





Brain entropy, fractal dimensions and predictability: A review of complexity measures for EEG in healthy and neuropsychiatric populations

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Abstract

There has been an increasing trend towards the use of complexity analysis in quantifying neural activity measured by electroencephalography (EEG) signals. On top of revealing complex neuronal processes of the brain that may not be possible with linear approaches, EEG complexity measures have also demonstrated their potential as biomarkers of psychopathology such as depression and schizophrenia. Unfortunately, the opacity of algorithms and descriptions originating from mathematical concepts have made it difficult to understand what complexity is and how to draw consistent conclusions when applied within psychology and neuropsychiatry research. In this review, we provide an overview and entry-level explanation of existing EEG complexity measures, which can be broadly categorized as measures of predictability and regularity. We then synthesize complexity findings across different areas of psychological science, namely, in consciousness research, mood and anxiety disorders, schizophrenia, neurodevelopmental and neurodegenerative disorders, as well as changes across the lifespan, while addressing some theoretical and methodological issues underlying the discrepancies in the data. Finally,

Abbreviations: ApEn, approximate entropy; CD, correlation dimension; DFA, detrended fluctuation analysis; FD, fractal dimension; FuzzyEn, fuzzy entropy; HE, Hurst exponent; HFD, Higuchi fractal dimension; KFD, Katz's fractal dimension; KSE, Kolmogorov–Sinai entropy; L1, largest Lyapunov exponent; LE, Lyapunov exponent; LZC, Lempel–Ziv complexity; MF-DFA, multifractal detrended fluctuation analysis; MFE, multiscale fuzzy entropy; MMSE, modified multiscale entropy; MPE, multiscale permutation entropy; MSE, multiscale entropy; Multivariate MSE, multivariate multiscale entropy; R/S analysis, rescaled range analysis; SampEn, sample entropy.

Zen J. Lau and Tam Pham contributed equally to the study.

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we present important considerations when choosing and interpreting these metrics.

KEYWORDS

complexity, EEG, entropy, fractal dimension, psychopathology

1 | INTRODUCTION

Complexity science is an umbrella term encompassing the study and characterization of ‘complex’ systems—systems consisting of multiple interdependent components that operate and interact at different levels (Fernandez et al., 2013). Such complex systems typically exhibit ‘chaotic’ behaviour. Rather than referring to a state of disorder or confusion, a chaotic system is characterized by unpredictability and irregularities, often the result of a multitude of nonlinear interactions (Faure & Korn, 2001). As such, small changes in the system can lead to exponential changes (a property popularized as the ‘butterfly effect’). For instance, the Earth’s atmosphere at any point in time and space is the result of a (virtually infinite) number of variables (e.g., temperature, particle composition and cloud density) interacting together, rendering any long-term projections challenging. Nevertheless, the overarching idea of complexity science is not necessarily to establish methods for making exact predictions, but rather to provide some insights into the characterization of the long-term trajectory of a given complex system (Faure & Korn, 2001). These principles have its roots in a branch of mathematics called *chaos theory* (see Thietart & Forgues, 1995 for an overview), which has led several disciplines (e.g., environmental science, meteorology and biology) to adopt the framework of complex dynamical systems (Burggren & Monticino, 2005; Kiel & Elliott, 1996).

The application of complexity science to nonlinear systems, referred to as ‘nonlinear dynamics’, is an emerging approach gaining traction in the study of human physiology and pathology (Ehlers, 1995). The theoretical conceptualization of the human physiological system as complex is warranted given that it is made up of multiple constituent subsystems (be it anatomical components or physiological processes) that are constantly interacting between themselves at different levels (i.e., from molecules to organs), as well as with the external environment to maintain homeostasis (Faure & Korn, 2001). The underlying assumptions are that physiological systems are inherently complex (Golbeter, 1996) and that pathological states (or ‘dynamical diseases’, see Mackey & Glass, 1977) can be characterized by disrupted or abnormal dynamic processes. One of the seminal works that

contributed to this perspective was Lipsitz and Goldberger (1992), who demonstrated that aging and disease are associated with a generalized loss of physiological complexity due to functional and/or structural alteration in the underlying organization of the physiological system (see Vaillancourt & Newell, 2002 for a schematic illustration of this idea).

2 | PHYSIOLOGICAL COMPLEXITY

The applications of complexity science have been largely centred on electrophysiological signals (Goldberger et al., 1985), as they confer sufficiently high temporal resolution for sensitive characterization of the chaotic nature of biological activity (Bornas, 2017; Fernandez et al., 2013). These research efforts initially focused on quantifying physical diseases, such as using the complexity of heart rate variability (HRV, Pham et al., 2021) to predict cardiovascular complications (Acharya et al., 2015; Khandoker et al., 2009; Song et al., 2014; Tang et al., 2015). Adherents of complexity science subsequently argued for its utility in the context of neuropsychiatric diseases and mental states characterization (An der Heiden, 2006; Tretter et al., 2011). While there are mixed results regarding the direction of change in neural complexity in diseased states, diminished complexity has been typically observed in the physiological states of individuals with psychopathological illnesses (e.g., lower HRV complexity observed in depression, Leistedt et al., 2011; Schulz et al., 2010; schizophrenia, Bar et al., 2007; and bipolar disorders, Henry et al., 2010), as well as in their associated symptoms (e.g., greater predictability in cognitive tasks, Paulus et al., 1996; mood fluctuations, Gottschalk et al., 1995; and psychotic symptoms, Paulus & Braff, 2003).

Propelled by mathematical innovations facilitating complexity analyses for multichannel signals (i.e., magneto/electroencephalography [M/EEG]), the evidence of ‘chaotic’ patterns has been since demonstrated beyond the behavioural and peripheral levels to include the neurophysiological level. In particular, the use of EEG for understanding the brain’s complex dynamics has been growing in popularity. Indeed, there

has been an acceleration in the number of studies attempting to delineate maladaptive psychopathological mechanisms by investigating EEG complexity in various experimental conditions, from sleeping (e.g., Chouvarda et al., 2010) and anaesthetic states (e.g., Zhang et al., 2001), to tasks that involve responding to emotional stimuli (e.g., Aftanas, Lotova, Koshkarov, Popov, & Makhnev, 1997) and mental arithmetic (e.g., Mohammadi et al., 2016). Complexity measures of EEG signals may supplement, if not provide greater utility and sensitivity than conventional EEG analysis techniques (such as event-related potentials or time/frequency analysis) in detecting changes in psychopathological states (Sohn et al., 2010) and potentially expediting diagnosis of diseases (Czigler et al., 2008). However, there is currently a mismatch between the rapid growth of such applications and the presence of comprehensive reviews needed to guide psychological researchers in the use of EEG complexity measures.

Despite the potential of neurophysiological complexity analysis in studying normal and abnormal psychological states and mechanisms, several limitations hinder its development. First, the strong mathematical nature of the field often makes methodologies and findings opaque to most psychology and neuroscience researchers (Yang & Tsai, 2013). Second, although most findings in physical diseases support the idea that a loss of complexity signifies deficient (or unhealthy) states and/or systems (Mackey & Glass, 1977), this notion does not fully concur with the existing literature. Based on existing reviews of complexity findings in several neuropsychiatric conditions (de Aguiar Neto & Rosa, 2019; Fernandez et al., 2013; Sun et al., 2020), researchers have not unanimously found a decrease in brain complexity across different psychiatric populations (Yang & Tsai, 2013). Hence, the aim of this paper is twofold: (1) to provide an entry-level explanation of the complexity measures and (2) to synthesize findings on EEG complexity across different areas of psychology and neuropsychiatry, highlighting methodological issues that may contribute to the existing discrepancies in the literature. Rather than providing an exhaustive list of complexity metrics (an endless endeavour given that new indices are developed on a regular basis), the focus of this paper is to summarize and provide a framework for understanding the main concepts and findings of EEG complexity to foster future advancements in this area.

