A Hitchhiker’s Guide to Temporal Complexity for Resting State fMRI Analysis

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2025-12-10

Abstract

For decades, fMRI analysis has predominantly focused on linear relationships, such as simple signal magnitude changes and pair-wise functional connectivity. Recent evidence suggests that failing to account for the brain’s non-linear, dynamic organization may lead to incomplete inferences about neural processes. More recently, cognitive and clinical neuroimaging have begun to draw on tools from complexity science to characterize the nonlinear dynamics of the brain. Temporal complexity metrics reflect a range of approaches to complexity in time series, including describing the system’s regularity and irregularity, predictability and unpredictability, information compressibility, long-term memory, and fractal patterns. In functional magnetic resonance imaging (fMRI), applications of temporal complexity are scattered across siloed literatures with varying clarity, which limits accessibility and therefore popularity. This review aims to bridge this gap by communicating the basics of temporal complexity to fMRI scientists. We offer a comprehensive guide to temporal complexity in fMRI, including an overview of fMRI temporal complexity metrics—Shannon entropy, variations of (multi-scale) sample entropy, Lempel-Ziv complexity, avalanche measures, and Hurst—followed by a comprehensive review of extant applications in fMRI.

## Introduction

The brain is a complex system that exhibits nonlinear dynamics across multiple dimensions, including in how its activity unfolds over time. Capturing these dynamics requires more than traditional static summaries of average activity or connectivity. Instead, approaches are needed that explicitly quantify the temporal richness of brain function. One informal grouping of such approaches is temporal complexity ([Figure 1](#fig-wordcloud)). Temporal complexity metrics reflect diverse assumptions and motivations, but broadly aim to quantify regularity and irregularity in time series, including features like recurring patterns, memory, and unpredictability (Varley, Luppi, et al. 2020). Temporal complexity is the most common of the complex-systems approaches to the brain (e.g., see Sun et al. (2020) for a domain-specific review (Alzheimer’s)), and is present across scales ([Figure 2](#fig-scales)), from the cellular to large-scale functional networks (Cofré et al. 2025).

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| Source: [Figures](https://WeberLab.github.io/A-guide-to-temporal-complexity-for-fMRI-scientists/notebooks/Figures.ipynb.html#cell-wordcloud)  Figure 1: **A variety of approaches to nonlinear analysis of fMRI data.** A Python-generated word cloud of fMRI complexity terms, weighted by number of results in PubMed. Keywords were selected from reviews of nonlinearity/complexity (including Sarasso et al. (2021), Sun et al. (2020), Hernández et al. (2023), Keshmiri (2020), Donoghue et al. (2024), and A. C. Yang and Tsai (2013)). |

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| Figure 2: **The multiscale brain.** The brain is displays complexity at the microscopic, mesoscopic, and macroscopic scales, both in temporal and spatial domains. Cartoons adapted from Ros et al. (2014); BOLD time series adapted from Cifre et al. (2020); layout inspired by Betzel and Bassett (2017). |

Functional magnetic resonance imaging (fMRI) can indirectly assess temporal complexity in brain activity through the blood-oxygen level dependent (BOLD) signal. Despite the fact that fMRI is traditionally considered unsuitable for rigorous temporal analyses — its time series are sampled slowly (0.5–3 s), and are relatively short in length (5–10 minutes), limiting access to fine-scale dynamics and data-hungry metrics (Grandy et al. 2016) — a growing literature shows that coarse-scale dynamics remain informative. These dynamics have been linked to both cognitive and clinical states and have be reliably characterized using fMRI recordings (Moses O. Sokunbi 2016; Xin et al. 2021).

Compared to other neuroimaging modalities (reviewed in (Sarasso et al. 2021; Sun et al. 2020; Hernández et al. 2023; Keshmiri 2020; A. C. Yang and Tsai 2013)), however, temporal complexity metrics are seldom used in fMRI research. This is reflected in a diffuse, unstandardized landscape in which methods are siloed across research groups, limiting accessibility and comparability. The present review aims to help bridge this gap. Going beyond previous reviews of complexity in fMRI [Moses O. Sokunbi (2016); xinApplicationComplexityAnalysis2021], we have attempted to systematically identify all temporal complexity metrics used in fMRI research and all relevant papers that used each metric. To our knowledge, no prior review has undertaken this task at this scope.

This review also differs from previous reviews in its primary aim. Rather than summarize prior clinical and neuroscience findings (although we do include this as well), we have focused on providing guidance for clinical and cognitive researchers who wish to implement temporal complexity metrics. Many such researchers lack a background in information theory and thus face a choice: either apply temporal complexity measures without fully understanding the theoretical context — risking methodological or interpretational errors — or invest considerable time learning about a vast, interdisciplinary, and technically varied literature. Our review aims to solve this problem by serving as both a comprehensive and accessible resource for temporal complexity methods in fMRI.

What follows is first a review of the basic concepts of various temporal complexity metrics and the history of their applications to fMRI, followed by a comparison of these metrics, and finally a discussion that explores their clinical and neuroscience findings. ## What is complexity?

Complexity is conceptualized as involving a balance between order and disorder (Sporns 2010). Uniform systems (e.g., crystal lattices; sine waves) are not complex, nor are completely irregular ones (e.g., randomly moving particles; white noise); rather, complexity lies between these two extremes, combining predictability and unpredictability (Varley, Luppi, et al. 2020).

## Variability

In order to get a sense of temporal complexity, it may help to first describe how it differs from variability. Complexity and variability are statistically distinct, yet closely intertwined. Temporal complexity and variability both describe the temporal dynamics of neurophysiological signal, with variability describing signal spread, and complexity describing within-series interactions and recurring patterns ([Figure 3](#fig-variability)). A time series that is more complex is not necessarily more variable, and vice versa. Indeed, some measures of complexity explicitly make use of variability as a measure across different scales (e.g., Approximate entropy (Richman and Moorman 2000) or Rescaled range to measure the Hurst exponent (Hurst 1951)). Nevertheless, complexity and variability are closely related. Across theoretical perspectives, changes in a system (i.e. it’s variance) are fundamentally related to the complexity of its behaviour (Lotfi et al. 2021; Garrett et al. 2013; Chialvo 2018; He 2011). Intuitively, a system cannot exhibit complex behaviour if its variability is either minimal or maximal — that is, if it is in complete stasis or fluctuates completely randomly.

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| Figure 3: Conceptual comparison of the features captured by the mean (a measure of central tendency), variance (a measure of variability), and Hurst exponent (a measure of complexity; specifically long-term memory). Synthetic waveform from B. Li et al. (2012); figure inspired by Garrett et al. (2010). |

A review of empirical comparisons between variability and complexity in fMRI is provided in the later section “Comparisons of All Metrics.”

## Entropy measures

Entropy, in the context of time-series and brain signals, is a measure of uncertainty, irregularity, or unpredictability in a signal. It originates from information theory and statistical mechanics, but has been adapted into many specialized forms for analyzing neural dynamics. It is by far the most popular and diverse measure we encountered in our literature search, and is thus where we begin our review.

### Shannon entropy

Shannon entropy serves as the foundation for virtually all contemporary entropy measures, and its introduction marked the birth of information theory (Shannon 1949). Shannon entropy quantifies uncertainty; it can be thought of as the difficulty of predicting an observation’s value ([Figure 4](#fig-entropyuncertainty)). Applied to a time series, it measures the minimum number of ‘bits’ needed to encode the series based on the frequency of values ([Figure 5](#fig-entropybolddistribution)).

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| Figure 4: Shannon entropy represents uncertainty about an observation’s value. Image adapted from Serrano (2018). |

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| Figure 5: Shannon entropy reflects the distribution of values; a wider distribution of BOLD signal values (blue) has higher entropy, while a narrower distribution of values (green) has lower entropy |

Shannon entropy can be calculated using the following equation:

where is entropy and is the frequency of a given value.

Shannon entropy is seldom used to assess the temporal complexity of neuroimaging time series. Instead, it is most commonly applied to spatiotemporal properties—for example, to quantify the complexity of functional connectivity (FC) (Viol et al. 2017; Pappas et al. 2019). This is because the classic formulation only considers frequency values, not their order. The resulting metric is poorly suited to describe how a series changes over time. Only one study, by Huang et al. (2025), used Shannon entropy of frequency of values (details in discussion). However, several studies have applied Shannon entropy to wavelet-decomposed time series (i.e., analyzing the entropy of different frequency bins; method described in Rosso et al. (2001)), which has been shown to be an improvement over the classic formulation. For example, Gupta et al. (2017) found that Shannon wavelet entropy was able to discriminate epilepsy patients from controls, whereas classical Shannon entropy was not.

Although Shannon entropy is rarely applied with the explicit aim of describing temporal complexity, it has been used as an innovative alternative to traditional methods of analyzing event-related fMRI data. Unlike traditional methods, entropy makes no assumptions about the shape of the evoked hemodynamic response, which can improve stability and sensitivity (Mikoláš et al. 2012; Ostwald and Bagshaw 2011). De Araujo et al. (2003) were the first to apply Shannon entropy to event-related fMRI analysis. They calculated entropy within time windows during activation and rest; the rise and fall of the entropy measure mirrored the theoretical hemodynamic response ([Figure 6](#fig-shannonentropyevents)). Compared to traditional cross-correlation, Shannon entropy was more stable with a changing signal-to-noise ratio (De Araujo et al. 2003). DiNuzzo et al. (2017) also used Shannon entropy in event-related analysis, which allowed them to capture changes induced by photic stimulation that were missed by traditional temporal descriptors (e.g., variance). Together, these results indicate that Shannon entropy may capture features of BOLD dynamics that escape conventional temporal measures. More broadly, this demonstrates the usefulness of temporal dynamics in situations where spatial maps are unable to fully capture condition differences.

