SUPPLEMENTAL INFORMATION

Document S1. Figures S1–S8, Tables S1, and supplemental references

FIGURE TITLES AND LEGENDS

Figure 1. Analytical workflow of the analysis.

From the preprocessed 7T HCP rs-fMRI data ($n=173$), Higuchi's fractal dimension (HFD) and connectivity measures were extracted at the parcel level ($n=100$ from Schaefer et al. and $n=16$ subcortical regions from Tian et al.). Three types of analyses were conducted: Panel A) A first-level analysis describing HFD values at the network and parcel levels, identifying regions and networks with the highest and the lowest HFD values. Panel B) A second-level analysis relating global and local connectivity measures (graph outcomes) to HFD values at the parcel and network level. Panel C) A third-level analysis exploring the relationship between HFD and cognitive variables (transformed through a factorial analysis - FA) of fluid and crystallized intelligence.

Figure 2. Low-dimensional embedding of complexity.

Panel A: Low-dimensional embedding of HFD values at the parcel level. The loadings of the first three principal components (PCs) are visualized for both cortical and subcortical regions. Panel B: loading matrix for the first three PCs. Each row represents a parcel grouped according to the Yeo's 7 network scheme. Abbreviations: VIS: visual network; SMN: sensorimotor network; VAN: ventral-attention network; DAN: dorsal-attention network; LIM: limbic network; FPN: frontoparietal network; DMN: default mode network; The dark-grey label represents the subcortical parcels. Panel C: variance explained (single: blue bars; cumulative: orange points) and eigenvalues for the first $n=20$ components. The KMO test is reported on the green box.

Figure 3. Lowest and highest complexity index at parcel and network levels.

Panel A: Parcels with the top 10%, 15%, and 20% HFD values are shown at surface level. Panel B: Parcels with the bottom 90%, 85%, and 80% HFD values are shown in the 3D render of the subcortical regions. The color bar indicates the HFD range of the parcels shown. Panel C: the upper panel illustrates the trend of HFD values grouped by network ($n=7$ cortical networks according to the Yeo et al. template, plus subcortical values). The subplot shows a detailed view for k from 30 to 70. The lower panel shows the trend of HFD values grouped by macro-categories of networks (sensory, attentional, and cognitive). ANOVA results are reported in the boxplots within the graphs. All the K considered in the analysis are displayed. Panel D: a 3D transformation of parcel-level HFD values for all individuals included in the analysis, grouped by cortical networks and subcortical parcels. The plots display the values for each of the three dimensions obtained from the dimensionality reduction of the $\text{HFD} \times \text{individuals}$ matrix using a representative K value of 50.

Figure 4. Complexity and functional connectivity centrality.

The results of the Partial Least Squares (PLS) analysis including HFD with graph measures from functional connectivity at the parcel level are shown. The blue plots in the graphs illustrate the relationship between the weights of the transformed variables. These weights are projected onto the surface for both included variables. Only components that are significant compared to a null distribution (red line in the KDE plot in the upper panel) are displayed. Results are presented for eigenvector centrality (upper panels) and participation coefficient (lower panels).

Figure 5. Statistical causal directionality estimated using DirectLiNGAM among four brain systems

The directed acyclic graph represents the inferred causal relationships among subcortical (aggregated), sensorial, attentional, and cognitive systems. Arrows indicate direction and strength of causal influence, with edge widths proportional to the absolute value of the effect size. Black edges denote positive causal influences, while red edges indicate negative effects.

Figure 6. Complexity and cognitive performance.

Top panels show the hierarchical factorial analysis performed on the selected cognitive tests. The panels on the left depict the loadings of the first two components for each test. The right matrix showed the correlation matrix of each cognitive test included. The bottom panels show the correlation between network HFD values and the two cognitive components. Only the p -values surviving the multiple comparison significance ($p < 0.05$ FDR) are reported. Abbreviations: ORRT: NIH Toolbox Oral Reading Recognition Test

90 (Age-Adjusted Scale Score); PVT: NIH Toolbox Picture Vocabulary Test (Age-Adjusted Scale Score);
91 PMAT: Penn Progressive Matrices (Number of Correct Responses); PLO: Variable Short Penn Line
92 Orientation (Total Number Correct); WMT: Working Memory Task (Overall Accuracy); LT: Language Task
93 (Overall Accuracy); CST: NIH Toolbox Dimensional Change Card Sort Test (Age-Adjusted Scale Score);
94 PST: NIH Toolbox Pattern Comparison Processing Speed Test (Age-Adjusted Scale Score); Flanker: NIH
95 Toolbox Flanker Inhibitory Control and Attention Test: Age-Adjusted Scale Score; PSM: NIH Toolbox
96 Picture Sequence Memory Test (Age-Adjusted Scale Score); LSWM: NIH Toolbox List Sorting Working
97 Memory Test (Age-Adjusted Scale Score).