3 | QUANTIFYING COMPLEXITY

Complexity metrics can be conceptualized as reflecting two aspects of the dynamics of a system, namely,

(1) predictability and (2) regularity. Though these two terms have often been used interchangeably in the literature, predictability and regularity are, at the coarse-grain level, two distinct characteristics of a chaotic system. Predictability of a dynamical system describes the temporal evolution of the system states whereas regularity describes the general amount of repetitions of patterns in the system's trajectory. Predictability can be thought of focusing on the process underlying the chaotic system while regularity corresponds to the product of that process. While we discuss the complexity metrics according to these two categories to facilitate conceptual understanding, readers should keep in mind that the two concepts remain strongly related in practice.

3.1 | Predictability

Methods that primarily capture the predictability of a dynamical system focus on evaluating the correlation present in the temporal evolution of its associated time series. Based on their algorithms, these methods can be further categorized into two subgroups (see Figure 1): (A) *Spatial dimensionality* requires a reconstruction of the 'state space' (see below) of the time series before estimating its predictability, and (B) *temporal dimensionality* characterizes the predictability of a dynamical system directly from the signal time series. In the context of EEG signals, these methods allow for the extraction of information underlying their cortical dynamics, where lower complexity of these dynamics (i.e., more predictable) may be driven by factors like a loss of neural connectivity and less activations of neural networks in diseased states like neurodegeneration (Jeong, 2002).

3.2 | Spatial dimensionality

Complex systems can be mathematically modelled by a set of differential equations, referred to as dynamic variables. These variables can be represented in a coordinate system called the state space where each isolated point in the space (a state vector) represents a system state. As it is beyond the scope of this paper to describe the reconstruction of the state space from the time series, readers can find these details in Deyle and Sugihara (2011). Extending from the concept of state space, the phase space is another coordinate system that captures the evolution of the states over time. Each point of the phase space represents a system state at a given time instant. If the dynamical system being modelled is a real-world signal (e.g., biological signal), after a long enough time, the evolution of the dynamic variables will reach an

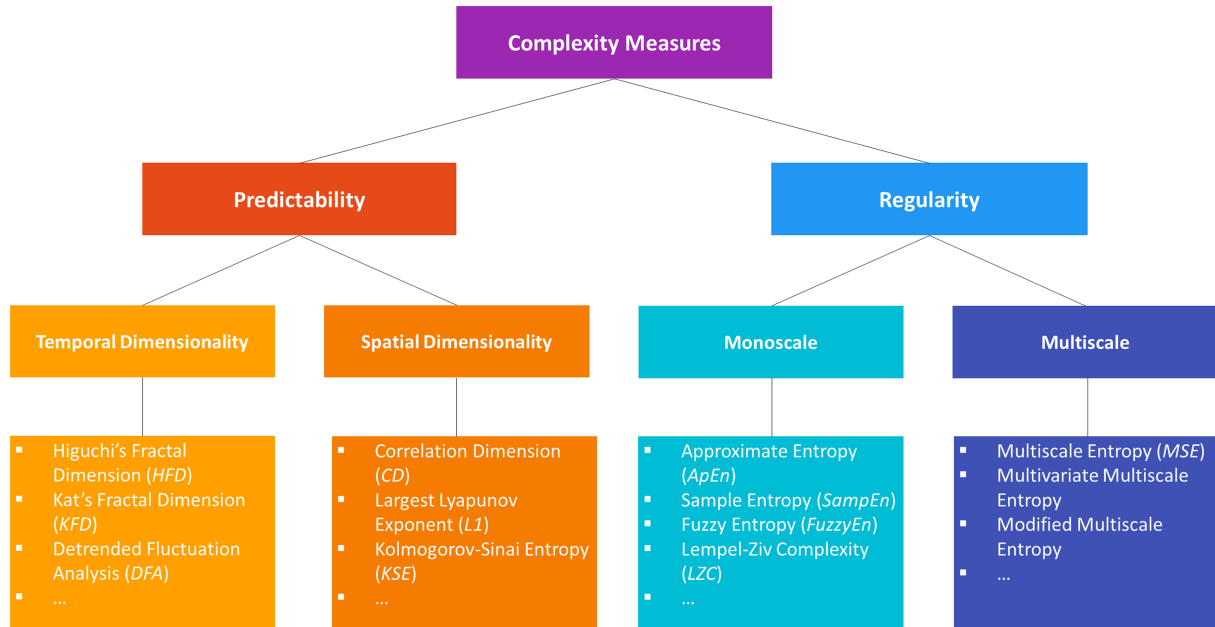


FIGURE 1 Complexity measures can be structured broadly into indices of predictability and regularity. Measures of predictability capture either spatial or temporal dimensionality of the dynamical system, and measures of regularity reflect the complexity of the system's output either on single scales or multiscales.

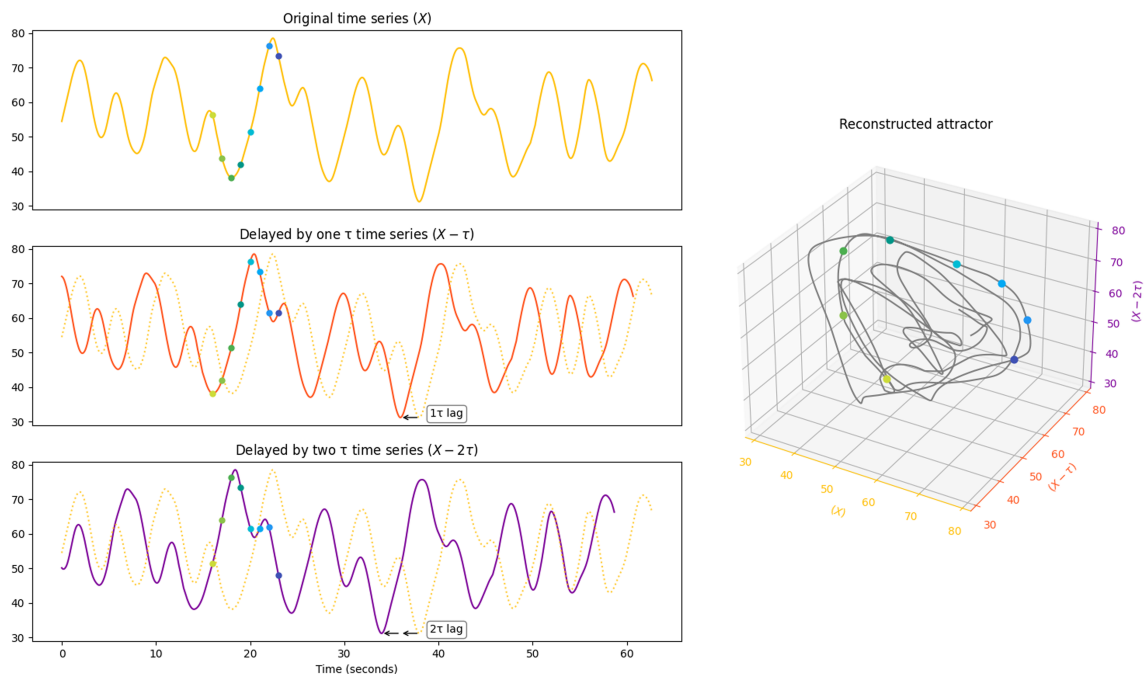


FIGURE 2 Time-delay embedding method to reconstruct an attractor in the phase space with delay (τ) = 2 and number of dimensions (m) = 3. The top left panel shows an example of a simulated signal. The middle left panel is the time series but delayed by 1τ of 2 s. the bottom left panel is the time series delayed by 2τ . the three time series are projected to a 3D space where each state vector (solid point) is plotted with the coordinates provided by the respective time series, forming an attractor (see plot on the right).

equilibrium where the dynamical system is bounded by a subset of the states with physical significance called the attractor (see Figure 2). The geometry of the attractor

constructed from a biological signal captures the unique information about the dynamic nature of the underlying physiological system, beyond what traditional signal

analysis in time and frequency domains can describe. As such, most of the spatial dimensionality indices described below actually refer to characterizations of the geometric shape of the reconstructed attractor (hence the ‘spatial dimensionality’ denomination).