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| Figure 6: An example of how Shannon entropy can be used to analyze event-related data (de Araujo et al., 2003). Image stylized/recreated from de Araujo et al. (2003). |

Several extensions to Shannon entropy have been introduced as alternatives to conventional methods for discriminating task conditions in event-related fMRI. A comprehensive list of these methods and their applications is as follows: Tsallis entropy (applied by (Sturzbecher et al. 2009; X. Wang et al. 2013), Renyi entropy (Gonzalez Andino et al. 2000; X. Wang et al. 2013), generalized relative entropy (Cabella et al. 2009; Welvaert and Rosseel 2012), adaptive entropy rate (Fisher et al. 2001), and time-lagged mutual information (Tsai et al. 1999; Fuhrmann Alpert et al. 2007; Alpert et al. 2008; Tedeschi et al. 2004, 2005; Von Wegner, Laufs, and Tagliazucchi 2018). Despite their potential effectiveness in event-related fMRI analysis, these methods have not been widely adopted, neither since they were first reviewed by Mikoláš et al. (2012), nor in the decade since. This may be because of their high computational demands or simply because there are better approaches to model-free hemodynamic response estimation (e.g., finite-impulse-response models (Metzak et al. 2011). No studies have systematically evaluated whether Shannon-entropy-based analysis methods discriminate task conditions better than conventional ones, which leaves a knowledge gap.

### Kolmogorov-Sinai entropy

In contrast to Shannon entropy, Kolmogorov-Sinai (KS) entropy takes time into account ([Figure 7](#fig-ksentropy)), allowing for application to dynamical systems (Nogueira 2017). As a system evolves, KS entropy quantifies the average rate at which information is produced with each new state; that is, it quantifies the difficulty of predicting future observations given past observations. Higher KS entropy implies that a higher amount of information is being introduced at each time point, meaning that the series is more unpredictable. Like Shannon entropy, KS entropy implements the intuitive notion that a broader distribution of data values should correspond to greater uncertainty.

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| Figure 7: Because Shannon entropy only depends on the distribution of values and not their order, two sequences may look different but have the same Shannon entropy. Here, both series have the same Shannon entropy, but the series on the right has higher KS entropy. Original image |

KS entropy is difficult to apply to real-world data, given that proper implementation requires exceedingly large amounts of time series data and an absence of noise (Pincus 1991; Richman and Moorman 2000). As such, a variety of measures have been developed as approximations for KS entropy. The following sections discuss approximations of KS entropy that have been applied to fMRI research.

### Approximate entropy

In fMRI, nearly all approximations of KS entropy can trace their historical lineage to approximate entropy (ApEn) (Pincus 1991). First used on cardiovascular time series and later adapted for neuroimaging (Richman and Moorman 2000; Bruhn, Röpcke, and Hoeft 2000), ApEn quantifies the probability that sequences of similar patterns in a time series will remain similar when sequence length is increased. A higher value of ApEn signifies that the signal contains fewer repeating patterns—that is, greater complexity.

In the calculation of ApEn, each sequence is counted as matching itself (Richman and Moorman 2000). This nuance leads to two limitations. First, it causes ApEn to be lower than expected for shorter time series (Richman and Moorman 2000). This is because counting self-matches inflates the estimation of regularity, and this over-inflation is more prominent for short series. Shortening datasets (e.g., by using one fMRI run instead of two) may change ApEn despite identical patterns. Second, ApEn lacks relative consistency, meaning that comparisons between datasets can be affected by the choice of time-window and tolerance parameters (i.e., if ApEn is higher for one dataset than another using one set of parameters, we would expect this to remain true for other choices of parameters, but this is not the case; (Richman and Moorman 2000)).

Given these limitations, ApEn is rarely used to characterize temporal complexity in fMRI. In most fMRI studies, ApEn is reported only alongside other entropy-based measures — such as sample entropy (SE) or fuzzy approximate entropy — and therefore these applications are included in our “Comparisons of All Metrics” section. Two ApEn studies that do not appear in that section are M. O. Sokunbi et al. (2011) and C. Y. Liu et al. (2013); both report clinical findings that align with the broader complexity literature, and we summarize these in the Discussion.

### Sample entropy

Sample entropy (SE) was developed to correct ApEn’s limitations (Richman and Moorman 2000). The difference between the calculation of SE and that of ApEn is that SE doesn’t count self-matches in the conditional probability, which corrects the two limitations described in the ApEn section (Richman and Moorman 2000) (for an example in fMRI, see (A. C. Yang et al. 2018). In addition to these advantages, SE is a better approximation of KS entropy and is simpler to compute (Richman and Moorman 2000). Accordingly, SE is more widely used than ApEn in fMRI and can be computed using the toolboxes BENtbx [https://github.com/zewangnew/BENtbx; e.g., Wang et al., 2014](https://github.com/zewangnew/BENtbx) (e.g., Z. Wang et al. (2014)) or Complexity Toolbox [http://loft-lab.org/index-5.html; e.g., Zhang et al., 2021](http://loft-lab.org/index-5.html) (e.g., N. Zhang et al. (2021)). SE is most often called “brain entropy” (BEN) (Z. Wang et al. 2014); however, because BEN can also refer to Shannon entropy (Akdeniz 2017), we elected to use the term SE in this review.

To calculate SE, the user specifies two parameters: the time window and the tolerance scale . is the number of values to be analysed at a time, and is the multiplied by the standard deviation (SD) of the series to obtain the tolerance. SE is equal to the negative average natural logarithm of the conditional probability that two sequences that are similar (i.e., within the tolerance) for points will stay similar for points (Richman and Moorman 2000). Because conditional probability is between 0 and 1, SE will always be positive. When the time series is highly regular (i.e., when similar runs remain similar), SE is low; when the time series is irregular, SE is high (Pincus 1991). See [Figure 8](#fig-sampleentropyconceptual) for a conceptual explanation, [Figure 9](#fig-sampleentropycomputational) for a computational explanation, and Delgado-Bonal and Marshak (2019) for a tutorial. For a detailed guide to selecting and , see Appendix A1 and Figure A1.

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| Figure 8: **Conceptual explanation of SE.** SE represents the probability that similar sequences will stay similar. Original image. |

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| Figure 9: **An example SE calculation with .** Original image, except for time series (black dots/lines) which is adapted from Roediger et al. (2024). |

What is the minimum number of timepoints needed for SE? In general, more timepoints improve results. For example, using simulated data, Grandy et al. (2016) found that SE accuracy and precision increased fairly linearly from the shortest to longest series tested (32 to 32,768). Wehrheim et al. (2024) ([Figure 10](#fig-SEtimepoints)) observed strong (albeit non-significant) correlations between reliability and series length, including a slow increase in split-half correlation from the shortest to longest series tested (100-800), along with a steady increase in test–retest correlation that plateaued around 500. Highlighting that the relationship between length and reliability is dataset–dependent, they found differences in rest versus task data (for a wide variety of tasks, including cognitive, motor, and social). As for the lower limit of length, A. C. Yang et al. (2018) identified a lower bound of 97 timepoints; for series shorter than 97, SE could still be calculated, but with a narrower range of and values. Moses O. Sokunbi (2014) found that SE discriminated between young and old adults in series as short as 85 in a small (n = 20) cohort. Again highlighting that length limitations are dataset-dependent, Moses O. Sokunbi (2014) found that more data points were needed in a larger (n = 86) cohort (potentially because the smaller cohort had low inter-individual variability).

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| Figure 10: SE accuracy (and precision, not shown) increase with increasing series length. Figure stylized based on results from Wehreim et al. (2024). |

Several innovations to SE have been made in recent years. G. Del Mauro and Wang (2024) introduced “cross” SE, a method for comparing multiple SE maps which can be applied to make comparisons between subjects, sessions, scan times, or regions. Thus far, cross entropy has been used to show a decoupling between the (across-subject) mean and variance of SE across different brain regions (G. Del Mauro and Wang 2024). Z. Wang (2021) pioneered dynamic SE, wherein SE is estimated for every segment of a sliding window. Despite its name, dynamic SE is not designed to be used to track changes in SE over the course of a run; instead, SE for each segment is combined into a final run mean. In a sample of 862 subjects, Z. Wang (2021) found minimal differences between static (traditional) and dynamic SE.

[Table 1](#tbl-fmriSE) includes a complete summary of fMRI-SE studies and their findings.