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STAR★METHODS

KEY RESOURCES TABLE

REAGENT OR RESOURCE	SOURCE	IDENTIFIER
Deposited data		
Resting state fMRI data	Human Connectome Project	WU-Minn Consortium (principal investigators: D. V. Essen and K. Ugurbil; 1U54MH091657)
Software and Algorithms		
MATLAB	MathWorks	Version 2024; https://www.mathworks.com
Python	Python Software Foundation	Version 3.10; https://www.python.org
Brain Connectivity Toolbox (Python)	GitHub repository	Version 0.6.1; https://github.com/aestrivex/bctpy
LINGAM	GitHub repository	Version 1.12.2; https://github.com/cdt15/lingam
scikit-learn	scikit-learn developers	Version 0.23; https://scikit-learn.org
NumPy	NumPy developers	Version 1.26.0; https://numpy.org
Polars	Polars developers	Version 1.17.1; https://pola.rs

EXPERIMENTAL MODEL AND STUDY PARTICIPANT DETAILS

We included 173 healthy participants (61% females, age range = 22-35 years) from the Human Connectome Project (HCP), WU-Minn Consortium (principal investigators: D. V. Essen and K. Ugurbil; 1U54MH091657) funded by the 16 NIH Institutes and Centers that support the NIH Blueprint for Neuroscience Research and by the McDonnell Center for Systems Neuroscience at Washington University. This participant cohort comprises healthy individuals born in Missouri and the sample approximates the ethnic and racial distribution of the U.S. population recorded in the 2000 decennial census. This could, at least partially, influence the generalizability of results.

METHOD DETAILS

Resting-state functional 7T MRI dataset and preprocessing

Resting-state functional magnetic resonance (rs-fMRI) data of individuals from the HCP scanned at 7 Tesla were pre-processed according to the minimal pre-processing pipeline for HCP. Briefly, the data were slice-timing corrected, hole-brain intensity normalized, distortion corrected using synthetic field map estimation, and spatially realigned within and across fMRI runs. Data were regressed to remove sources of spurious variance from head motion (six parameters), the average signal over the whole brain, ventricles, and CSF, and white matter. These masks were identified through Structural MRI segmentation using Freesurfer (<https://surfer.nmr.mgh.harvard.edu>). Finally, temporal filtering was applied to retain frequencies in the 0.009–0.08-Hz band. Frame censoring was computed using framewise displacement with a threshold of 0.5 mm. Finally, the preprocessed rs-fMRI signal was averaged at parcel level using the Schaefer atlas (n=100).⁵⁴ For the subcortical regions we used the Tian et al.⁵³ parcellation (n=16 levels).

Higuchi's fractal dimension

The characterization of rs-fMRI signal by HFD was extensively described in our previous works,^{10,23,55–57}. Briefly, a fractal is a shape that retains its structural detail despite a scaling factor. For this reason, complex objects can be described by using HFD.^{15,58} HFD is a highly sensitive measure to detect hidden information contained in physiological time series,^{17,19,22,59} and one of the most widely accepted, and accurate.^{15,58} Here, we computed HFD, as described in the **Supplementary Materials**.

Univariate complexity patterns description

The first level of description (univariate) included the reporting of the parcels, across the group, showing the highest and the lowest complexity values. From each individual, the parcel with the highest or lowest

values were assigned a value of 1 (winner-take-all approach), while the remaining parcels were assigned a value of 0. A frequency map was then computed, corresponding to the number of individuals showing highest or lowest complexity values within a specific parcel. Secondly, we identified for each network the associated HFD value. Specifically, HFD values of parcels belonging to a specific network (according to the Yeo's 7 network template adapted for Schaefer parcellation plus the subcortical regions) were averaged for each K value.^{38,54} The 7 network were: visual network (VIS), sensorimotor network (SMN), dorsal-attention network (DAN), ventral-attention network (VAN), limbic network (LIM), frontoparietal network (FPN), and default mode network (DMN). Yeo's template was selected as one of the most widely used functional parcellation schemes in rs-fMRI studies, facilitating direct comparison with previous literature. An ANOVA was performed to assess differences on HFD values considering networks ($n=8$) and K values ($n=170$; from 10 to 180 with steps of 1). Further, we assessed differences by means of the ANOVA grouping cortical networks for macro-scale domains (sensorial: VIS and SMN; cognitive: DMN and FPN; attentional: DAN and VAN).

Multivariate description

A group PCA was performed to describe the HFD patterns. Specifically, the matrix $m \times n$ expressing the fractal values at each parcel (m) for each participant (n) was z-scored for subsequent dimensionality reduction. A hierarchical factorial analysis was conducted to reduce the dimensionality of the fractal data. The Kaiser-Meyer-Olkin (KMO) measure was used to assess the suitability of the data for dimensionality reduction. All the factor models ranging from 1 to the maximum possible number of factors were evaluated based on three criteria: (a) model fit, (b) interpretability, and (c) robustness. Model fit was determined by the percentage of total variance explained, aiming for a high value while balancing the other two criteria. For interpretability, we followed the guidelines of⁶⁰, requiring that each factor should have variables with loadings greater than 0.4. To enhance robustness, only latent factors explaining more than 10% of the variance were retained according to our previous publications.⁶¹ This analysis was performed on $K=50$ (main analysis) and secondary assessed for robustness spanning from $K=10$ to $K=180$ with steps of 5. Finally, we projected the group patterns into a 3D space for visualization purposes using the UMAP algorithm, according to our previous publication.⁶² For a specific value of K, we projected the entire matrix (individuals $\times$ parcels) into the new 3D space and grouped the data based on the networks to which the parcels belong.