One of the important geometric features of an attractor is its dimensionality (referred to as fractal dimensionality), which is different and more informative than the usual concept of (topological) dimensionality. Mathematically, fractal dimensions (FDs) define the minimum number of coordinates needed to locate any point within the phase space and can be interpreted as a measure of the structural complexity of a dynamical system. Correlation dimension (*CD*, also referred to as *D₂*) is one such FD index (Grebogi et al., 1986). By estimating the probability of any two pairs of points on the attractor separated by a distance smaller than a predefined value, *CD* sets the lower bound to the actual number of dimensions of the attractor, which in turn indicates the number of independent variables needed to model the system (Ding et al., 1993; Grebogi et al., 1986). Generally, the larger the *CD* of a biological signal, the more complex the dynamics of the underlying physiological system.

The complexity of a system can also be measured by a set of Lyapunov exponents (*LEs*) which describe the system’s predictability and stability. Mathematically, *LE* is quantified as the rate of exponential separation with time of two initially neighbouring points on the attractor (Wolf et al., 1985). Positive and negative values of *LE* correspond to whether the trajectories of the states exponentially diverge or converge over time. Visually, it characterizes the stretching and shrinking of the phase space respectively. While a system can have multiple *LEs*, the predictability of the system is mainly determined by the largest *LE* value (*L₁*). Similar to *CD*, a larger *L₁* is suggestive of a more complex signal.

Another important measure also derived from the quantification of the reconstructed attractor is the Kolmogorov–Sinai entropy (*KSE*, Sinai, 1959). Although originating from information theory (which will be discussed later), *KSE* can be defined, with respect to the spatial dimensionality perspective, as the rate at which two points that were initially close in the phase space move apart. This definition strongly aligns with that of *LE* since both measures seek to quantify the changes in volume (stretching/shrinking) of the phase space over time (Kamizawa et al., 2014). In fact, *KSE* is related to the sum of all positive *LEs*, which intuitively translates to the total amount of divergence of the temporal trajectories of the states (Pesin, 1977). Generally, a more positive *KSE* reflects a less predictable system.

3.3 | Temporal dimensionality

While the methods under the spatial dimensionality group estimate the complexity of the signals by quantifying the characteristics of the attractor, the methods under the temporal dimensionality group directly treat the time series itself as a geometric figure.

For instance, the Higuchi’s fractal dimension (*HFD*) is another common FD index. However, unlike *CD*, which approximates the FD of a physiological system indirectly from the phase-space reconstruction, *HFD* calculates the FD directly from the time series (Higuchi, 1988) and is therefore faster and more efficient. Additionally, contrary to other indices such as *CD*, that assumes signal stationarity (a condition usually not fulfilled by neurophysiological signals), *HFD*, which does not share this assumption, provides in this context a more accurate estimation of FD (Accardo et al., 1997; Spasic et al., 2011).

Similar to *HFD*, Katz’s fractal dimension (*KFD*) also estimates FD directly from the time series. In various comparisons of their performances, *HFD* generally has a higher accuracy, while *KFD* tends to underestimate the true value of the FD of a system (Castiglioni, 2010; Esteller et al., 2001; Shi, 2018). While both methods are equally efficient for short recording lengths, *HFD* becomes increasingly more efficient than *KFD* as the length increases (Esteller et al., 2001). Nevertheless, *KFD* is shown to be less sensitive to noise and more consistent at discriminating different states of brain function or different fractal features (Esteller et al., 2001; Shi, 2018). In other words, for short recording lengths, and/or when estimating changes in FD is more important than its exact quantification, *KFD* is considered as a more desirable method as compared with *HFD*.

A phenomenon that is closely related to FD is the Hurst exponent (*HE*, Hurst, 1951; Mandelbrot & Wallis, 1969). Both FD and HE seek to approximate the temporal correlation properties of a system. However, while FD represents the structural complexity of the dynamical structure over shorter periods, HE is a measure of the complexity over longer periods (Gneiting & Schlather, 2004). The two most popular estimators of HE are the *rescaled range (R/S) analysis* (Bassingthwaight & Raymond, 1994; Hurst, 1951) and the Detrended Fluctuation Analysis (*DFA*, Peng et al., 1995). As *DFA* measures the fluctuations of the trends rather than of the signal ranges, it is naturally more sensitive to non-stationarity (i.e., local trends) than the *R/S analysis* and thus more suitable and commonly used for the analysis of biological signals (Grech & Mazur, 2004; Krivstoufek, 2010). In essence, *DFA* derives a single scaling exponent, α , that describes how signal fluctuations in short windows of

time are related to the fluctuations observed in larger windows of time. The details about its computational steps can be found in Peng et al. (1995) and Hardstone et al. (2012). In general, for a biological signal and for $.5 < \alpha < 1$, the closer the value of *DFA* exponent to 1, the more complex the dynamical system. For $\alpha > 1$, the higher the *DFA* exponent value, the less complex the dynamical system.

DFA was originally formulated as a monofractal index where a single scaling exponent is computed to characterize a time series and which assumes that the *DFA* exponent is homogeneous over the entire time series. However, mounting evidence has suggested that our biological dynamics are, in fact, multifractal (Racz et al., 2018; Sassi et al., 2009; Suckling et al., 2008; Xue & Bogdan, 2017), rendering a single scaling exponent inadequate to capture their true dynamical nature (Ihlen & Vereijken, 2010). Therefore, *DFA* has been extended under the term multifractal *DFA* (*MF-DFA*), in which multiple exponents (i.e., multifractal exponents) are computed (Ihlen, 2012; Ihlen & Vereijken, 2010; Kantelhardt et al., 2002). The degree of multifractality of a time series can be quantified with statistical metrics such as the mean or range of the exponent value (mean or max–min α) (Vergotte et al., 2018; Zorick & Mandelkern, 2013). A higher degree of multifractality is often associated with a greater number of interacting underlying networks or mechanisms (Torre et al., 2019; Vergotte et al., 2018).

3.4 | Regularity

Methods that capture the regularity of a dynamical system focus on evaluating the presence of repetitive patterns in the time series. Most of the common metrics categorized under this group belong to a family of statistics called *entropy*, which corresponds to the amount of uncertainty present in a system (Cover & Thomas, 2006). As such, the regularity indices describe the regularity of a dynamical system by approximating the amount of (un)certainly present in the inference about the trajectory of an entire system, such as that of chaotic neuronal firing. For instance, greater magnitudes of these indices assessed from EEG signals of schizophrenia patients have been taken to imply a higher level of uncertainty (and hence, lower regularity), or in more technical terms, an increase in the probability that new time series patterns are generated in the brain (Xiang et al., 2019). One of the first application of entropy into the study of complexity resulted in the formulation of *KSE* (Sinai, 1959), described above. Nevertheless, unlike other entropy indices, *KSE* is not categorized as a regularity index since its

theoretical foundation is more akin to that of the aforementioned indices in the predictability group.

While *KSE* or other spatial dimensionality measures such as *CD* and *LE* require a full reconstruction of the attractor, approximate entropy (*ApEn*) can work with a lower resolution of phase space, trading off details for robustness; the sacrifice of resolution allows *ApEn* to be applicable to time series that are noisier, or of smaller sizes—those for which *KSE* could not be reliably computed (Delgado-Bonal & Marshak, 2019; Pincus, 1995; Pincus & Goldberger, 1994). Generally, higher *ApEn* values indicate more independence between the data points, that is, a lower number of repetitive patterns (lower regularity), and thus a higher level of complexity. However, regardless of its robustness, *ApEn*'s tendency to overestimate the level of regularity in the signal (Al-Angari & Sahakian, 2007; Delgado-Bonal & Marshak, 2019; Xie et al., 2008) has led to the proposal of another regularity index, sample entropy (*SampEn*, Richman & Moorman, 2000). *SampEn* can be interpreted in a similar fashion as *ApEn*, but is formulated differently so that not only does it address the root cause of *ApEn*'s bias, it is also less affected by the length of the time series (Henriques et al., 2020).