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| Table 1: **fMRI-SE and MSE studies.** An attempt to gather all published fMRI studies that have used SE or MSE, some stats, and the main findings. Main findings are more nuanced than how we have reported them here; we have attempted to condense the findings as succinctly as possible. Excludes analyses that do not meet the definition of temporal complexity used in this review: SE of the spatial map, SE of dynamic functional connectivity, and SE computed on synthetic data. For sample size and age, commas separate cohorts. / = Higher / lower; ACC = anterior cingulate cortex; AD = Alzheimer’s; ADHD = attention-deficit hyperactivity disorder; ASD = autism spectrum disorder; corr. w/ = Correlated with; diff. = difference; DLPFC dorsolateral prefrontal cortex; DMN = default mode network; DPN = diabetic peripheral neuropathy; FG = frontal gyrus; FPN = frontoparietal network; HC = Healthy controls; MCI = mild cognitive impairment; MDD = major depressive disorder; MOFC = medial orbitofrontal cortex; MTL = medial temporal lobe; n.s. = not significant; OA = older adults; OCD = obsessive-compulsive disorder; OFC = orbitofrontal cortex; oppositional defiance disorder = ODD; PFC = prefrontal cortex; SMA = supplementary motor area; TPJ = temporal parietal junction; VMPFC = ventromedial prefrontal cortex; YA = young adults.   | Reference | Sample | Age (mean sd or min - max; years) | TR (s) | Volumes | Rest/task | Method (SE or MSE) | Parameters | Results | | --- | --- | --- | --- | --- | --- | --- | --- | --- | | Maximo, Nelson, and Kana (2021) | ASD 45, HC 45 | 8-14 | 2.5, 2 | 120, 152 | rest | SE | = 2,  = 0.46 | ASD: SE in left angular gyrus, superior parietal lobule, right inferior temporal gyrus; SE in superior FG. | | Z. Wang et al. (2017) | Cocaine 20, HC 19 | 42 4, 40 5 | 2 | 180 | rest | SE | = 3, = 0.6 | Cocaine addiction: SE in VMPFC, OFC, DLPFC, ventral striatum, basal ganglia, visual cortex, parietal cortex. | | Fu et al. (2024) | ASD 42, HC 42 | 1-1.5 | 2 | 240 | rest | SE | = 3, = 0.6 | ASD: SE in right inferior FG. | | Sevel et al. (2020) | 26 | 25-45 | 1.5 | 360 | rest | SE | = 3, = 0.6 | Acute alcohol intake: Within regions with reductions in regional signal variability, SE in bilateral middle FG, right superior FG. | | D. Song and Wang (2024b) | YA 44, OA 43 | 18-31, 63-81 | 2 | 240 | rest | SE | = 3, = 0.6 | Intranasal oxytocin: In left TPJ, SE in YA, in OA. | | Z. Zhao et al. (2024) | 280 | 38-44 weeks postmenstrual age | 0.392 | 2300 | rest | SE | = 3, = 0.3 | Postnatal age corr. /w SE in sensorimotor-auditory cortex, association cortex, right rolandic operculum. Gestational age corr. /w SE in sensorimotor-auditory cortex and association cortex, and corr. /w SE in right rolandic operculum. Pre-term infants: SE in visual-motor cortex. | | Varley, Luppi, et al. (2020) | Dataset A 25, Dataset B 19 | 19-52, 18-40 | 2, 2 | 150, ? | rest | SE | = 2, = 0.3 | SE with increasing propofol sedation. | | X. Jiang et al. (2021) | OCD 74, HC 93 | 18-60 | 2 | 200 | rest | SE | = 3, = 0.2 | SE map did not match brain structure. No difference between OCD and HC, or between genders. | | Sharma et al. (2024) | Concussion 28, HC 379 | 9-17 | 2 | 180 | rest | SE | = 3, = 0.6 | Concussion vs HC classification: Most important region for classification was subcallosal cortex. | | Çatal et al. (2022) | Dataset A 50, Dataset B 130 | 18-50, 21-50 | 2, 2 | 288, varies | rest, task | SE | = 2, = 0.5 | Interoceptive > Exteroceptive > Mental ROIs for all of rest and task. Rest > Task. | | Xiaoyun Liu et al. (2020) | MDD 85, follow-up 30, HC 45 | 44 14, 41 14, 43 12 | 2 | 240 | rest | SE | = 3, = 0.6 | MDD: SE in MOFC/subgenual-ACC; SE in motor cortex. | | D. Song, Chang, Zhang, Ge, et al. (2019) | 107 | 31 14 | 2 | 240 | rest | SE | = 3, = 0.6 | Cerebral blood flow corr. /w SE in OFC, posterior inferior temporal cortex. n.s. direct relationship between SE and gender. | | Gianpaolo Del Mauro and Wang (2024) | 1206 | 29 4 | 0.72 | 1200 | rest | SE | = 3, = 0.6 | n.s. relationship between SE and heart rate. Cognition corr. /w SE in fronto-parietal cortex, in sensorimotor system. | | Easson and McIntosh (2019) | ASD 20, HC 17 | 10-18, 8-18 | 2 | 180 | rest | SE | = 1-4, = 0.05-0.8 | ASD: n.s. diff. in SE. SE corr. /w global efficiency of structural connectome and age, corr. /w behavioural ASD score. | | Saxe, Calderone, and Morales (2018) | 892 | 18-35 | 3 | 120 | rest | SE | = 3, = 0.6 | Intelligence corr. /w SE, especially in PFC, inferior temporal lobes, cerebellum. | | Xiang Liu et al. (2023) | Classical trigeminal neuralgia 85, HC 79 | 56 (interquartile range 13), 55 (interquartile range 14) | 2 | 240 | rest | SE | = 3, = 0.6 | Classical trigeminal neuralgia: SE in thalamus and brainstem, SE in inferior semilunar lobule. | | Shi et al. (2019) | 386 | 17-27 | 2 | 242 | rest | SE | = 3, = 0.6 | Divergent thinking corr. /w SE in left dorsal ACC/pre-SMA, left DLPFC. Fluency, flexibility, and originality corr. /w SE in left inferior FG and left middle temporal gyrus. | | Z. Wang and for the Alzheimer’s Disease Neuroimaging Initiative (2020) | HC 54, Significant memory concern 27, early MCI 58, late MCI 38, AD 34 | 65-95, 65-83, 56-89, 57-88, 56-87 | 3 | 140 | rest | SE | = 3, = 0.6 | Aging in HC: SE. Aging in AD: SE. In AD SE corr. /wimpairment. Cerebrospinal fluid Aβ depositions corr. /w SE in HC, while corr. in AD. Important regions: DMN, MTL, PFC. | | Z. Wang et al. (2014) | Small cohort 16, Large cohort 1049 | 25 5, 27 11 | 3, 3, 0.75-3 | 220, 220, 72-395 | task, rest | SE | = 3, = 0.6 | Atlas of SE: SE reproduces known network parcellations. | | Shan et al. (2018) | Chronic fatigue syndrome 43, HC 26 | 47 12, 43 14 | 0.798 | 1100 | task | SE | = 3, = 0.2 | Chronic fatigue syndrome: SE in 10 of 50 regions. | | Allendorfer et al. (2024) | TBI only 60, TBI with psychogenic nonepileptic seizures 21, TBI with epileptic seizures 56 | 38 12, 39 12, 38 12 | 1 | 1200 | task | SE | = 3, = 0.6 | TBI with psychogenic nonepileptic seizures: SE corr. /w depression. TBI only, TBI with epileptic seizures: n.s. correlation between SE and depression. Across all groups: SE in FPN. | | Chang et al. (2018) | 60 | 23 3 | 2 | ? | rest | SE | = 3, = 0.6 | Caffeine intake: SE across cerebral cortex, with the highest increase in lateral PFC, DMN, visual cortex, motor network. | | Mackenzie Joel Leavitt (2022) | 31 | 19-25 | 3 | 100 | task, rest | SE | = 3, = 0.6 | Task vs rest: n.s. diff. in SE. | | W. Jiang, Cai, and Wang (2023) | Marijuana dependence 59, HC matched to marijuana group 59, Nicotine dependence 34, HC matched to nicotine group 34, Alcohol dependence 35, HC matched to alcohol group 34 | ? | 0.72 | 1200 | rest | SE | = 3, = 0.6 | Marijuana dependence: SE. Nicotine dependence: SE. Alcohol dependence: SE. | | Niu et al. (2020) | 3 cohorts of HC: 29, 35, 36 | 21 2, 31 9, 27 8 | 2.5, 2, 1.75 | 197 | rest | SE | = 2, = 0.25 | Reliability (test-retest): Poor to fair to good. | | Gale, Nezafati, and Keilholz (2021) | 412 | 22-35? | 0.72 | 1200? | rest, task | SE | = 3, = 0.6 | Task (emotion, language, sensorimotor, gambling/risk-taking, relational processing, social processing, combination working memory/category-specific representation): Regions predicted to be active based on the literature < Regions predicted to be less relevant. Amplitude corr. /w SE in task but not rest. | | Xue, Wang, et al. (2019) | MDD 46, HC 32 | 28 9, 27 10 | 2 | 240 | rest | SE | = 3, = 0.6 | MDD: SE across whole brain and in bilateral thalami, bilateral insula, bilateral putamen, left caudate, right inferior FG. Depression score corr. /w SE. | | Gianpaolo Del Mauro et al. (2024) | YA 577, OA 424 | 29 4, 61 15 | 0.72, 0.8 | 478, 478 | rest | SE | = 3, = 0.6 | n.s. relationship between SE and pain intensity. | | Gianpaolo Del Mauro, Zeng, and Wang (2025) | 2415 | 8-89 | 0.72 | 488 | rest | SE | = 3, = 0.6 | Increase in SE from childhood to older adulthood | | Gianpaolo Del Mauro et al. (2025) | Chronic pain 13132, HC 18173, Non-chronic pain 4922 | 64 8, 64 8, 63 8 | 0.735 | 490 | rest | SE | = 3, = 0.6 | Chronic pain: SE. | | R. Zhang, Cen, et al. (2025) | ADHD 61, ODD 38, OCD 48, comorbid ADHD/ODD/OCD 833, HC 269 | 10 1, 10 1, 10 1, 10 1, 10 1 | varied | varied | rest | SE | = 2, = 0.3 | In executive function networks: Comorbid-free ADHD < HC, comorbid-free ODD < HC, comorbid-free OCD = HC, ADHD < HC, within comorbid ADHD/ODD/OCD: ADHD < HC. | | Nezafati, Temmar, and Keilholz (2020) | 100 | 22-36 | 0.72 | 1200, ~400 | rest, task | SE | = 3, = 0.6 | Atlas of SE at rest and task. SE task < Rest. | | Moses O. Sokunbi et al. (2015) | 86 | 19-85 | 2 | 133 | rest | SE | = 2, = 0.3 | No effect of age or sex. | | Z. Li et al. (2016) | Chronic smoking 68, HC 66 | 19-58, 21-51 | 2 | 150 | rest | SE | = 3, = 0.6 | Chronic smoking: SE in right limbic area and frontal region. | | Chi et al. (2025) | 1087 | 6-30 | varied | varied | rest | SE | = 1, = 0.15 | n.s. diff. between ASD and HC. | | Kielar et al. (2016) | Stroke 19, OA 19, YA 20 | 65 2, 66 2, 25 1 | 2 | 180 | rest | SE | = 2, = 0.2 | YA < OA. n.s. diff. between stroke patients and HC. n.s. corr. /w hypoperfusion in perilesional tissue. | | H. Liu et al. (2023) | Bipolar-II 19, HC 17 | 15 2, 14 2 | 2 | ? | rest | SE | = 3, = 0.6 | Bipolar-II: SE in parahippocampal gyrus and inferior occipital gyrus. | | De Carvalho Santos et al. (2025) | MCI 44, HC 40 | 75 8, 77 7 | 2.2 | 164 | rest | SE | = 3, r = 0.6 | MCI: SE in left middle temporal gyrus. | | D. Song et al. (2024) | Depression 46 (14 treatment), HC 20 | 33 9, 30 8 | 2.5 | 100 | rest | SE | = 3, r = 0.6 | Depression: SE in left DLPFC and limbic system; increase can be reversed through treatment. | | Xue and Guo (2018) | AD 26, HC 26 | 73 8, 75 6 | 3 | 140 | rest | SE | = 3, = 0.8 | AD: SE in middle temporal gyrus and precentral gyrus. Network connectivity more corr. /w SE in AD vs HC. | | S. Zhang et al. (2021) | 410 | 22-36 | 0.72 | 1200 | rest | SE | = 2, = 0.5 | Reliability (test-retest): Good. | | Festor (n.d.) | High-grade glioma 85, Low-grade glioma 76, HC 51 | 47 15 | 2 | 220-301 | rest | SE | = 1-3, = 0.7 | Glioma: SE. | | L. Lin et al. (2022) | 862 | 22-37 | 0.72 | 1200 | rest, task | SE | = 3, = 0.6 | Rest SE corr. /w magnitude of (de)activation in regions activated by task. | | D. Song and Wang (2024a) | 176 | 29 3 | varies | varies | rest, movie | SE | = 3, = 0.6 | Identified regions where neurotransmitters (5HT1a, 5HTT, D1, D2, DAT, H3, MU, NMDA, VAChT, 5HT1b) contribute to structure-function coupling (incl. visual cortex, temporal cortex, paracentral lobule, DLPFC). | | N. Wang et al. (2018) | 20 | 42-57 | 2 | 160 | rest | SE | = 2, = 0.3 | Seafarers: SE in orbital-FG and superior temporal gyrus, SE in cerebellum. | | Chang et al. (2024) | 24 | 58-77 | 2 | ~151 | rest | SE | = 3, = 0.6 | YA > OA (longitudinal). Earlier-born > Later-born (cohort effect). With age, SE decreases faster in primary and intermediate networks than in higher-order association networks. | | X.