Complexity and connectivity features relation

We explored the association between rs-fMRI signal complexity and connectivity features by means of Partial Least Squares (PLS) analysis. We selected a fixed $k=50$ to assess the parcel-wise latent relationship with two main connectivity measures from graph theory, one expressing global/centrality connectivity (i.e., eigenvector centrality), and the other as a measure of local connectivity (i.e., clustering coefficient). First, for each individual we computed the functional connectivity matrix using the preprocessed Brain rs-fMRI signal at parcel-level, resulting in a 100×100 matrix (using the same parcel scheme applied for HFD). The connectivity matrix of each individual was first sparsified according to the 80% connectivity value. The two selected graph measures were computed for each parcel and individual through the python version of the brain connectivity toolbox (<https://pypi.org/project/bctpy/>). To assess the robustness of the analysis, the same approach was performed considering two additional graph measures as local (local efficiency; LE) and global/centrality connectivity (participation coefficient; PC). The analytical details are reported in the

Supplementary Materials.

For each measure independently, we run a PLS analysis aimed at assessing the relationship between local and global functional connectivity measures and HFD on the same parcels and individuals (through Spearman's correlation of the resulting component weights considered significant at $p < 0.05$ Bonferroni corrected for the number of components extracted). Specifically, the aim of the PLS was to identify the latent components within complexity (HFD) and connectivity matrices that maximize the covariance between them. Data were z-scored and fed into a PLS analysis by means of the scikit-learn library in python (*PLSCanonical* function). We considered only the component significant compared vs a null model (permutation $n=1000$; stringent $p < 0.01$). Weights were plotted onto the surface at parcel-level to assess the spatial topology of the components and the correlation between values from the corresponding transformed values.

Linear Non-Gaussian Acyclic Model

To explore the statistical directional relationships cross-sectionally between brain systems characterized by different levels of functional hierarchy, we applied the Linear Non-Gaussian Acyclic Model (DirectLiNGAM)⁶³ algorithm. This method assumes that the observed variables are generated according to a linear, acyclic model with non-Gaussian, statistically independent error terms. Specifically, the model can be expressed as:

$$x = Bx + e$$

where x is the vector of observed variables, B is a strictly lower triangular matrix representing the direct causal effects, and e is a vector of external influences. The model was implemented using the lingam Python package (v1.5.9), and the adjacency matrix of direct effects was extracted after fitting. The causal ordering was derived from the inferred topological sort of the directed acyclic graph (DAG). The analysis was applied to the grouped cortical networks for macro-scale domains (sensorial: VIS and SMN; cognitive: DMN and FPN; attentional: DAN and VAN) and the aggregated subcortical parcels (as in the univariate analysis). First we tested the distribution of each variable using the Shapiro-Wilk test. We then computed the model residuals as:

$$E = X - B \cdot X,$$

with B as the adjacency matrix and X the data matrix ordered according to the model and tested them for non-Gaussianity. The resulting causal structure was visualized as a directed graph, with edge colors and thicknesses reflecting the sign and strength of the direct effects.

Network complexity and cognitive profile

To characterize the relationship between HFD and cognitive profile, we assessed the linear relationship between performance in a series of cognitive tests and HFD averaged at network level. For cognitive outcomes, we selected a series of cognitive tests to characterize the fluid and crystallized intelligent profile. The full list of cognitive variables included are reported in the *Supplementary Material*. First, cognitive variables were fed into a hierarchical factorial analysis to retrieve the most significant components. Components were selected according to three main criteris: i) model fit (percentage of total variance explained and should be as high as possible while still maintaining the other two criteria); ii) b) interpretability (from Velicer and Fava⁶⁰ - each factor should have at least three variables with loadings > 0.4, here ehnahted to 0.5); iii) robustness. Further, latent factors with eigenvalues higher than 1 and explaining more than 5% of variance were retained.⁶¹ A non-parametric relationship was then assessed between the included cognitive components and the HFD at network level. A $p < 0.05$ corrected for FDR was considered statistically significant.