However, as both *ApEn* and *SampEn* rely on a single constant parameter to evaluate the repetitiveness of the patterns present in the signals, both indices have been shown to be significantly sensitive to the value of the chosen parameters used to compute them (Castiglioni & Di Rienzo, 2008; Liu et al., 2010). An alternative way of measuring repetitiveness was proposed where an exponential function (i.e., fuzzy function, Zadeh, 1996) was used instead of a fixed constant, leading to the formulation of another entropy-based measure named Fuzzy entropy (*FuzzyEn*, W. Chen et al., 2009). Generally, *FuzzyEn* has been shown to outperform *SampEn* in its stability and ability to discriminate physiological systems with hypothesized differences in complexity (Simons et al., 2018; Tibdewal et al., 2017).

A common characteristic of *ApEn*, *SampEn* and *FuzzyEn* is that they infer the level of complexity from the regularity of the signal at the original time scale (single-scale based). Though relatively efficient, critics believe that this single-scale approach might prevent them from thoroughly capturing the complex nature of biological systems, as their signals hold different meanings at various spatial and temporal scales (Busa & van Emmerik, 2016; Costa et al., 2002, 2005; Yakovleva et al., 2020). Thus, to accurately estimate the multiscale spatiotemporal complexity of physiological signals, Costa et al. (2002) introduced a new entropy-based measure called multiscale entropy (*MSE*). This index is obtained from computing *SampEn* on multiple scales that are

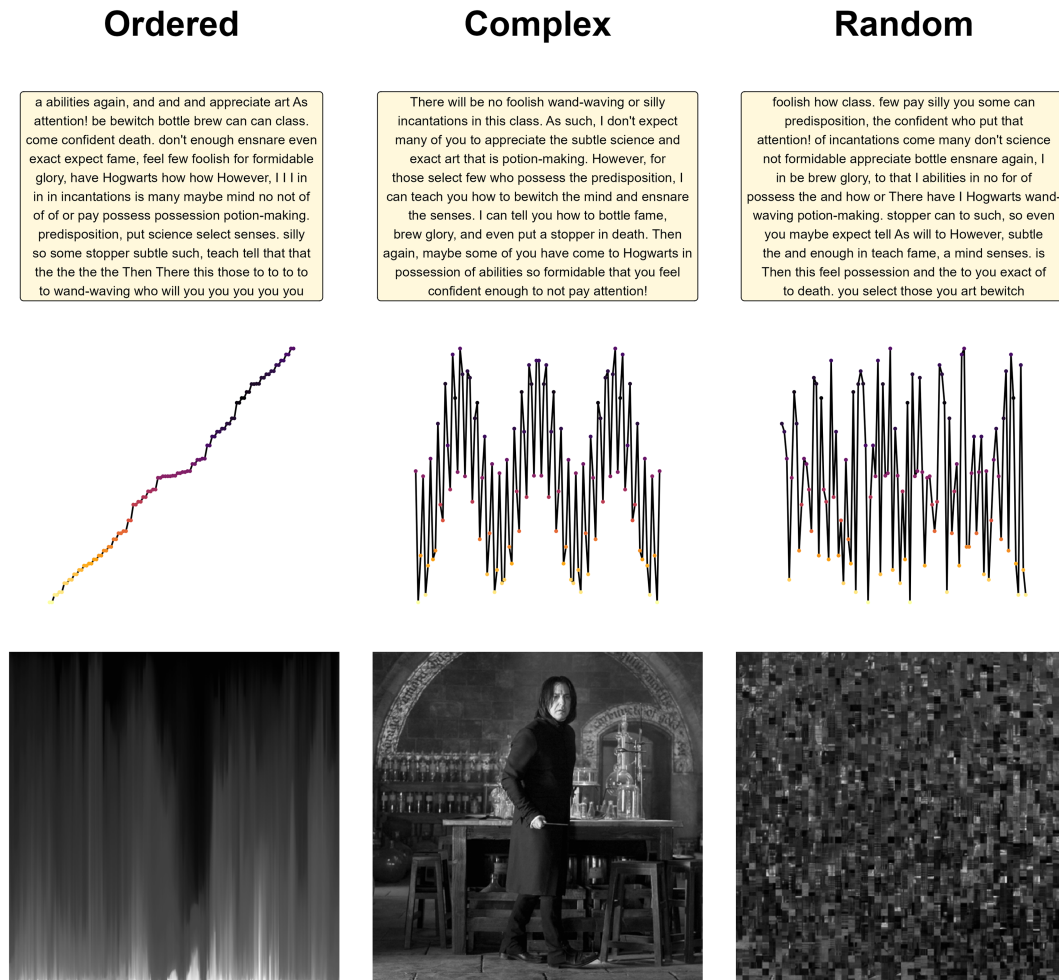


FIGURE 3 Illustrations of the difference between complexity, order and randomness, using various examples, namely, text, signal and pictorial examples. In the text example, words are arranged in alphabetical order in the *ordered* output, indiscriminately arranged in the *random* output and structured according to semantic and syntactic rules in the *complex* output. In the signal example, the *ordered* signal contains data points sorted according to amplitude, the *random* signal consists of data points sampled at random and the *complex* signal is a mixture of signals of multiple frequencies. In the pictorial example, pixels are vertically ordered by luminance in the *ordered* output, scrambled in the *random* output, and the *complex* arrangement of pixels creates a meaningful picture. The aim is to show that complex is not synonymous with random, an important conceptual distinction in complexity science.

derived from the original signal through the means of coarse graining (i.e., downsampling). Recognizing the appropriateness of the multiple-scale approach in the study of physiological signals, newer MSE-based indices have also been proposed. Some examples include the multiscale permutation entropy (*MPE*) where permutation entropy is used instead of *SampEn* (Aziz & Arif, 2005; Davalos et al., 2019), multiscale fuzzy entropy (*MFE*, Zheng et al., 2014), refined *MSE* where time scales are defined more rigorously (Valencia et al., 2009), multivariate *MSE* which is more appropriate for multichannel signals such as EEG (Ahmed & Mandic, 2011; Azami et al., 2017) and modified *MSE* (*MMSE*) where the variance, instead of the mean, is used in the coarse graining process (Costa &

Goldberger, 2015). This multiscale approach has also been incorporated into the *MF-DFA* algorithm (Gieraltowski et al., 2012) to improve its validity. Even though new multiscale-based indices are continually introduced, each attempting to address limitations of previous indices, their relative performance has not yet been formally investigated.

Another regularity index that is popular in the examination of biological dynamical systems is the Lempel–Ziv complexity (*LZC*, Lempel & Ziv, 1976). Unlike the other indices under this category, *LZC* is not based on entropy but originates from a family of techniques that maps time series into sequences of symbolic representations. Simply put, the regularity of the signal is determined through scanning the symbolic sequences for new patterns,

increasing the complexity count every time a new sequence is detected. As compared with other complexity measures, *LZC* is relatively less computationally expensive, easy to implement and can be applied directly on biological signals without any preprocessing steps (Aiordachioaie & Popescu, 2020; Zhang et al., 2001). Therefore, even though the performance of *LZC* relative to other indices remains indeterminate (Aiordachioaie & Popescu, 2020; Fathillah et al., 2017; Ibanez-Molina et al., 2015), it has been applied extensively in various fields. The next section summarizes and discusses the quantification of brain dynamical complexity in the fields of psychology and neuroscience.