-H. Wang, Jiao, and Li (2017) | ADHD 74, HC 69 | 12 2, 12 2 | varies | varies | rest | SE | = 2, = 0.2 | SE (and phase synchronization, IQ, age, ADHD diagnosis, and head motion) can be input into a predictive model to predict inattention and impulsivity. | | D. Song, Chang, Zhang, Peng, et al. (2019) | Sham 18, rTMS 30 | 23 3, 23 3 | 2 | 180 | rest | SE | = 3, = 0.6 | rTMS to left DLPFC: SE in MOFC/subgenial-ACC. | | Liang et al. (2020) | Stroke 23, HC 19 | 35-80 | 2 | 240 | rest | SE | = 2, = 0.3 | Stroke: SE in contralesional precentral gyrus, bilateral dorsolateral FG, bilateral SMA. | | D.-H. Song and Wang (2024) | 176 | 22-36 | 1 | 900, 921, 918, 915, 901 | rest, movie | SE | = 3, = 0.6 | Movie < Rest in sensory cortex. Movie > Rest in association cortex. Higher inter-scan reliability in movie (esp. in vmPFC and PCC) than in rest. | | Lu et al. (2024) | 41 | 23 4 | 2 | 240 | rest, task | SE | = 3, = 0.6 | Rest < Task in DLPFC, TPJ, posterior cingulate cortex,precuneus. Rest > Task in visual cortex. Rumination < Sad memory in visual cortex. Distraction < Sad memory in posterior cingulate cortex/precuneus. Distraction < Rumination in posterior cingulate cortex/precuneus. | | F. Zhou, Zhuang, et al. (2016) | RRMS 34, HC 34 | 20-58, 21-58 | 2 | 240 | rest | SE | = 3, = 0.6 | Relapsing-remitting multiple sclerosis (RRMS): SE in SMA, right PFC, right angular gyrus; SE in right precentral operculum, left middle temporal gyrus, bilateral parahippocampus, brainstem, right posterior cerebellum. Disease severity and tissue damage corr. /w SE. | | Moses O. Sokunbi et al. (2013) | ADHD 17, HC 13 | 30 10, 30 8 | 3 | 100 | rest | SE | = 2, = 0.46 | ADHD: SE across whole brain and in frontal and occipital lobes. Symptoms corr. /w SE. | | Xue, Yu, et al. (2019) | Schizophrenia 43, HC 59 | 39 14, 35 11 | 2 | 150 | rest | SE | = 3, = 0.6 | Schizophrenia: SE in right middle PFC, bilateral thalamus, right hippocampus, bilateral caudate. Schizophrenia: SE in left lingual gyrus, left precuneus, right fusiform face area, right superior occipital gyrus. In left cuneus and middle occipital gyrus, symptoms corr. /w SE. In right fusiform gyrus and left insula, age of onset corr. /w SE. | | Mauro and Wang (2025) | 989 | 29 4 | 0.72 | 1200 | rest | SE | = 3, = 0.6 | SE corr. /w gray matter volume and surface area (in lateral frontal and temporal lobes, inferior parietal lobules, and precuneus), n.s. relationship /w cortical thickness. | | Moses O. Sokunbi (2014) | YA 10, OA 10, YA 43, OA 43 | 22 3, 70 9, 29 9, 59 10 | 2 | 85-128 | rest | SE | = 2, = 0.3 | YA > OA in frontal and parietal lobes. SE discriminated between YA and OA across series lengths (N = 85-128) in a small, low-variability cohort, but only in long series (N = 128) in a large, high-variability cohort. | | C. Zhao et al. (2024) | 1642 | 18-65 | varies | varies | rest | SE | = 2, = 0.2 | Females > Males (largest effect in DMN), difference associated with expression of genes that were enriched in estrogen-signaling pathway. | | Camargo, Del Mauro, and Wang (2024) | 1096 | 29 4 | 0.72 | varies | rest, task | SE | = 1, = 0.6 | Task < Rest in peripheral cortical area. Task > Rest in centric part of sensorimotor and perception networks. | | Z. Wang (2021) | 862 | 22-37 | 0.72 | 1200 | rest | SE | = 3, = 0.6 | SE corr. /w activity in default mode and executive control networks. SE corr. /w age in prefrontal executive control network and frontal-temporal-parietal DMN. Women > Men in visual cortex, motor area, some parts of precuneus. SE corr. /w years of education, fluid intelligence, and performance during working memory/language/relational tasks in DMN and executive control network. | | D. Song and Wang (2025) | 44 | 23 2 | 2.53 | 240 | rest | SE | = 3, = 0.6 | SE corr. /w progesterone in FPN and limbic network. SE corr. /w impulsivity in left DLPFC. | | Jordan et al. (2023) | 42 | 18-45 | 0.8 | 600 | rest | SE | = 3, = 0.3 | Smoking: SE. rTMS in DLPFC reduced resting SE in insula and DLPFC. | | Hull (2022) | 66 | 9-37 | ? | ? | rest | SE | ? | SE corr. /w PCA components. | | D.-H. Song et al. (2024) | cTBS 18, LF-rTMS 23 | 23 3, 26 3 | 2, 1.25 | 180, 600 | rest | SE | = 3, = 0.6 | Continuous TBS (cTBS) to left DLPFC: SE in MOFC. Low-frequency rTMS (LF-rTMS) to left DLPFC: SE in MOFC/subgenual-ACC, putamen. LF-rTMS to left TPJ: SE in right TPJ. LF-rTMS to L occipital cortex: SE in the posterior cingulate cortex. | | X. Wang et al. (2013) | Short- and long-term scans 25, multi-band EPI 22, eyes open or closed 48 | 29 9, 32 12, 22 2 | 2, 0.645, 1.4, 2.5, 2 | 195, 930, 430, 120, 180 | rest | SE | ? | Reliability (inter- and intra- scan, varying TR, eyes-open vs eyes-closed) for 10 networks: Fair to high. | | Moses O. Sokunbi et al. (2014) | Schizophrenia 13, HC 16 | 41 12, 42 12 | 2.5 | 244 | task | SE | = 2, = 0.32 | Schizophrenia: SE in frontal lobe. | | Q. Li et al. (2025) | HC 640, Schizophrenia 288, Bipolar 183 | 36 13, 36 13, 37 13 | 1.5 | 200 | rest | SE | = 1, = 0.32 | Schizophrenia and bipolar: SE in sensorimotor network, DMN, central executive network, FPN, visual network, auditory network. | | P. Liu et al. (2025) | 16 | 28 7 | 2 | 300 | rest | SE | = 3, = 0.6 | Subthreshold intermittent theta burst stimulation (iTBS) to DLPFC: SE in striatum. Suprathreshold iTBS to DLPFC: SE in striatum. | | Stobbe, Forlim, and Kühn (2024) | 35 | 28 ? | 2 | 450 | task | SE | = 2, = 0.4 | Listening to nature sounds < Listening to urban sounds in posterior cingulate gyrus, cuneus, precuneus, occipital lobe/calcarine. | | F. Zhou, Huang, et al. (2016) | CPI 29, HC 29 | 43 11, 42 12 | 2 | 240 | rest | SE | = 3, = 0.6 | Chronic primary insomnia (CPI): SE in central part of DMN, anterior regions of task-positive network, hippocampus, basal ganglia; SE in right postcentral gyrus and right temporal-occipital junction. | | Q. Li et al. (2023) | 288, 183 | 36 13, 37 13 | 1.5 | 200 | rest | SE | = 1, = 0.32 | Bipolar > Schizophrenia, largest differences in visual domain, temporal domain, somatomotor domain, high-cognitive domain. | | Fan et al. (2023) | GAD 38, HC 37 | 40 2, 28-47 | 2.4 | 217 | rest | SE | = 3, = 0.6 | Generalized anxiety disorder (GAD): SE in right middle occipital gyrus and right inferior occipital gyrus. | | M. Zhou et al. (2019) | rTLE 31, HC 33 | 28 7, 28 5 | 2 | 180 | rest | SE | = 3, = 0.6 | Right temporal lobe epilepsy (rTLE): SE in right middle temporal gyrus and inferior temporal gyrus; SE in right middle FG and left SMA. | | R. Zhang, Aksman, et al. (2025) | HC 156, HC to MCI 16, MCI 80, MCI to AD 20, AD 23 | 72 7, 76 7, 74 8, 73 8, 76 8 | 3 | 140-200 | rest | SE, MSE | = 2, = 0.3, scale factors = 1-4 | Fine-scale MSE: Faster longitudinal in HC-to-MCI than in HC (in PFC and lateral occipital cortex). Coarse-scale MSE: Faster longitudinal in AD than in HC (in various frontal and temporal regions). | | A. C. Yang et al. (2018) | 354 | 21-89 | 2.5 | 200 | rest | SE, MSE | = 1, = 0.35; = 2, = 0.5; = 3, = 0.7 | Age corr. /w mean MSE. | | Guan et al. (2023) | Small cohort 10, Large cohort 272 | 24-34, 21-50 | 2.2, | ~800, 300? | rest | SE, MSE | = 2, = 0.25, scales = 1-10 | ADHD/Bipolar/Schizophrenia diff. from HC across regions and atlases (mixed directions) for SE and some scales of MSE. | | Wehrheim et al. (2024) | 330 | 22-36 | varies | varies | rest, task | SE, MSE | = 2, = 0.2; = 3, = 0.2, scales = 1-varies | Reliability (split-half, test-retest correlations): Moderate to good. Dependence on scan length: Low. | | Grandy et al. (2016) | 20 | 20-30 | 0.645 | 900 | rest | SE, MSE | = 2, = 0.5, scales = 1-10 | MSE can be accurately estimated across discontinuous segments. Dependence on scan length: Low. | | Jann et al. (2025) | 147 | 73 8 | 3 | 197 | rest | SE, MSE | = 2, = 0.5, scales = 1-6 | Classifier (HC vs AD) performance was similar for SE, mean MSE, and tau-PET. Salience network regions were most important for SE; dorsal attention regions were most important for MSE. | | Su et al. (2022) | Parkinson’s with depression 28, Parkinson’s without depression 25 | 64 8, 64 9 | 0.75 | ~495 | rest | MSE | = 1, = 0.35, scales = 1-10 | Depression in Parkinson’s: mean MSE in posterior cingulate