Sensitivity analysis

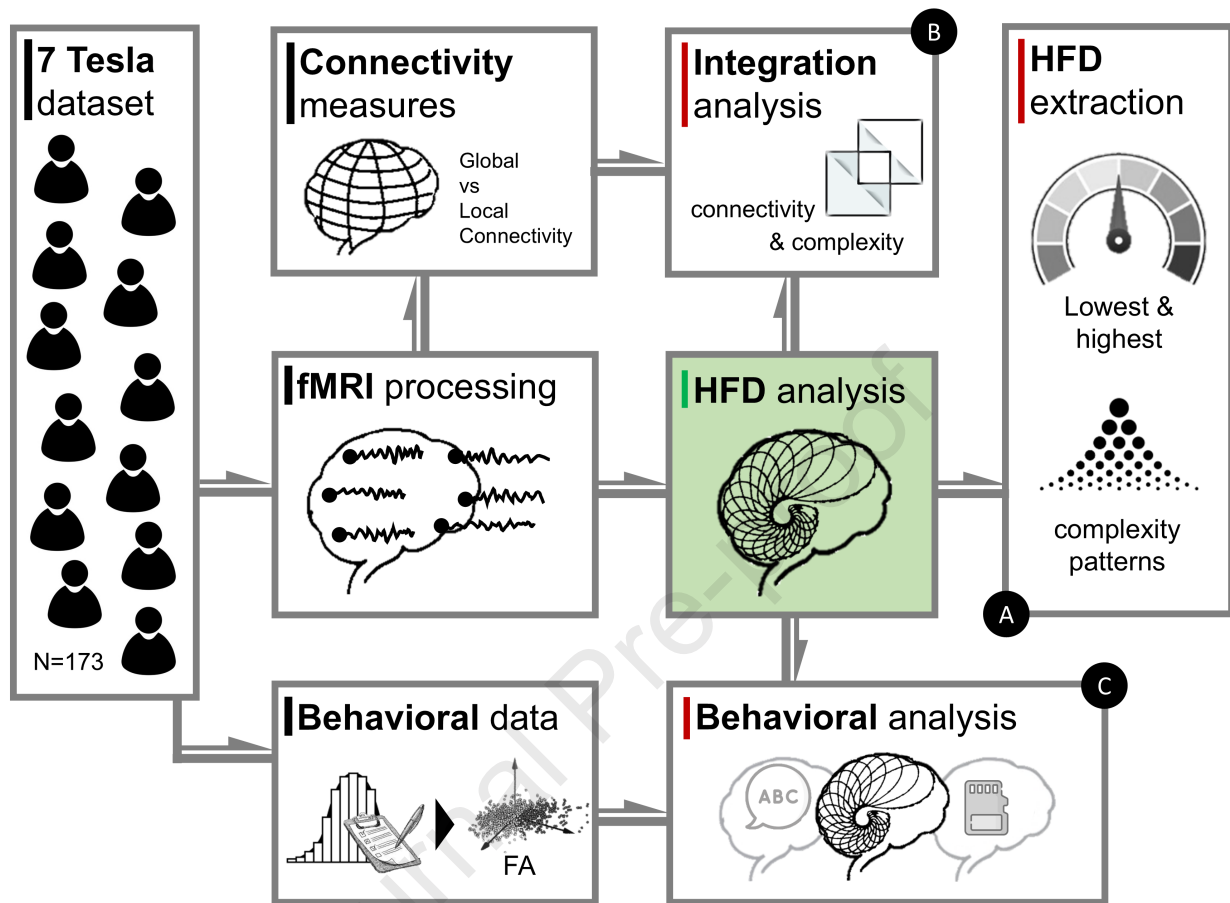
We applied a detrended fluctuation analysis (DFA), a technique quantifying the fractal scaling behavior of non-stationary time series, on the same data assessing the robustness of our results. Details of DFA computation are reported in the *Supplementary Materials*. Cortical networks and aggregated subcortical (plus hippocampus) DFA values were compared using repeated-measures analysis of variance (ANOVA), with statistical significance set at $p < 0.05$. The same network and subcortical parcellation applied to HFD were used. Post-hoc pairwise comparisons were performed with Bonferroni correction, considering adjusted p -values < 0.05 as significant. Furthermore, we characterized the inter-regional correlation of DFA values, averaged across the $n=173$ HCP participants, across the cortical networks (plus subcortical aggregation). The resulting 8×8 correlation matrix was subjected to k-means clustering. The optimal number of clusters was determined using the silhouette score computed from a consensus clustering procedure testing solutions ranging from 3 to 8 clusters.