4 | BRAIN COMPLEXITY IN PSYCHOLOGY AND NEUROSCIENCE

One important distinction when it comes to the application of complexity analysis to biological systems is the distinction between complexity and randomness (see Figure 3). While both complex and random systems can generate ‘seemingly’ unpredictable and irregular outputs (indexed by different complexity measures), Grassberger (2012) and other researchers emphasized the importance of the *meaningfulness* that underlies a complex system, which is absent from a random system (Goldberger et al., 2002; Yang & Tsai, 2013). However, given our current understanding of the physical meaning of different complexity measures, the distinction between pure measures of randomness and meaningful measures of complexity remains fuzzy at best, especially in the context of physiological systems. This may partly explain the

contradictory findings in the literature, as higher values of complexity indices could indicate either a more complex system, or a more random system (de la Torre-Luque et al., 2016).

4.1 | Schizophrenia

Of all psychopathological disorders, schizophrenia has been the most widely studied in terms of brain complexity (Yang & Tsai, 2013)—unsurprisingly—as the wide range of positive and negative symptoms resulting in multifaceted manifestations makes schizophrenia an ideal candidate for complexity investigations (Yang & Tsai, 2013). Researchers have expected altered brain dynamics in schizophrenia as a consequence of abnormal fluctuations in dopamine levels (An der Heiden, 2006). Interestingly, both increases and decreases in neural complexity have been frequently reported in schizophrenia populations. Diminished EEG complexity has been observed with several measures (see Table 1). This decrease in complexity, especially in the frontal areas of the brain (Akar, Kara, Latifoglu, & Bilgic, 2015a, 2015b; Jeong, Kim, Chae, et al., 1998; Kim et al., 2000; Raghavendra et al., 2009) may be interpreted as a reduction in information processing and inflexible neural networks (Kim et al., 2000). This is consistent with previous neuroimaging findings regarding hypofrontality (i.e., abnormal underactivation of frontal regions, Gattaz et al., 1992) in schizophrenia patients, which has been linked to the severity of negative symptoms, such as blunted affect, abulia and asociality (Wolkin et al., 1992). On the other hand, there is also an equally large number

TABLE 1 List of EEG complexity measures used in schizophrenia research

Psychological state/ condition	EEG complexity	Measures
Schizophrenia	Healthy controls > schizophrenia	<i>LI</i> (Akar, Kara, Latifoglu, & Bilgic, 2015a; Kim et al., 2000; Kutepov et al., 2020; Rockstroh et al., 1997) <i>HFD</i> (Raghavendra et al., 2009; Sabeti et al., 2009) <i>KFD</i> (Akar, Kara, Latifoglu, & Bilgic, 2015b) <i>LZC</i> (Sabeti et al., 2009) <i>CD</i> (Jeong, Kim, Chae, et al., 1998; Jin et al., 2003) <i>ApEn</i> (Akar et al., 2016; Sabeti et al., 2009)
	Schizophrenia > healthy controls	<i>LI</i> (Roschke et al., 1995) <i>MSE</i> (Takahashi et al., 2010) <i>LZC</i> (Y. Li et al., 2008; Ibanez-Molina et al., 2018) <i>ApEn</i> (Thilakvathi et al., 2017) <i>HFD</i> (Raghavendra et al., 2009; Thilakvathi et al., 2017)
	Severe and acute > mild schizophrenia	<i>CD</i> (Koukkou et al., 1993)

of studies indicating increased EEG complexity in schizophrenia, demonstrated through *L1* during REM sleep (Roschke et al., 1995), *MSE* during resting state (Takahashi et al., 2010) and various other measures during different mental tasks (see Table 1).

One hypothesis to explain these contradictory results was put forth by S.-H. Lee et al. (2008), who observed that increased complexity was more typically reported in patients with a more recent onset of schizophrenia, that were also younger, pre-medicated and with more positive symptoms. This was corroborated by Raghavendra et al. (2009) who underscored symptomatology as an important factor, demonstrating that individuals with positive symptoms exhibited similar or greater *HFD* values than healthy controls, whereas those with negative symptoms had lower complexity scores. At first glance, this finding may seem contrary to the idea that healthy physiological systems are always indexed by greater complexity (Lipsitz & Goldberger, 1992). However, as mentioned above, this unidirectional view of physiology cannot be accommodated by existing measures of complexity, which can generate equally high indices from both random and complex systems (such as *LZC*, discussed in later sections). In the case of schizophrenia, extremely high values of complexity could be indicative of greater polyrhythmic, disorganized brain activity, particularly in patients with active psychosis (Fingelkurts et al., 2006), which can be interpreted as greater randomness (de la Torre-Luque et al., 2016). This thus suggests a breakdown in the functional integration of schizophrenia brains (see *disconnection syndrome hypothesis*, Friston, 2002) with cortical desynchronization manifesting as positive symptoms (e.g., hallucinations and delusions). Consistent with this, Koukkou et al. (1993) reported that schizophrenia patients who were in remission and were free from antipsychotic medication had lower *CD* values than first-episode, pre-medication acute patients. Another important aspect of schizophrenia research is investigating task-related changes in neural complexity. A few studies have made progress in this, showing that schizophrenia patients exhibit less complexity change than healthy controls in response to stimulus presentation, indicating lower reactivity during information processing in patients (*FuzzyEn*, Bachiller et al., 2015; *LZC*, Ibanez-Molina et al., 2018; *Spectral Entropy*, Xiang et al., 2019). Further research using event-related paradigms may provide further insight on the mechanisms underlying abnormal cognition in schizophrenia and their relationship with neural complexity.

Overall, while it is certain that neural complexity levels in schizophrenia differ from healthy controls, the

direction of change is modulated by several factors, such as positive symptoms that increase complexity and antipsychotic agents that attenuate complexity level.

4.2 | Mood and anxiety disorders

Relatedly, several researchers have long underscored the association between enhanced cortical dynamics, healthy cognitive functioning and emotional regulation (Aftanas, Lotova, Koshkarov, Pokrovskaja, et al., 1997; Aftanas, Lotova, Koshkarov, Popov, & Makhnev, 1997; Carlino et al., 2012; Gregson et al., 1993; Lamberts et al., 2000; Martinez-Rodrigo et al., 2019; McIntosh et al., 2008). As cognitive and emotional dysregulation are transdiagnostic markers of mood and anxiety disorders (Holtzheimer & Mayberg, 2011), one might expect lower neural complexity in these patient populations as compared with their healthy counterparts. However, as with the literature on schizophrenia, conflicting trends in mood and anxiety disorders also appear to challenge the unidirectional notion of complexity as being adaptive.

Depression is a disorder characterized by dysthymia and anhedonia, reinforced by inflexible cognitive patterns manifesting primarily as negative ruminations (Holtzheimer & Mayberg, 2011). Studies have reported overall lower values of complexity in the EEG signals of individuals with depression as compared with healthy controls (see Table 2). This may be explained by maladaptive emotion regulation (ER) styles (e.g., self-blaming and rumination, Bornas et al., 2013) and a tendency to fixate on negative emotional states (Holtzheimer & Mayberg, 2011), both of which have been associated with lower EEG complexity.