gyrus, SMA, cerebellum. | | The Alzheimer’s Disease Neuroimaging Initiative et al. (2020) | 168 | 60-90 | 3 | 140 | rest | MSE | = 1, = 0.35, scales = 1-4 | Scale-1 MSE: HC > Amnestic MCI (aMCI) > AD in hippocampus, middle FG, intraparietal lobe, superior FG. Scale-4 MSE: HC < aMCI < AD in middle FG and middle occipital gyrus. Cognitive functions corr. /w fine-scale MSE, while corr. /w coarse-scale MSE. | | N. Zhang et al. (2021) | Schizophrenia 50, Bipolar 49, HC 49 | 21-50 | 2 | 152 | rest | MSE | = 2, = 0.3, scales = 1-5 | Schizophrenia and bipolar: mean MSE across whole brain and in calcarine fissure, precuneus, inferior occipital gyrus, lingual gyrus, cerebellum; mean MSE in median cingulate, thalamus, hippocampus, middle temporal gyrus, middle FG. Differences between schizophrenia and bipolar in precuneus and inferior occipital gyrus. | | Whiteside et al. (2021) | Progressive supranuclear palsy 94, HC 64 | 65 10, 70 7 | 2 | 305 | rest | MSE | = 1, = 0.35, scales = 1-3 for dataset 1; 1-4 for dataset 2 | Progressive supranuclear palsy: mean MSE in one of two datasets. MSE corr. /w the fractional occupancy component that differed between people with progressive supranuclear palsy and controls. | | Peña et al. (2022) | HC 8, MCI 9 | 74 4, 79 8 | 2 | 720 | task | MSE | = 2, = 0.2, scale = 6 | MCI: scale-6 MSE. Age: scale-6 MSE. | | Ho et al. (2017) | MDD 35, HC 22 | 68 6, 69 6 | 2 | 180 | rest | MSE | = 2, = 0.6, scales = 1-5 | MDD: scale-2 MSE in left FPN. | | A. C. Yang et al. (2013) | OA 99, YA 56 | 81 5, 28 4 | 2.5 | 200 | rest | MSE | = 1, = 0.35, scales = 1-5 | Cognitive score corr. /w MSE in 26 of 33 regions. OA (vs YA): MSE in left olfactory cortex, right posterior cingulate gyrus, right hippocampus, right parahippocampal gyrus, left superior occipital gyrus, left caudate. | | Trevino et al. (2024) | 10 | 24-34 | 2.2 | 136 | rest | MSE | = 2, = 0.5, scales = 1-30 | Atlas of MSE: MSE reproduces known network parcellations. | | Kung et al. (2022) | 44 | 25 4 | 2.5 | 3000 | sleep | MSE | = 1,  = 0.35, scales = 1-3 | Deeper sleep: fine-scaled MSE, consistent coarse-scaled MSE. | | A. C. Yang et al. (2015) | Schizophrenia 105, HC 210 | 43 9, 43 11 | 2.5 | 200 | rest | MSE | m = 1, r = 0.35, scales = 1-5 | Schizophrenia (SC): SE at all scales in inferior temporal gyrus, middle FG, superior FG, left SMA, cerebellum posterior lobe, left cerebellum anterior lobe. Regions with SC > HC at fine scales & SC < HC at coarse scales: Inferior FG, occipital, right insula, postcentral gyrus, left middle cingulum. | | Grieder et al. (2018) | HC 14, AD 15 | 68 4, 67 9 | 1.6 | 400 | rest | MSE | = 2, = 0.2, scales = 1-10 | AD: mean MSE in right hippocampus; functional connectivity corr. /w MSE at scales 1 and 2 in DMN. | | Niu et al. (2018) | HC 30, early MCI 33, late MCI 32, AD 29 | 74 6, 72 6, 73 8, 72 7 | 3 | 140 | rest | MSE | = 2, = 0.35, scales = 1-6 | Generally: HC > MCI > AD; regions with differences include thalamus, insula, lingual gyrus and inferior occipital gyrus, superior FG and olfactory cortex, supramarginal gyrus, superior temporal gyrus, middle temporal gyrus; in regions with differences, cognitive decline corr. /w MSE (varying directions). | | Amalric and Cantlon (2023) | Adults 14, Children 18 | 20 ?, 9 0.2 | 2 | 175 | task | MSE | = 2, = 0.5 | Math and grammar tasks: Children < Adults in association cortex. | | McDonough et al. (2019) | YA 20, middle-aged adults 31, OA 35 | 23 3, 54 3, 66 4 | 1.72 | 175 | rest | MSE | = 2, = 0.5, scales = 1-7 | Memory encoding task: In DMN: for all scales MSE pre-encoding = post-encoding; age and memory accuracy corr. w/ pre-post difference. | | Jann et al. (2025) | HC 88, MCI 50, AD 7 | 73 8, 72 7, 67 8 | 3, 0.72 | 197, 420 | rest | MSE | = 2, = 0.5, scales = 1-6 | Mean MSE in HC > MCI > AD across whole brain. Tau-PET and cognitive impairment corr. /w mean MSE in MTL. | | R. Zhang et al. (2024) | ADHD 63, HC 92 | 10 1, 10 1 | 0.8 | 383 | rest | MSE | = 2, = 0.3, scales = 1-15 | ADHD: mean MSE in FPN. Functional connectivity corr. /w MSE in HC, but not ADHD, in FPN and reward and motivation-related circuits. | | C. Lin et al. (2019) | Depression 35, HC 22 | 68 6, 69 6 | 2 | 180 | rest | MSE | = 2, = 0.6, scales = 1-5 | Late-life depression: n.s. diff. in mean MSE; scale-1 MSE in right posterior cingulate gyrus; varying-scale MSE in affective processing (putamen and thalamus), sensory, motor, temporal nodes; scale-2 MSE in left FPN. | | Wijesinghe et al. (2025) | 504 | 6-85 | 1.4 | ~430 | rest, task | MSE | = 2, = 0.3, scales = 1-13 | Mean MSE peaks at age 23. Mean MSE corr. /w 4 of 6 executive function tasks. | | Smith, Yan, and Wang (2014) | Long scan on YA 5, Short scan YA 8, Short scan OA 8 | 21 2, 23 2, 66 3 | 1.37, 2 | 1000, 240 | rest | MSE | = 2, = 0.3, scales = 1-10 | MSE in BOLD did not differ from simulated noise. Motion correction MSE in both gray and white matter. Increasing echo time MSE in gray matter, but not white matter. MSE with age. | | J. Zhou et al. (2018) | 53 | 72-96 | 3 | 120 | rest | MSE | = 1, = 0.35, scales = 1-5 | MSE corr. /w walking speed and dual-tasks costs. | | Yuan et al. (2022) | Diabetes+DPN 10, Diabetes without DPN 10, HC 10 | 40-80 | 2 | 240 | rest | MSE | = 2, = 0.3, scales = 1-4 | DPN < HC or diabetes without peripheral neuropathy, in basal ganglia. | | Kadota et al. (2021) | PSP 14, MSA 18 | 74 6, 69 9 | 2 | 150 | rest | MSE | = 2, = 0.3, scales = 1-4 | Progressive supranuclear palsy (PSP) < Multiple system atrophy (MSA) in PFC. MSE corr. /w cognitive function in PFC. | | McCulloch et al. (2023) | 28 | 33 8 | 2 | 300 | rest | MSE | = 2, = 0.3, scales = 1-5 | MSE corr. /w various measures of psilocybin level: at scale 1 (in 7 of 17 networks), n.s. at scales 2-4, at scale 5 (in 14 of 17 networks). | | McDonough and Nashiro (2014) | 20 | 22-35 | 0.72 | 1200 | rest | MSE | = 2, = 0.5, scales = 1-25 | Inverted-U pattern of SE across scales. MSE differs from noise (white, pink, red). MSE differs between networks (default mode, cingulo-opercular, left and right frontoparietal). Across networks, MSE corr. /w strength and extent of functional connectivity at fine scales but corr. at coarse scales. | | Hager et al. (2017) | Bipolar 125, Schizophrenia 98, Schizoaffective disorder 107, HC 156 | 36 12, 38 12, 35 13, 36 13 | 1.5 | 200 | rest | MSE | = 1, = 0.35, scales = 1-5 | Bipolar, schizophrenia, schizoaffective disorder: mean MSE. | | D. J. J. Wang et al. (2018) | 20 | ? | 0.72 | 1200 | rest | MSE | = 2, = 0.5, scales = 1-40 | MSE corr. /w functional connectivity at fine scales, corr. /w functional connectivity at coarse scales (default mode, left and right executive control, salience networks). | | F. Yang et al. (2022) | 98 | >60 | 2 | 180 | rest | MSE | = 1, = 0.5, scale = 5 | Classification of cognitive ability based on MSE in 9 regions. | | Zheng et al. (2020) | Early MCI 87, Late MCI 82, HC 176 | 73 9, 74 7, 75 9 | 3 | 140-200 | rest | MSE | = 2, = 0.3, scales = 1-6 | Early and late MCI: all-scale MSE (in left fusiform gyrus in early MCI, in rostral ACC in late MCI). | | Xiao Wang et al. (2017) | Schizophrenia 35, HC 30 | 12-18 | 2 | 240 | rest | MSE | = 1, = 0.35, scales = 1-5 | Schizophrenia: all-scale MSE in left superior parietal lobule and left cuneus; all-scale MSE in right ventral of middle FG, right superior parietal lobule, right precuneus, bilateral cingulate gyrus. | | Omidvarnia, Zalesky, et al. (2021) | 987 | 22-35 | 0.72 | 1200 | rest | MSE | = 2-10, = 0.15, 0.5, scales = 1-25 | MSE in DMN and FPNs, MSE in subcortical areas and limbic system. MSE at temporal resolution. Test-retest correlation varies across parameters. Functional brain connectivity corr. /w MSE, direction dependent on scale. Head motion < Resting-state. Intelligence corr. /w MSE. | | A. C. Yang et al. (2014) | YA 100, OA 112 | 20-39, 60-79 | 2.5 | 200 | rest | MSE | = 1, = 0.35, scales = 1-5? | In OA but not in YA: APOE ɛ4 allele carriers: mean MSE in precuneus and posterior cingulate. | | Zhen et al. (2024) | 1206 | 22-35 | 0.72 | 1200 | rest | MSE | = 2, = 0.5, scales = 1-12 | Fine-scale MSE corr. /w surface area, coarse-scale MSE corr. /w surface area (in lateral frontal and temporal lobes, inferior parietal lobules, and precuneus). Fine-scale MSE corr. /w cortical myelination, coarse-scale MSE corr. /w cortical myelination (PFC, lateral temporal lobe, precuneus, lateral parietal cortex, and cingulate cortex). | | Xiao and Jones (2025) | ASD 179, HC 218 | 16 7, 16 6 | 2 | 150-300 | rest | MSE | = 2, = 0.6, scales = 1-5 | ASD: In posterior midline regions, fine-scale MSE, coarse-scale MSE; in prefrontal regions, coarse-scale MSE. | | McDonough and Siegel (2018) | Primary 121, Matched replication 122, Non-matched replication 121 | 28 3, 28 4, 30 3 | 0.72 | 1200 | rest | MSE | = 2, = 0.5, scales = 1-25 | White-matter integrity corr. /w fine-scale MSE and corr. /w coarse-scale MSE. | | Omidvarnia et al. (2023) | 20000 | 40-69? | 0.735 | 490 | rest | MSE | = 2, = 0.5, scales = 1-10 | In a predictive model, MSE could predict cognitive phenotypes (fluid intelligence, processing speed, visual memory, and numerical memory), age, and gender moderately well. | | Omidvarnia et al. (2022) | 100 | 22-35 | 0.72 | 263-399 | rest, task | MSE | = 2, = 0.5, scales = 1-10 | Tasks can be classified using MSE. Task < Rest. Highest MSE in frontoparietal, dorsal attention, visual, and default mode. | | Smith et al. (2015) | HC 25, MCI 25 | 70 4, 74 5 | 2.2, 2.2 | 164, 164 | rest | MSE | = 1, = 0.3, scales = 1-4 | MCI: scale-2 MSE in R insula, R superior orbitofrontal cortex, and L inferior orbitofrontal cortex. | |