QUANTIFICATION AND STATISTICAL ANALYSIS

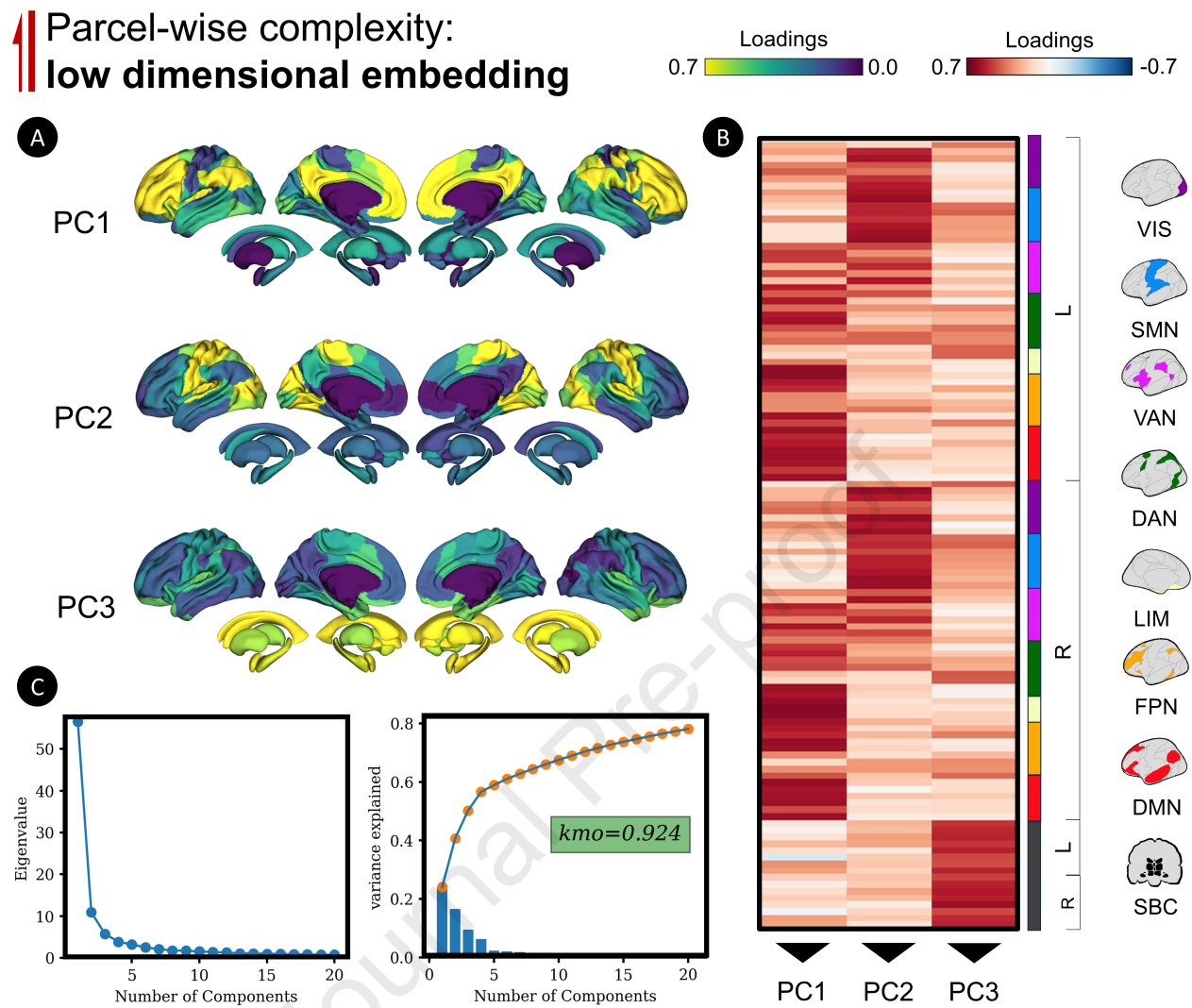
All the analysis was run on Python 3.9 through an ASUS TUF Dash F15 machine (12th Gen Intel(R) Core (TM) i7-12650H 2.30 GHz) running on an Ubuntu 20.04.6 LTS (Focal Fossa) environment while a HP machine (11th Gen Intel(R) Core(TM) i7-1165G7 2.80 GHz) running on Windows 11 (v. 24H2) with Matlab v2021a was used for computing the fractality of the data. Data are reported as mean $\pm$ standard deviation (SD). Variables were z-scored prior to multivariate analyses. Repeated-measures ANOVA was used for network-level comparisons, with Bonferroni correction for post hoc tests. Spearman's rank correlation was

371 used for non-parametric associations. For PLS analyses, significance was assessed using permutation
372 testing ($n=1000$; $p<0.05$), and correlations between component scores were Bonferroni-corrected ($p<0.05$).
373 False Discovery Rate (FDR) correction was applied for cognitive analyses. Normality was tested using the
374 Shapiro-Wilk test when required. No randomization or sample size estimation was performed, as this study
375 is a secondary analysis of a publicly available dataset. Inclusion was based on data availability and quality
376 control procedures of the HCP release.

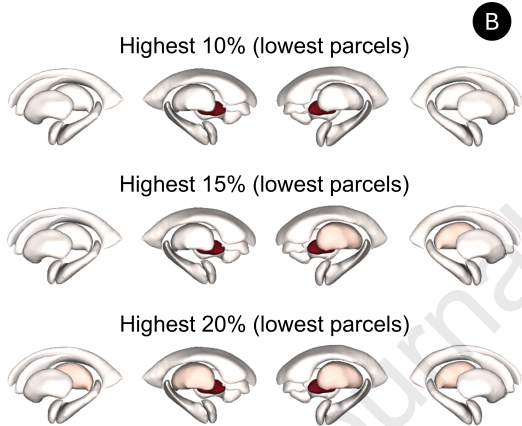
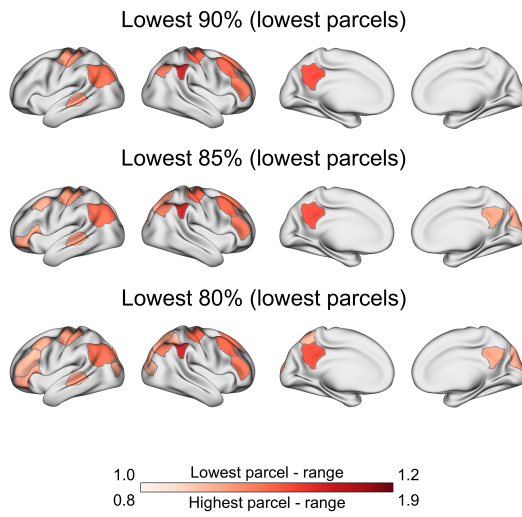
Workflow of the analysis