However, several other studies report an opposite trend (see Table 2) across different paradigms such as resting state (Bachmann et al., 2015), mental arithmetic tasks (Li et al., 2008) and emotional stimuli tasks (Akar, Kara, Agambayev, & Bilgic, 2015). Positive relationships between the severity of depressive symptoms and various EEG complexity indices have also been reported (see Table 2), with treatment associated with greater complexity reductions (Thomasson et al., 2000). Moreover, Mendez et al. (2012) demonstrated that antidepressant treatment reduced *LZC* (MEG signals) in unmedicated depressed patients (who had higher pre-treatment *LZC* scores than healthy controls) and that these complexity reductions were correlated with the extent of symptom remissions. With regard to this, Mohammadi and Moradi (2021) hypothesized that more severe depressive symptoms are associated with lower connectivity across different brain regions, in turn giving rise to greater randomness in brain activity. Nevertheless, the attempts

TABLE 2 List of EEG complexity measures used in mood and anxiety disorders research

Psychological state/ condition	EEG complexity	Measures
Post-traumatic stress disorder (PTSD)	Healthy controls > PTSD	<i>CD</i> (Chae et al., 2004)
Depression	Healthy controls > depression	<i>LZC</i> (Kalev et al., 2015) <i>DFA</i> (values close to .5, Bachmann et al., 2017)
	Depression > healthy controls	<i>KFD</i> (Akar, Kara, Agambayev, & Bilgic, 2015) <i>HFD</i> (Akar, Kara, Agambayev, & Bilgic, 2015; Bachmann et al., 2018; Cukic et al., 2020; Mahato & Paul, 2019) <i>DFA</i> (values close to 1, Lee et al., 2007) <i>SampEn</i> (Cukic et al., 2020) <i>LZC</i> (Akar, Kara, Agambayev, & Bilgic, 2015; Bachmann et al., 2015; Li et al., 2008)
	Severe > mild depression	<i>DFA</i> (values close to 1, Lee et al., 2007) <i>LZC</i> (Mohammadi & Moradi, 2021) <i>FuzzyEn</i> (Mohammadi & Moradi, 2021) <i>KSE</i> (Thomasson et al., 2000)
Bipolar mood disorders	BMD > healthy controls	<i>HFD</i> (Bahrami et al., 2005)
Anxiety disorders	Anxiety disorders > healthy controls	<i>CD</i> (Wang et al., 2016) <i>LI</i> (Bob et al., 2006)

to explain conflicting findings in the complexity literature do not sufficiently account for factors like symptomatology and severity, which may be critical in understanding the discrepancies.

These contradictory findings have also been observed in other mood and anxiety disorders which have received significantly less attention in the field of EEG complexity. For instance, under resting state, generalized anxiety disorder (GAD) was found to be associated with higher *CD* values (Wang et al., 2016), but post-traumatic stress disorder (PTSD) was conversely associated with globally diminished *CD* (Chae et al., 2004). A mechanistic interpretation based on different modes of information processing may help to clarify this inconsistency (Molle et al., 1999). Indeed, the dominant mode of cognitive processing in GAD patients may be more akin to mind wandering and divergent thinking (e.g., catastrophizing, Schoenberg, 2020) whereas in PTSD, where individuals are under a state of hyperarousal, and excessively focused on traumatic memories, information processing may be more rigid (Chae et al., 2004). However, it is important to note that these interpretations may be specific to the neural complexity during resting state, which entails spontaneous information processing (Bob & Svetlak, 2011), as some evidence indicates that being under active states of anxiety, such as during the processing of stressful memories, results in greater neural complexity than at rest (*LI* in panic disorder patients, Bob et al., 2006).

Given the prevalence of comorbidities and heterogeneity of mood and anxiety disorders, inconsistency in the literature may be partially due to the classification of disorders as specific diagnostic entities. One starting point may be to distinguish between episodes of the disorder (e.g., Cukic et al., 2020). For example, some preliminary evidence shows that in bipolar mood disorder, manic episodes are higher in EEG complexity than depressive episodes (Bahrami et al., 2005; Thomasson et al., 2002). Alternatively, a transdiagnostic approach may be more useful in understanding how EEG complexity relates to different psychopathological symptoms (Dalglish et al., 2020). As a complex interplay of biopsychosocial processes underlie these disorders, investigating neural complexity based on their common factors and processes (whether psychological, environmental or biological), rather than traditional diagnostic boundaries, may help us better understand the relationship between complexity and mental health difficulties. Currently, there is no existing study relating EEG complexity to transdiagnostic markers of mood and anxiety disorders. The EEG complexity literature insofar has largely focused on depression more than any other affective/anxiety disorder. The scarce results from these other disorders, on top of the different interpretations put forth by authors for conflicting findings, means that it is still premature to conclude what this means about the brain's adaptation to environmental changes in mood and anxiety disorders.

TABLE 3 List of EEG complexity measures used in neurodevelopmental and neurodegenerative disorders research

Psychological state/condition	EEG complexity	Measures
Autistic spectrum disorder (ASD)	Healthy controls > ASD	<i>MMSE</i> (Bosl et al., 2011; Catarino et al., 2011) <i>SampEn</i> , <i>FuzzyEn</i> (Kang et al., 2019)
	Mild > severe ASD	<i>MSE</i> (Hadoush et al., 2019)
Attention deficit hyperactivity disorder (ADHD)	Healthy controls > ADHD	<i>ApEn</i> (Chen et al., 2019; Khoshnoud et al., 2018; Sohn et al., 2010) <i>SampEn</i> (Chen et al., 2019)
	ADHD > healthy controls	<i>ApEn</i> (Mohammadi et al., 2016) <i>HFD</i> (Mohammadi et al., 2016) <i>LZC</i> (Zarafshan et al., 2016)
Alzheimer's disease (AD)	Healthy controls > AD	<i>L1</i> (Jeong et al., 2001; Jeong, Kim, & Han, 1998; Stam et al., 1995) <i>CD</i> (Besthorn et al., 1995; Jeong, Kim, & Han, 1998; Pritchard et al., 1991; Stam et al., 1995) <i>LZC</i> (Abasolo et al., 2006) <i>HFD</i> (Nobukawa et al., 2019; Smits et al., 2016) <i>MSE</i> (Ando et al., 2021; Mizuno et al., 2010; Park et al., 2007)
	Mild > moderate > severe AD	<i>MSE</i> (Fan et al., 2018; Yang et al., 2013)

4.3 | Neurodevelopmental and neurodegenerative disorders

There are also reasons to suspect a reduction in EEG complexity in autistic spectrum disorder (ASD), a disorder characterized by a repertoire of unvaried behaviours, stereotyped thoughts and interests. Initial evidence for this comes from investigations using *MMSE*, which was found to be reduced in adults with ASD during a mental task (Catarino et al., 2011), and in infants at high risk of developing ASD during resting-state EEG (Bosl et al., 2011) as compared with control subjects. *MSE* was also able to discriminate mild from severe ASD, with the latter displaying lower entropy levels (Hadoush et al., 2019). Recently, Kang et al. (2019) demonstrated diminished patterns of several entropy measures (see Table 3) in frontal, temporal and central regions in ASD children, pointing to possible neural underconnectivity and dysfunctional integration in these areas, which have been reported by other studies using EEG power spectrum and coherence analyses (Coben et al., 2008; Murias et al., 2007).

As for attention deficit hyperactivity disorder (ADHD), the few studies that employed complexity measures show that children with ADHD exhibit lower EEG entropy during resting relative to healthy controls (see Table 3), with some disagreements regarding which regions of the brain elicit the greatest differences (*ApEn* in frontal regions, Fernandez et al., 2009; Khoshnoud et al., 2018; Sohn et al., 2010; vs. *ApEn* and *SampEn* in occipital regions, Chen et al., 2019). Other indices like *LZC* (Fernandez et al., 2009) and *FuzzyEn* (Monge et al., 2015) are also reportedly lower in ADHD when

applied to resting-state MEG signals, but no similar results have been found with EEG yet. In light of this, proponents have suggested that a deficit in the allocation of attentional resources (i.e., poor sustained attention, Robertson et al., 1997) leads to insufficient levels of cortical activation, subsequently resulting in diminished neural complexity (Clarke et al., 2001; Sohn et al., 2010). However, findings are less clear when ADHD children are placed under conditions of various cognitive tasks. It has been reported that during auditory attentional tasks, ADHD children display lower neural complexity than healthy controls in right frontal regions, while other studies show an opposite trend of greater complexity in ADHD groups across different brain regions during visual and arithmetic processing (see Table 3). These discrepancies may be attributed to specific task requirements and the brain region of interest (such as those responsible for higher order planning vs. pure attentional deficits). Given the heterogeneity of ADHD dysfunctions, it may be important to delineate between ADHD subtypes and their common co-occurring neurodevelopmental conditions. The first attempt to do so came from a recent study by Tor et al. (2021), who found several entropy measures (including *MMSE*) to be higher in children with conduct disorder (i.e., rule breaking, violent and antisocial behaviour), though there seemed to be little differences between the ADHD-only group and the group with ADHD and conduct disorder comorbidities, a pattern that makes it difficult to draw any clear interpretations. That being said, distinguishing between different neurodevelopmental disorders (many of which have shared behavioural symptoms) with complexity measures is still in its beginning stages, and the mechanisms

underlying any potential differences in neural complexity remain unclear (H. Chen et al., 2019). In sum, although resting-state EEG complexity is commonly attenuated in ADHD children relative to healthy controls, task-based paradigms have generated mixed results and the sources of these conflicts are still unclear.