The entropy-based metrics discussed thus far — Shannon entropy, KS entropy, ApEn, and SE — are maximized with maximum randomness ([Figure 11](#fig-entropymonotonic)). That is, these metrics are formulated to increase monotonically with series randomness. Contrast this with the definition we described at the beginning, where complexity is a balance of regularity and irregularity, the “ideal” complexity metric should be maximized at an intermediate point these two extremes. Therefore, strictly speaking, despite being the most commonly used metrics, ApEn and SE do not truly capture “complexity.”

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| Figure 11: A) Entropy measures (Shannon entropy, SE, ApEn, and others; but not MSE) increase monotonically with data randomness. B) In contrast, an ideal complexity measure would represent the balance between order and disorder. Image adapted from A. C. Yang and Tsai (2013) |

How much of a limitation is this? We are reluctant to dismiss the breadth of good work that has already been completed; in our view, the conflation of complexity and irregularity is only a limitation if, at the scale under investigation, the comparison involves shifts past the midpoint from the domain of irregularity to that of regularity. Although we recognize the limitations of comparing across metrics, we observe that metrics that can describe the full irregularity–regularity range tend to regularity in the adult brain (e.g., avalanche (Xu, Feng, and Yu 2022); Hurst (Campbell, Vanderwal, and Weber 2022); both described later in this review). Therefore, the distinction between complexity and irregularity may be only semantic, at least in the healthy adult brain (but perhaps not in other populations – e.g., infants (Mella et al. 2024)). Such distinctions could account for the observed inconsistencies across clinical studies, especially across neuroimaging modalities (see Discussion). In the next section, we will discuss a method to address this limitation: multiscale sample entropy (MSE).

### Multiscale sample entropy

Is a single-scale, like the entropy metrics discussed above, sufficient to describe the brain? The brain is complex across a range of temporal scales, from very short time windows to very long ones, and understanding how these layers interact is essential to understanding the system as a whole (Beggs 2022; Buzsáki 2006; Betzel and Bassett 2017). For example, in cross-frequency coupling, high-frequency oscillations (i.e., patterns that recur at fine scales) interact with slow ones, allowing for information transmission (Canolty and Knight 2010). More broadly, fractality — or being similar across multiple scales (discussed below) — appears to be essential to brain function (Werner 2010). A better complexity metric would therefore have the ability to describe patterns that occur across multiple scales.