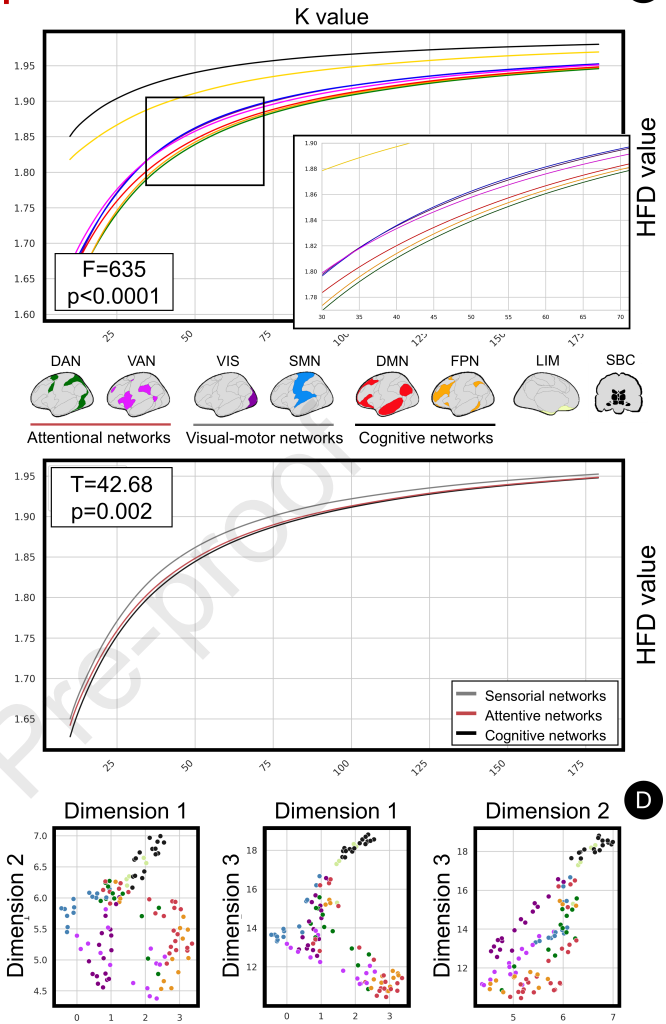
Parcel-wise complexity: low dimensional embedding