To our knowledge, comprehensive reviews on EEG complexity in dementia have not been conducted since nearly two decades ago (Jeong, 2002). Nevertheless, the literature on Alzheimer's disease (AD) is significantly less equivocal than that of depression or schizophrenia, with most studies showing that AD is typically associated with a loss of EEG complexity (see Table 3). In particular, *MSE* has been shown to be sensitive to the severity of AD symptoms (Fan et al., 2018; Yang et al., 2013), with entropy significantly declining from moderate to severe AD stages, and early-stage AD being nearly indistinguishable from healthy controls (Fan et al., 2018). These complexity decrements may suggest information processing deficits and/or diminished brain dynamics in response to external stimuli (Pritchard et al., 1991). Some possible candidate explanations also include neuronal death and a loss of functional connectivity (Abasolo et al., 2006). Although the precise mechanism underlying complexity reductions remains contentious, more insights may be obtained through comparisons with other dementia pathologies such as Parkinson's disease (associated with lower *CD*, Stam et al., 1995) and vascular dementia (associated with lower *LI*, Jeong et al., 2001). Overall, the studies reviewed here agree on the finding that AD is associated with lower levels of complexity. To corroborate this, future research could expand beyond the conventional use of *CD* and *MSE* to include other newer measures of entropy for investigating complexity in AD.

4.4 | Changes across lifespan

Interestingly, EEG complexity in healthy populations appears to follow an inverse U-shaped pattern, increasing during early years of life (*MSE* during auditory and visual tasks, Lippe et al., 2009), early and late adolescence (*MSE* in resting state, van Noordt & Willoughby, 2021; and cognitive tasks, McIntosh et al., 2014; McIntosh et al., 2008), adulthood (*HFD* in resting-state EEG, Smits et al., 2016; Zappasodi et al., 2015) and declining in old age (*HFD* in resting-state EEG, Smits et al., 2016; Zappasodi et al., 2015; *MSE* in cognitive tasks, A. McIntosh et al., 2014). Results from other brain measures also echo this inverted U-shaped trajectory, such as in the connectivity of neural circuitry responsible for cognitive and motor functioning (Bo et al., 2014). Moreover, van Noordt and Willoughby (2021) recently revealed that the

largest increases in neural complexity are between late childhood and adolescence in fronto-central regions, reflecting maturational changes towards more sophisticated cognitive-behavioural processes. The subsequent decrease in neural complexity with normal ageing could be due to a multitude of deficiencies across different levels, such as neurotransmission (Backman et al., 2006), and/or structural and functional connectivity (Grady et al., 2010). This is also in agreement with Goldberger et al.'s (2002) seminal findings demonstrating reduced fractal dynamics of HRV and gait in older adults. Although most of the evidence supports the idea of an age-related complexity loss, there are a few contradictory findings (see Table 4, inverse relationship between age and *SampEn*, Hogan et al., 2012; and *CD*, Anokhin et al., 1996) yet to be resolved. Similar to the state of the literature in patient populations, this may potentially be due to the choice of algorithm that inherently presents some interpretational ambiguity (since *CD* and *SampEn* can hardly differentiate between *randomness* and *complexity*). Additionally, some evidence indicates that complexity reductions in old age occur in regions underlying cognitive abilities rather than those responsible for ER (i.e., insula and temporal lobe); while this has been found using resting-state fMRI signals (Dong et al., 2018), this dissociation has not yet been thoroughly investigated using EEG complexity measures that could provide more refined temporal information.

TABLE 4 List of EEG complexity measures used in developmental and aging research

Psychological state/condition	EEG complexity	Measures
Early years of life	Increase with adolescence and adulthood	<i>MSE</i> (Lippe et al., 2009; McIntosh et al., 2014; McIntosh et al., 2008; van Noordt & Willoughby, 2021) <i>HFD</i> (Smits et al., 2016; Zappasodi et al., 2015)
Old age	Decrease with normal aging	<i>MSE</i> (McIntosh et al., 2014) <i>HFD</i> (Smits et al., 2016; Zappasodi et al., 2015)
	Increase with normal aging	<i>SampEn</i> (Hogan et al., 2012) <i>CD</i> (Anokhin et al., 1996)

In general, EEG complexity research conducted in healthy populations demonstrates a largely consistent trend of increasing complexity from young age to adulthood and decreasing complexity with healthy aging, suggestive of neurodevelopmental and network changes across the lifespan.

4.5 | Consciousness

Finally, one of the most popular applications of complexity science is in quantifying human awareness and consciousness levels, and in particular, on how normal waking consciousness differs from states of reduced consciousness (Carhart-Harris et al., 2014; Seth et al., 2011), with a general consensus that the latter displays lower neural complexity. Several consciousness theories converge on the idea that complex neuronal interactions occur when receiving multiple rich sensory signals in alert states (Miller, 2015) and that these brain activities become less complex and more periodic at reduced consciousness (e.g., greater sleep depth) due to the lower need for information processing (Hou et al., 2021). For instance, several studies that used EEG complexity agree that levels of complexity are higher during wake and REM (rapid eye movement) sleep, but lower during deep sleep (i.e., slow wave sleep) using various complexity measures (see Table 5). The particular exception of REM sleep has been attributed to its association with dreaming, which tends to be characterized by vivid phenomenological experiences (Mateos et al., 2018). Apart from sleep stages (for a comprehensive review, see Ma et al., 2018), there is also convincing evidence showing reduced EEG complexity in other states of relative 'loss' of consciousness (see Table 5), including anaesthetized

states (Schartner et al., 2015; Zhang et al., 2001), seizures (Kannathal et al., 2005; Krystal et al., 1996) and disorders of consciousness (i.e., vegetative and minimally conscious states, see Perturbational Complexity Index [*PCI*], Casali et al., 2013).

More recently, researchers have attempted to understand the other end of the consciousness spectrum—'expanded consciousness'—states that include REM sleep, psychedelic states and onset phases for psychosis (based on a modern perspective of neuroscience and pharmacology, see Carhart-Harris et al., 2014; Carhart-Harris, 2018), which are said to be more complex than normal waking consciousness. Some preliminary evidence demonstrates that subjects under psychedelic-induced states have elevated levels of neural complexity and report increasingly intense subjective experiences, suggesting that these states are characterized by rich conscious experiences, although these findings have mostly been derived from MRI (Atasoy et al., 2017) and MEG signals (Schartner, Carhart-Harris, et al., 2017).

As compared with findings from other areas of complexity research (e.g., schizophrenia and depression), consciousness research has been the most robust in demonstrating a consistent link, that is, that higher levels of neural complexity are associated with enhanced consciousness. This area of research has also gained significant attention in the last decade, in part because of its potential for generating insights for treating mental health disorders (Carhart-Harris, 2018).