MSE was developed to more fully characterize the complexity of physiological signals by describing SE over multiple scales (Costa and Healey 2003; A. C. Yang et al. 2013). This is achieved by downsampling the original time series to multiple lower temporal resolutions to create “new” series across a range of lower frequencies ([Figure 12](#fig-downsampling)). The SE algorithm is then applied to each series, resulting in a unique SE value for each temporal resolution. That is, the final output consists of a vector of SE values, one for each resolution. Because the output is not a single number, it can be hard to interpret. Typically, results are presented as a plot of SE versus sampling resolution ([Figure 13](#fig-msescale)). As an attempt to summarize the output, the slope of this plot or the mean value across scales may also be reported.

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| Figure 12: An illustration of downsampling of a time series. Image recreated from Kadota et al. (2021). |

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| Figure 13: MSE profiles for simulated white, pink, and red noise; preprocessed CSF; and grey matter (resting-state networks). Figure stylized based on results from McDonough et al. (2014). Note that scale is not the same as frequency, as scale and frequency are terms from entirely different frameworks; however, were these concepts to be compared, fine scales (low values) would roughly map to high frequencies (fast-moving oscillations) and coarse scales (high ) to low frequencies (slow drifts). CSF = cerebrospinal fluid |

Unlike single-scale entropy, MSE can differentiate randomness from complexity. The shape of the scale-MSE curve is neurophysiologically meaningful ([Figure 13](#fig-msescale)). MSE for white noise is high at short scales (where there are random fluctuations) and decreases at coarser scales, as fluctuations are smoothed out [McDonough and Nashiro (2014); hoComplexityAnalysisResting2017] (however, this may be due to bias; see Kosciessa, Kloosterman, and Garrett (2020)). Fully preprocessed cerebrospinal fluid signal, which in theory consists of a series of uncorrelated random observations and therefore approximates white noise, has a high-then-low MSE curve (McDonough and Nashiro 2014). On the other hand, complex signals, which contain meaningful information across scales, have approximately horizontal MSE curves. For instance, pink and brown noise — which, unlike white noise, contain autocorrelation (i.e., future values are influenced by past ones) — both have characteristic MSE curves ([Figure 13](#fig-msescale)), with pink noise, which contains more autocorrelation than brown, having the flatter curve [McDonough and Nashiro (2014); Omidvarnia, Zalesky, et al. (2021); hoComplexityAnalysisResting2017; smithMultipleTimeScale2014]. Consistent with the idea that BOLD signal from grey matter contains the more meaningful information, its MSE more closely resembles that of pink noise than that of white noise [McDonough and Nashiro (2014); Omidvarnia, Zalesky, et al. (2021); hoComplexityAnalysisResting2017; smithMultipleTimeScale2014]. See Appendix A2 for an in-depth discussion of the significance of the MSE curve in grey matter, and for a discussion of the major limitations of MSE. See Appendix A3 for detailed instructions on choosing parameters for MSE calculation, including and .

What are the limitations of MSE? MSE requires a long time series for proper computation and utilization. Series must be long enough such that the longest scale (e.g., lowest temporal resolution) produced through downsampling contains at least 80 timepoints (Moses O. Sokunbi 2014). Furthermore, for MSE to be most useful, the series must be long enough to support multiple resolutions of downsampling. Fortunately for fMRI researchers, Grandy et al. (2016) showed that MSE can be effectively estimated across discontinuous segments (i.e., multiple runs of fMRI can be concatenated and the estimation will still be effective).

See [Table 1](#tbl-fmriSE) for a complete summary of fMRI-MSE studies and their findings.

### Other entropy-related measures

Beyond Shannon entropy, ApEn, SE, and MSE, a range of other entropy metrics are available ([Figure 14](#fig-otherentropymeasures)). These metrics are commonly used in other neuroimaging modalities (see Keshmiri (2020)), but are seldom employed in fMRI. [Table 2](#tbl-otherentropy) summarizes fMRI studies using these metrics.

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| Figure 14: **Historical development of several entropy-related measures used in fMRI time series.** Time proceeds down arrowheads. This is not comprehensive; for instance, RangeEn can be computed using the algorithm for ApEn as well as SE, but only SE is displayed for simplicity. Temporal complexity measures are much more extensively used in EEG/MEG than in fMRI; accordingly, the entropy measures identified in our search represent a small subspace of what is possible. Also, note that nearly all these measures have multiscale versions — e.g., MSE, multiscale permutation entropy, and multiscale fuzzy entropy — are not displayed |

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| Table 2: fMRI studies that use permutation entropy (PE), fuzzy entropy (FuzzyEn), permutation fuzzy entropy (PFE), range entropy (RangeEn), dispersion entropy (DispEn), differential entropy (DiffEn), and Lempel-Ziv complexity (LZC). Studies that use several metrics are not in this table; instead, they are in **?@tbl-comparisons**. HC means healthy controls.   | Reference | Sample size | Age (mean sd or min - max; years) | TR (s) | Volumes | Rest/task | Method | Results | | --- | --- | --- | --- | --- | --- | --- | --- | | Niu et al. (2022) | 319 | 6-85 | 0.645 | ? | rest | PE | Inverted-U relationship between age and PE | | B. Wang et al. (2017) | HC 30, early MCI 33, late MCI 32, AD 29 | 74 6, 72 6, 73 8, 72 7 | 3 | ? | rest | PE | Low PE was associated with Alzheimer’s, decreased cognitive function scores, and reduced grey matter volume | | Ji et al. (2025) | HC 63, Bipolar 48, Schizophrenia 47, ADHD 40 | 32 9, 35 9, 36 9, 32 10 | 2 | 152 | rest | PE | PE can predict risky behaviour in these groups (but was outperformed by measures related to the entropy of FC) | | Wohlschläger et al. (2018) | 97 | 18-30 | 2.8 | 134 | rest | Multiscale PE | ROI x group (depression vs HC) interaction | | L. Liu et al. (2024) | Schizophrenia 44, HC 30 | 28 10, 28 8 | 2 | 190 | rest | wPE | wPE is reduced in schizophrenia | | Xiang et al. (2021) | Bipolar 49, HC 49 | 35 1, 32 1 | 2 | 152 | rest | PFE | PFE is altered in bipolar (regional increases or decreases) | | Velioglu et al. (2024) | Smokers 11, Non-smokers 13 | 28 7, 29 8 | 3 | 255 | rest | DispEn | DispEn is lower in non-smokers than in smokers | | L. Liu et al. (2024) | 998 | 22-35 | 0.72 | 1200 | rest | DispEn | DispEn can predict cognitive ability and is related to brain anatomy features | | Çatal et al. (2022) | 50, 130 | 18-50 | 2, 2 | 273, varies | task | LZC | LZC decreases during all of seven tasks | | Golesorkhi et al. (2022) | 1200 | 22-35 | 1 | varies | task | LZC | LZC decreases during two tasks | | Mediano et al. (2021) | 650 | 18-88 | varies | varies | task | LZC | LZC increases during task | |

#### Permutation entropy

Permutation entropy (PE) considers only the order of amplitude values, not absolute amplitudes (Bandt and Pompe 2002). PE calculation uses a sliding window to slice the series into overlapping segments called “embedded vectors.” Each embedded vector is matched to a motif (called a “permutation pattern” or an “ordinal pattern”), which represents the relative order of the values in the vector. PE is the Shannon entropy of the relative frequencies of the ordinal patterns. That is, PE is closely related to Shannon entropy, but considers the order of values. Compared to ApEn and SE, PE is simpler to compute, makes fewer assumptions, and is more robust in the presence of noise (Zanin et al. 2012).

There are several variants of PE. PE can be computed on downsampled versions of the data (i.e., multiscale permutation entropy), resulting in a description of PE across frequencies (Wohlschläger et al. 2018). Weighted permutation entropy (wPE) was introduced by Fadlallah et al. (2013), and incorporates amplitude information into the PE calculation by multiplying each embedded vector by a weight. Unlike PE, wPE is affected by spikes and abrupt amplitude changes.

#### Fuzzy entropy

Fuzzy entropy (FuzzyEn) is identical to SE but defines similarity differently (McDonough et al. 2019). SE defines similarity using the Heaviside function; unfortunately, this function has a rigid boundary, leading to limitations including information loss and parameter-dependence. In contrast, FuzzyEn defines similarity using a fuzzy function and is thus an improved measure of complexity.

#### Permutation fuzzy entropy

Permutation fuzzy approximate entropy (PFE) was introduced by Niu et al. (2020). It is calculated by first performing permutations on the original time series — which reduces the impact of noise — then computing FuzzyEn. Note that, despite its name, PFE is not directly related to PE.

#### Range entropy

Range entropy (RangeEn) was introduced to address a limitation of SE and ApEn that is pertinent in EEG, which is that these metrics are not robust to variations in signal amplitude (Omidvarnia et al. 2018). Compared to SE and ApEn, RangeEn is also less affected by variations in signal length, which makes it an option for short-length fMRI time series (Omidvarnia et al. 2018, 2023). There are two versions of RangeEn: RangeEnA is an improvement to ApEn, and RangeEnB is an improvement to SE (Omidvarnia et al. 2018).

#### Dispersion entropy

Dispersion entropy (DispEn) originates from SE and PE and corrects a few problems with these respective techniques — namely, that SE is slow to calculate, and that PE does not thoroughly describe changes in amplitude (Rostaghi and Azami 2016). DispEn is faster to compute than both SE and PE and a better descriptor of frequency and amplitude changes than PE (Rostaghi and Azami 2016).