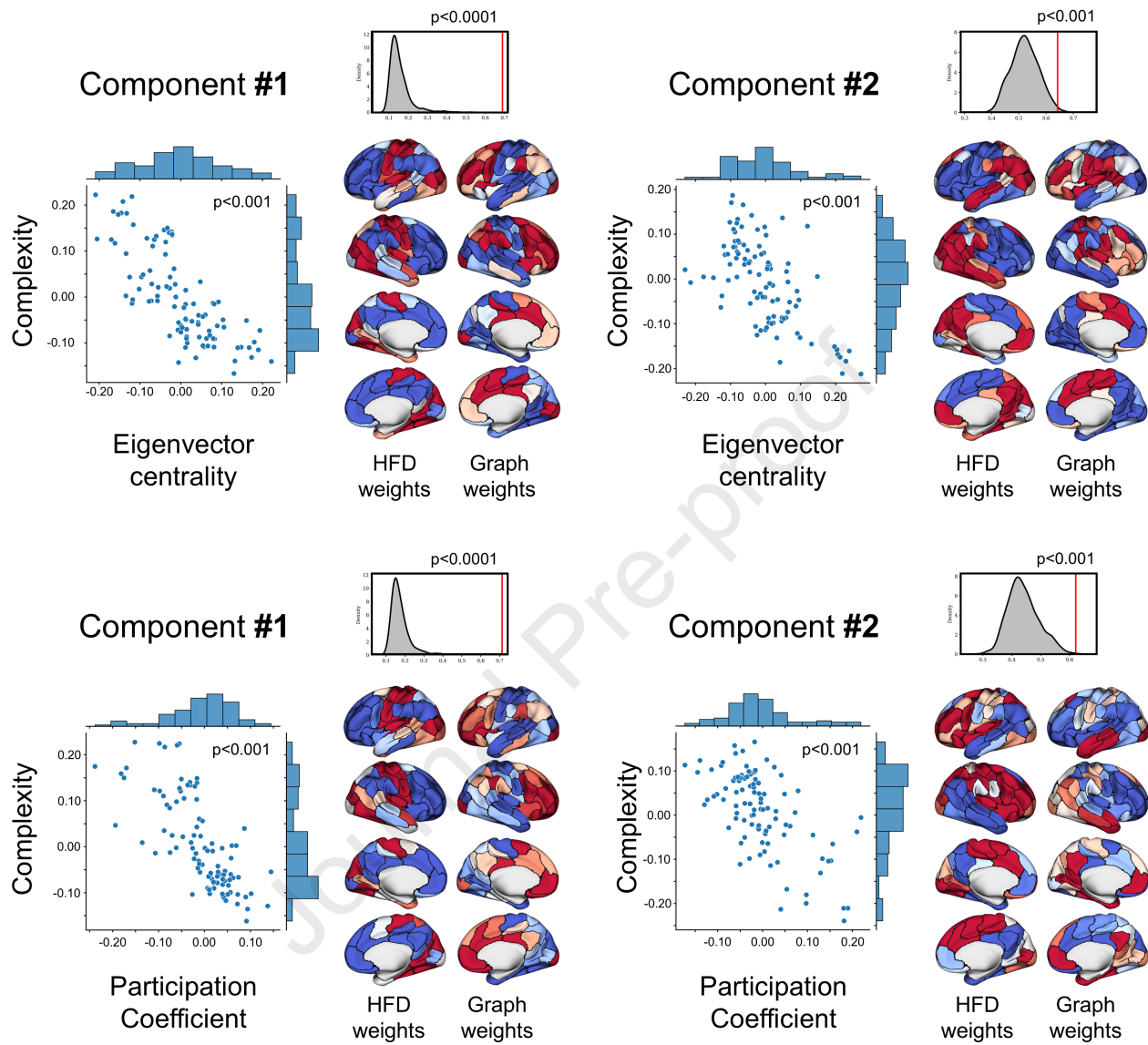
Lowest and highest HFD parcels



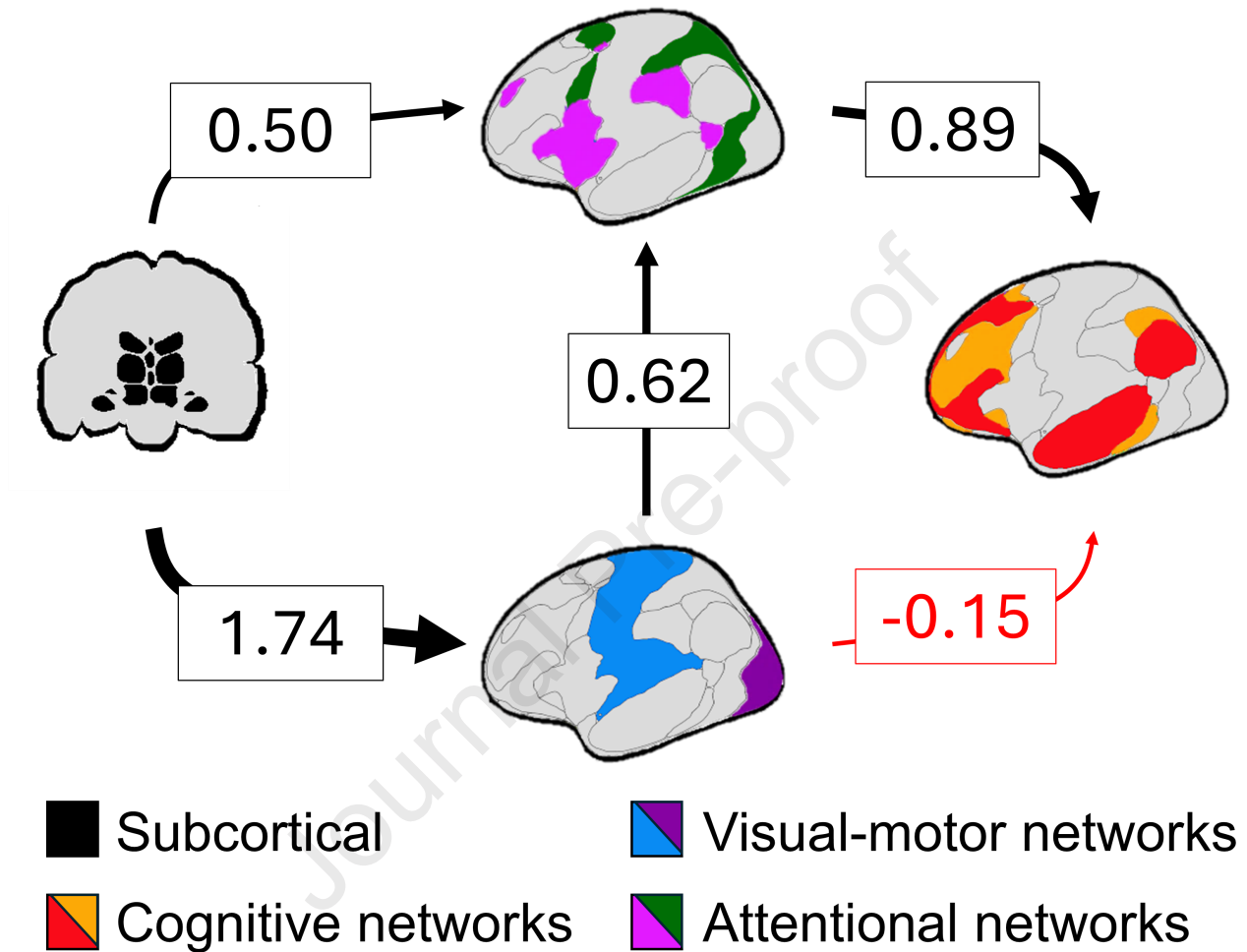
Functional networks and HFD values



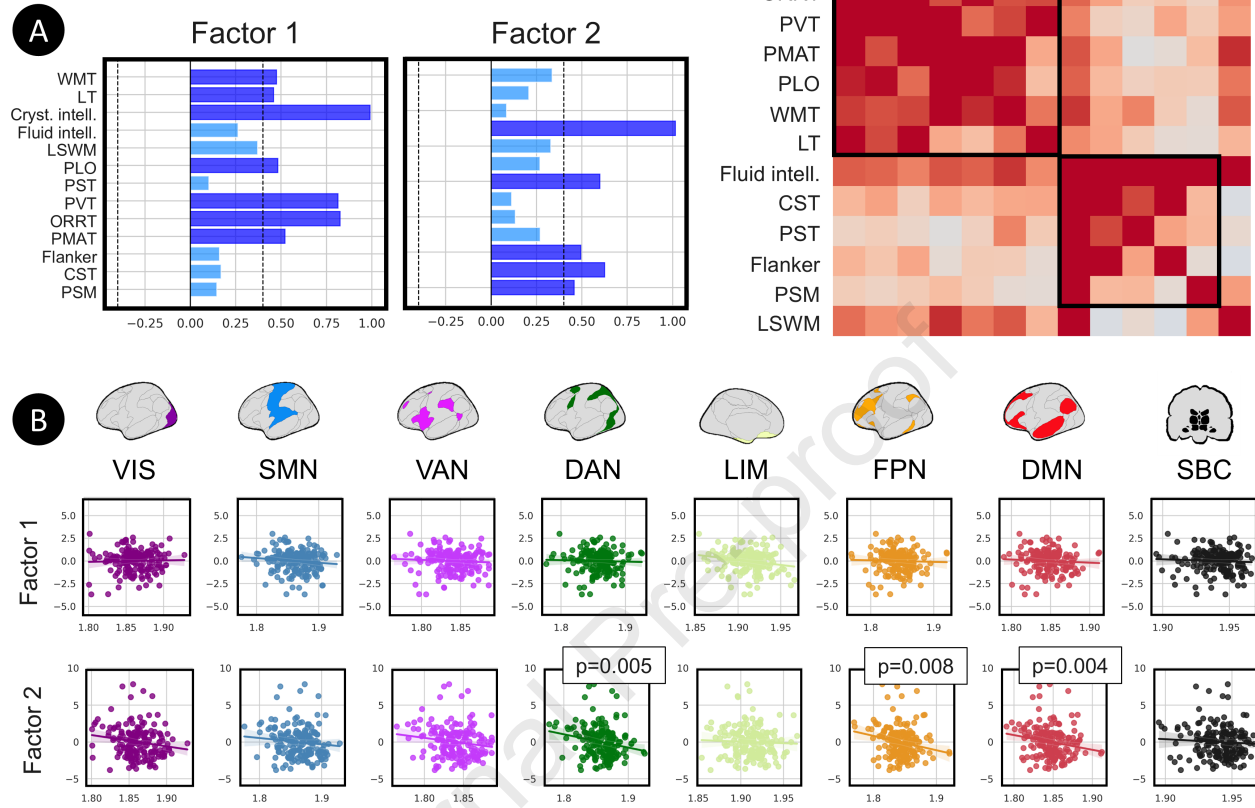
Relationship between FC and HFD



Linear non-Gaussian acyclic model



Relationship between cognitive and complexity



Fractal Dynamics of the Healthy Human Brain: A Subcortical-to-Cortical Gradient of Temporal Complexity

Lorenzo Pini,^{1,2,3} Sadaf Moaveninejad,¹ Maurizio Corbetta,^{1,2,3,6} Camillo Porcaro^{2,4,5,6,7*}

¹ Department of Neuroscience, University of Padova, Italy

² Padova Neuroscience Center, University of Padova, Italy

³ Veneto Institute of Molecular Medicine (VIMM), Padova, Italy

⁴ Institute of Cognitive Sciences and Technologies (ISTC) - National Research Council (CNR), 00185, Rome, Italy

⁵ Centre for Human Brain Health, School of Psychology, University of Birmingham, B15 2TT, Birmingham, UK

⁶ These authors contributed equally

⁷ Lead contact

* Correspondence: camillo.porcaro@unipd.it

HIGHLIGHTS

- Fractal map reveals subcortical-to-cortical complexity gradient
- Globus pallidus shows highest temporal complexity across individuals
- Cortical brain complexity inversely relates to global connectivity centrality
- Brain cortical complexity predicts higher fluid intelligence