4.6 | Clinical interventions

Apart from being studied as potential biomarkers for different pathological conditions, complexity measures may

TABLE 5 List of EEG complexity measures used in consciousness research

Psychological state/ condition	EEG complexity	Measures
Sleep stages	REM and wake states > shallow sleep stages > deep sleep	<i>LZC</i> (Li & Wang, 2010; Aamodt et al., 2021; M. M. Schartner, Pigorini, et al., 2017) <i>HFD</i> (Chouvarda et al., 2010) <i>SampEn</i> (Chouvarda et al., 2010) <i>ApEn</i> (Acharya et al., 2005; Burioka et al., 2005; He et al., 2006; Lee et al., 2013) <i>MSE</i> (Mariani et al., 2016; Shi et al., 2017) <i>CD</i> (Achermann et al., 1994; Fell et al., 1996; Kobayashi et al., 2000, 2001)
Anaesthetized states	Wake states > anaesthetized states	<i>LZC</i> (M. Schartner et al., 2015; Zhang et al., 2001)
Seizures	Wake states > seizures	<i>L1</i> (Krystal et al., 1996) <i>ApEn</i> (Kannathal et al., 2005) <i>KSE</i> (Kannathal et al., 2005)

be important in the context of promising clinical interventions. In particular, the use of psychedelics (which has recently faced a resurgence of interest in mental health) has been posited to expand consciousness, with potential benefits for the treatment of pathological conditions (based on the 'entropic brain hypothesis', Carhart-Harris, 2018). In the case of psychopathological disorders such as depression, more complex states are thought to facilitate psychological insight in terms of ideas, emotions and behaviours. This is in line with research indicating that neural complexity is positively correlated with the ability to adapt to changes in environmental demands (Kloosterman et al., 2020). Increasing brain entropy would thus be expected to lead to meaningful and long-lasting changes in perspective and behaviours (Carbonaro et al., 2018; Carhart-Harris, 2018). In addition, the treatment of consciousness disorders may also be possible by enhancing levels of consciousness with psychedelics (Carhart-Harris, 2018). In light of this literature, complexity science could provide a framework to shape and validate experimental paradigms and clinical interventions for a wide range of conditions.

5 | DISCUSSION

Taken together, researchers need to exercise caution in interpreting the direction of change in these metrics and what it means in terms of the health status of a physiological system. Given that an ideal biological system is assumed to convey a rich amount of information, both extremely ordered outputs (i.e., repetitive, stereotyped behaviours and perseveration) and random-like variability (i.e., impulsivity, irrational behaviours and confabulation) are undesirable (de la Torre-Luque et al., 2016; Yang & Tsai, 2013). With regard to the latter, increases in complexity values may be wrongly misunderstood as enhanced complexity in the system when in fact the nature of the system is random. For distinguishing true dynamical complexity from randomness, *MSE* and *DFA* exponents, which can account for the scale-invariant nature of physiological signals (i.e., similar properties at different time scales), may be more sensitive relative to other measures (Bornas, 2017). For instance, with *MSE*, randomness can be identified when high entropy at short time scales decreases at larger time scales (Ma et al., 2018). de la Torre-Luque et al. (2016) elegantly proposed that rather than considering the absolute increase or decrease in complexity, deviations from *optimum variability* (Bornas & de la Torre-Luque, 2016; Guastello, 2015; Schuldberg, 2015) may provide greater insights for clinical applications. However, the notion of an 'optimally variable' system is

still relatively unexplored in existing literature. Moving forward, the utility of complexity metrics as biomarkers of disease may be reinforced through identifying ranges of normal and abnormal complexity values (i.e., normative data) unique to each pathological condition.

Many of these complexity indices can be computed using free open-source software such as *NeuroKit2* (Makowski et al., 2021) and *antropy* (Vallat, 2021). To further aid researchers in selecting a suitable EEG complexity measure, studies have also employed machine learning algorithms to assess the predictive power of each measure at classifying different psychopathological disorders or states. These studies have been conducted in schizophrenia (Akar et al., 2016; Sabeti et al., 2009), depression (Bachmann et al., 2017; Bornas, 2017; Cai et al., 2016; de Aguiar Neto & Rosa, 2019; Hosseinifard et al., 2013), ASD (Bosl et al., 2011), AD (Fan et al., 2018), ADHD (Boroujeni et al., 2019; Ghassemi et al., 2012) and in healthy and pathological ageing (Smits et al., 2016). However, the generalizability of the findings beyond the scope of their comparison is hard to assess, and inferences about the general utility of any given index should be avoided.

5.1 | Practical considerations

Other crucial considerations when choosing a complexity metric include the length of the time series and the signal-to-noise ratio (Bravi et al., 2011). For instance, it may be difficult to achieve reliable complexity estimates with *CD* and *LI* due to their underlying assumptions of noise-free and lengthy data (for reconstructing the phase space), as these assumptions are not realistic in clinical applications. On the other hand, *LZC* may be more robust for short and noisy data (Fernandez et al., 2013). As for *HFD*, while it has been said to be more accurate for measuring FD as compared with other algorithms and is computationally simpler and faster, its values may be positively skewed by the amount of noise in data (Fernandez et al., 2013). In fact, *LZC* and *HFD* have been frequently used as measures of anaesthesia and sedation depth due to their reliability in assessing short data segments, thus making it appropriate for real-time monitoring of patient outcomes (Ferenets et al., 2006; Zhang et al., 2001). In general, it is recommended that researchers report the length of the recordings, as well as the sampling rate (both of which determine the number of data points) to facilitate reproducibility and comparisons of complexity analyses.

Regarding entropy measures, it is important to note that *ApEn* and *SampEn* (which are essentially

irregularity statistics) have been criticized to be unreflective of a system's complexity. Although *MSE* was proposed to address this specific limitation (Costa et al., 2002), it requires a substantially longer time series to be accurate. Instead, other versions of *MSE* (e.g., *MMSE*) have been proposed to work better with short-term signals (Karmakar et al., 2020), but their performances have not yet been established with physiological signals. Additionally, while entropy-based indices such as *ApEn* and *SampEn* are among the most common indices, their sensitivity to the selection of computational parameters have prompted development of more robust approaches, such as *FuzzyEn*, which uses a fuzzy function instead of a single value of parameter, as well as entropy profiling where variations of parameter values are used to capture the complete profile of entropy (Karmakar et al., 2020; Udhayakumar et al., 2017). Note, however, that entropy profiling has not yet been investigated in the context of EEG signals.

5.2 | Conclusions

With the constant introduction of new complexity estimators (Tripathy et al., 2017) and modified versions of existing ones (Bai et al., 2015), it has become increasingly difficult for researchers to choose an optimal measure specific to the psychological state of interest. It may seem convenient to use a wide range of them, but this is a practice fraught with issues such as results fishing, cherry-picking and biased interpretations. By enhancing the ease for researchers in navigating the plethora of complexity measures and clarifying their underlying theoretical meanings and their differences, we hope to facilitate new developments in the field. Apart from the inexplicit boundaries between measures of randomness and meaningful complexity, rigorous investigations that consider key factors such as experimental task demands and symptom trajectories are important for well-grounded conclusions to be made about neural complexity. Future meta-analyses of EEG complexity data could be useful in understanding the source of the discrepancies in existing literature and determine the reliability of change in neural complexity with pathology, healthy aging or consciousness states. We may also gain some clarity by investigating complexity-based approaches in relationship with other EEG analysis approaches, such as time/frequency or connectivity analysis. In summary, as witnessed by the recent rise in related publications, the complexity analysis of EEG signals offers encouraging prospects for both research and clinical endeavours to further our understanding of normal and abnormal neurophysiological functioning.

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CONFLICT OF INTEREST

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

AUTHOR CONTRIBUTIONS

D.M. conceived the study. Z.J.L. and T.P. coordinated the study and drafted the manuscript. D.M. and S.H.A.C. performed a critical review of the manuscript. All authors have read and agreed to the published version of the manuscript.

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DATA AVAILABILITY STATEMENT

No datasets were generated or analysed in the present study.

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