

Brain criticality through nonadditive entropic analysis of electroencephalograms

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On the grounds of nonadditive entropies—appropriate for complex systems—we investigate the electroencephalogram amplitudes of typical and attention-deficit hyperactivity disorder (ADHD) children. The corresponding probability distributions are q -Gaussians, i.e., $\rho(x) \propto e_q^{-\beta x^2} \equiv [1 + (q - 1)\beta x^2]^{1/(1-q)}$, where (q, β) are, respectively, the entropic index characterizing complexity and the inverse width. We show that q tends to monotonically vary with β for both typical and ADHD subjects, thus strongly suggesting a scalinglike organization of electroencephalogram amplitude statistics compatible with nearly critical or multiscale brain dynamics. Moreover, we verify that ADHD subjects have a higher complexity than the typical ones. Consistently, biomarkers for objective psychiatric diagnosis could emerge along this path.

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I. INTRODUCTION

In recent years, numerous scientific advances have provided a growing understanding of the neurobiological correlates of mental disorders. However, there are still no specific markers that can support an objective diagnosis, which currently remains based on clinical and statistical descriptive criteria [1]. Insufficient knowledge about the neurobiology of mental disorders still prevents the establishment of precision psychiatry, given the complexity of the brain [2], fundamentally manifesting a state of criticality between great order and great disorder underlying self-organizing transitions to different collective functional states [3–5].

In particular, attention-deficit hyperactivity disorder (ADHD) remains a condition whose underlying mechanisms are not fully understood and, therefore, objective diagnosis remains elusive [6,7].

The study of the complexity of the brain function and its criticality is an issue of clinical interest [2,5]; thus, complexity measures appear as a possible path to solve the problem of accurately diagnosing mental disorders. Numerous evidence links pathological states and maladaptive processes

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(such as aging and mental disorders) to changes in the complexity of the dynamics of crucial systems such as the brain [2,8–13].

A promising approach lies in the statistical analysis of neurophysiological signals such as electroencephalograms (EEGs), which encode rich information about brain activity across multiple temporal and spatial scales [14]. Beyond traditional spectral and time-domain analyses, recent efforts have focused on identifying universal statistical features capable of distinguishing typical from atypical brain dynamics [13].

Spontaneous scalp EEG has previously been shown to exhibit non-Gaussian and state-dependent amplitude fluctuations [15], while quantitative EEG measures may display fat-tailed population distributions [16]. Related forms of multiscale organization have been reported in the long-range temporal correlations of oscillatory amplitude envelopes [17] and in the aperiodic $1/f$ -like component of electrophysiological power spectra, which should be distinguished from narrowband oscillatory peaks [18]. These findings motivate the use of statistical descriptions that extend beyond Gaussian models. However, heavy-tailed distributions and scaling relations alone are not sufficient to establish brain criticality. Although neuronal-avalanche studies provide evidence compatible with critical dynamics [19,20], theoretical work has demonstrated that power laws and apparent scaling can also emerge from noncritical dynamics [21] or slowly varying external drive [22]. We therefore interpret the observed q -Gaussian distributions and q - β collapse as scalinglike features compatible with critical-like or multiscale brain dynamics, rather than as definitive evidence of a critical state.

Through statistical distributions of intervals between events in the EEG, which describe its temporal regularity, neural complexity has been studied in adults and children, both typical and with ADHD, in various functional brain states, using q -exponential functions emerging within nonextensive statistical mechanics [23–25], which, grounded on nonadditive entropies, generalize the standard Boltzmann-Gibbs theory (recovered as the $q = 1$ particular instance). In particular, the sensitivity of q -statistics in order to differentiate between typical children and those with ADHD has been shown [24].

In this work, we investigate the statistical properties of EEG signal segments from both typical individuals and subjects diagnosed with ADHD. Our approach is based on the hypothesis that brain dynamics, as reflected in EEG signals, may exhibit non-Gaussian features that are not adequately captured by standard statistical frameworks.

To address this, we analyze the probability distributions of EEG segments within the context of nonextensive statistical mechanics. In particular, we consider the q -Gaussian distribution,

$$\rho(x) = \rho_0 e_q^{-\beta x^2}, \quad (1)$$

where ρ_0 is a normalization constant, q is the entropic index, and β is a positive parameter inversely proportional to the squared width; $e_q^x \equiv [1 + (1 - q)x]_+^{1/(1-q)}$, where $[\dots]_+ = [\dots]$ if $[\dots]$ is positive and zero otherwise ($e_1^x = e^x$) [26]. At this point, let us also remind that q -Gaussians with $q \geq 1$ are analytical extensions, for real number of degrees of freedom, of the Student's t -distributions [27]. Similarly, q -Gaussians with $q \leq 1$ are analytical extensions of the r -distributions.