#### Differential entropy

Differential entropy (DiffEn) describes the spread of the probability density function for a random variable (Cover and Thomas 2005). It has an interesting history; Claude Shannon thought it was the analogue of discrete entropy for continuous variables, but it is not, and thus is not derived from information-theoretic first principles (Cover and Thomas 2005). Hence, it changes with simple operations to the series like scaling or shifting, and its values are not always meaningful (as they are sometimes negative) (Cover and Thomas 2005). Despite these major limitations, DiffEn has been used in EEG (e.g., Duan, Zhu, and Lu (2013)); in fMRI, it has been shown to have test-retest reliability comparable to or exceeding those of other temporal complexity metrics [X. Wang et al. (2013); guanComplexitySpontaneousBrain2023], though no other fMRI studies have used it.

## Lempel-Ziv complexity

## Figures

See [Figure 15](#fig-sierpinskitriangle)

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| Source: [Figures](https://WeberLab.github.io/A-guide-to-temporal-complexity-for-fMRI-scientists/notebooks/Figures.ipynb.html#cell-sierpinskitriangle)  Figure 15: **Ideal mathematical fractal.** The 2D Sierpinski triangle starts with a simple equilateral triangle (left), and subdivides it recursively into smaller equilateral triangles. For every iteration, each triangle (in blue) is further subdivided it into four smaller congruent equilateral triangles with the central triangle removed. The first such iteration is shown in the centre, with the fifth iteration shown on the right. |

See [Figure 16](#fig-statisticalfractal)

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| Figure 16: **A comparison of statistical and exact fractal patterns.** The two basic forms of fractals are demonstrated. Zooming in on tree branches (left), an exact self-similar element cannot be found. Zooming in on an exact fractal (right), exact replica of the whole are found. Photo by author. Branching fractal made in Python. Figure inspired by Taylor (2006) |

See [Figure 17](#fig-fourproperties)

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| Source: [Figures](https://WeberLab.github.io/A-guide-to-temporal-complexity-for-fMRI-scientists/notebooks/Figures.ipynb.html#cell-fourproperties)  Figure 17: **Main properties of a fractal time-series** A-C show a raw time-series (fractional Gaussian noise in this example) at different scales: B is the first half of A (shown as vertical dashed lines in A), while C is half of B (shown in vertical dashed lines in B). D is a power spectral density plot of A. E shows D but on a log-log plot, demonstrating the linear nature of fractal signals when plotted on a log-log scale. The slope of E is . In this example, is calculated to be 0.6, which translates to an H of 0.8. F shows a modified version of E, which imagines that E only demonstrates a power law scaling relationship between two distinct frequencies. The equation for calculating the scaling range in decades is shown. Exact fractal time-series (A) was created using the Davies-Harte method. |

See [Figure 18](#fig-typicalsamplepaths)

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| Source: [Figures](https://WeberLab.github.io/A-guide-to-temporal-complexity-for-fMRI-scientists/notebooks/Figures.ipynb.html#cell-typicalsamplepaths)  Figure 18: **Simulated fractional Gaussian noise and fractional Brownian motion.** Raw simulated time-series with 1,024 time-points and known Hurst values are plotted on the left. The top three time-series are fractional Gaussian noise, while the bottom three are fractional Brownian motion. H values are displayed on the left, while values are displayed on the right. Note how fractional Gaussian noise remain centered around a mean (i.e. stationary), while fractional Brownian motion wanders away from the mean (i.e. non-stationary). Log-log power spectral density plots of the signals on the left are shown on the right. Linear-regression fits are shown in red, which are used to calculate and H using the appropriate equation (on the right). Exact fractal time-series were created using the Davies-Harte method. Figure inspired by Eke et al. (2000). |

See [Table 3](#tbl-fmrihurst)

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| Table 3: **fMRI-Hurst studies.** An attempt to gather all published fMRI studies that have used Hurst or Hurst-like analysis, some stats, and the main findings. Main findings are almost certainly more nuanced than how we have reported them here; we have attempted to condense the findings as succinctly as possible. n = number of subjects in the study; TR = repition time; MLWD = maximum likelihood wavelet; PSDWelch = power spectral density Welch method; DMN = default mode network; DFA = detrended fluctuation analysis; DA = dispersional analysis; SWV = scaled window variance; RS = rescaled range; LW = local Whittle;   | Study | n | Age range | Methods | Volumes | TR (s) | Results | | --- | --- | --- | --- | --- | --- | --- | | Akhrif et al. (2018) | 103 | 19-28 | AFA | task: 425, resting: 350 | 2 | impulsivity: | | Barnes, Bullmore, and Suckling (2009) | 14 | 21-29 | MLW | 2048 | 1.1 | cognitive effort: H | | Campbell, Vanderwal, and Weber (2022) | 72 | mean 29 | PSDWelch | 900 | 1 | movie-watching: H in visual, somatosensory, and dorsal attention; frontoparietal and DMN | | Churchill et al. (2015) | 97 (28 chemo; 37 radiation; 32 HC) | n/a | DFA, Wavelet | 285 | 1.5 | worry: H | | Churchill et al. (2016) | three datasets (98): 19; 49; 30 | 20-82 | DFA, PSDWelch | 300 | 2 | age, task novelty and difficulty: H | | Ciuciu, Abry, and He (2014) | 17 | 18-27 | Wavelet | 194 | 2.16 | networks | | Dona, Hall, and Noseworthy (2017) | 71 (56 ASD; 15 HC) | mean 13 | PSD, DA, SWV | 300 | 2 | ASD: H | | Dona et al. (2017) | 110 (55 mTBI; 55 HC) | mean 13 | PSD, DA, SWV | 180 | 2 | mTBI: H | | Dong et al. (2018) | 116 | 19-85 | RS | 260 | 2.5 | age: H frontal and parietal lobe; H insula, limbic, occipital, temporal lobes | | Drayne et al. (2024) | 98 | preterm | PSDWelch | 100 | 3 | preterm: H; differentiates networks | | Erbil and Deshpande (2025) | 7 | 21-28 | Wavelet | 1,000; 1,000, 3,000 | 1; 0.6; 0.2 | microstates | | Gao et al. (2018) | 110 | mean 21 | PSD, Wavelet | 232 | 2 | reappraisal scores: H | | Gao et al. (2023) | 195 (100; 95) | 18-28 | Wavelet | ? | 2 | rumination: H | | Gentili et al. (2017) | 31 | mean 25 | Wavelet | 512 | 1.64 | neuroticism: | | Gentili et al. (2015) | 36 | mean 27 | Wavelet | 450 | 2 | social anxiety: H | | He (2011) | 17 | 18-27 | DFA, PSD | 194 | 2.16 | task: H; differentiates networks; brain glucose metabolism and neurovascular coupling | | Jäger et al. (2024) | 40 (20 task; 20 no task) | 20-32 | DFA | 512 | 1.13 | motor sequence learning: H | | Lai et al. (2010) | 63 (33 ASD; 3- HC) | n/a | Wavelet | 512 | 1.3 | ASD: H | | X. Lei, Zhao, and Chen (2013) | 17 | 18-29 | Wavelet | 200 | 1.5 | extroversion: H in DMN | | Y. Lei et al. (2021) | 75 (16 HMMD; 34 IMMD; 25 HC) | mean 41 | RS | 240 | 2 | moyamoya disease: H | | Linke et al. (2024) | 83 | 1.5-5 | WML | 400 | 0.8 | age of children ASD: H in vmPFC | | Maxim et al. (2005) | 21 | n/a | LW, Wornell, MLW | 150 | 2 | AD: H | | Mella et al. (2024) | 716 | preterm | PSDWelch | 2,300 | 0.392 | preterm: H; H starts < 0.5 at preterm age ; differentiates networks | | Omidvarnia, Liégeois, et al. (2021) | 100 | 22-35 | PSD, DFA | min 250 | 0.72 | cognitive load: H; H and entropy-based complexity highly correlated; H highest in frontoparietal network and default mode network | | Rubin, Fekete, and Mujica-Parodi (2013) | 22 | ? | Many | ? | ? | HFFT and PSDWelch outperform other methods | | Moses O. Sokunbi et al. (2014) | 29 (13 SZ; 16 HC) | ? | DA, DFA | ? | ? | SZ: H | | Suckling et al. (2008) | 22 (11 old; 11 young) | 22 and 65 | MLW | 512 | 1.1 | multifractal reanalysis of (Alle Meije Wink et al. 2006) | | Tetereva et al. (2020) | 23 | mean 23.9 | DFA | 300 | 2 | fear: H then H | | Uscătescu et al. (2022) | 124 (55 TD; 30 AT; 39 SZ) | ? | Wavelet | 947? | 0.475 | ASD and SZ: H | | Varley, Craig, et al. (2020) | 33 (15 HC; 10 min conscious; 8 veg) | ? | HFD | ? | ? | Lower consciousness: H | | Von Wegner, Laufs, and Tagliazucchi (2018) | ? | ? | Wavelet, DFA | 1500 | 2.08 | multiscale variance effects produce Hurst phenomena without long-range dependence | | Warsi, Molloy, and Noseworthy (2012) | 46 (33 AD; 13 HC) | ? | PSD, RD | 2,400 | 0.25 | AD: H | | Weber, Soreni, and Noseworthy (2014) | 14 | 22-38 | Wavelet | 512 | 2 | acute alcohol intoxication: mix of and H | | Alle Meije Wink et al. (2006) | 22 (11 old; 11 young) | 22 and 65 | MLW | 512 | 1.1 | age: H in bilateral hippocampus; scopolamine: H; faster task: H | | Alle-Meije Wink et al. (2008) | 11 | mean 35 10 | Wavelet | 136 | 1.1 | latency in fame decision task: H | | Xie et al. (2024) | 70 | ? | Wavelet | 700 | 0.6 | pharmaco-resistant TLE: H | |

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