The present framework provides a natural generalization of the Gaussian distribution and has been successfully applied to systems exhibiting long-range correlations, memory effects, and anomalous fluctuations [28–32]. While alternative formulations like stretched exponentials or log-normal mixtures present exponential or subexponential asymptotic decays, our empirical data explicitly exhibit a scale-invariant, power-law tail decay of the form $\rho(x) \sim x^{-2/(q-1)}$ as $x \rightarrow \infty$. This structural feature justifies the q -Gaussian framework and underpins the documented q - β scaling collapse.

Within this setting, we aim to characterize the statistical behavior of EEG signals and to explore whether the parameters (q, β) can serve as meaningful descriptors for distinguishing between typical and ADHD brain activities.

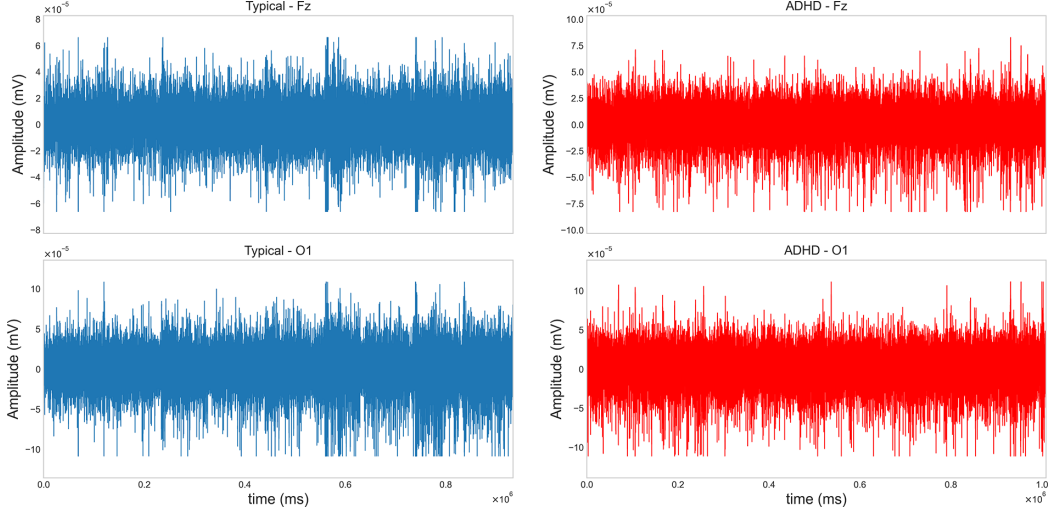


FIG. 1. Signal segments for the Fz and O1 channels for four arbitrarily chosen patients.

II. MODEL AND METHODS

We have analyzed, through a different property, the complexity of the same EEG signals recorded during attention network test from 19 typical and 19 ADHD boys (11–13 years old) from our previous studies [24,33]. The raw EEG signals were bandpass filtered between 0.5 and 100.0 Hz and corrected for power-line interference using a notch filter at 60, 120, and 180 Hz. Each channel was zero-meaned along time, and extreme amplitude values were clipped using a z -score threshold of $z = 5.0$. After this preprocessing, the signals were segmented into stationary time windows, and the amplitude fluctuations within each segment were normalized to define the variable x used in the subsequent statistical analysis. In Fig. 1, some arbitrarily chosen signal segments are depicted. The amplitude fluctuations are very distinct from Gaussian noises, showing large peaks around their mean values. In the present signals, these large-amplitude events occur intermittently. Heavy-tailed q -Gaussian statistics can originate from intrinsic mechanisms such as intermittency and multiscale organization—which are physically associated with long-range temporal or spatial correlations—as well as from nonstationary slow modulation of signal parameters. The connection between intermittency and q -Gaussian distributions has been established in coupled chaotic systems at the edge of chaos [34] and has been observed in experimental data from very different physical contexts, such as the magnetic field fluctuations in the interstellar medium [35]. On the other hand, nonstationary slow modulation of local parameters, such as variance, is naturally described by the superstatistics framework [36], which also yields q -Gaussian distributions as a result of parameter fluctuations [37]. While fitting a q -Gaussian directly characterizes amplitude statistics rather than explicitly measuring spatiotemporal correlation functions, the combined observation of q -Gaussian distributions and intermittency supports a physical interpretation in terms of multiscale, strongly correlated dynamics. A recent nonlinear-dynamical example illustrates how intermittently recurring mechanisms can generate a long-tailed q -Gaussian invariant density near the edge of chaos [38]. These peaks are well known to be very common in systems characterized by potentially strong nonlocal correlations; hence, the theoretical approach through q -statistics is *a priori* plausible.

For each preprocessed EEG segment, the empirical probability distribution of the fluctuations x was estimated via a normalized histogram, using a bin count adapted to the number of samples. The resulting probability density $\rho(x)$ was then fitted to the q -Gaussian ansatz

$$\rho(x) = \rho_0 e_q^{-\beta x^2},$$

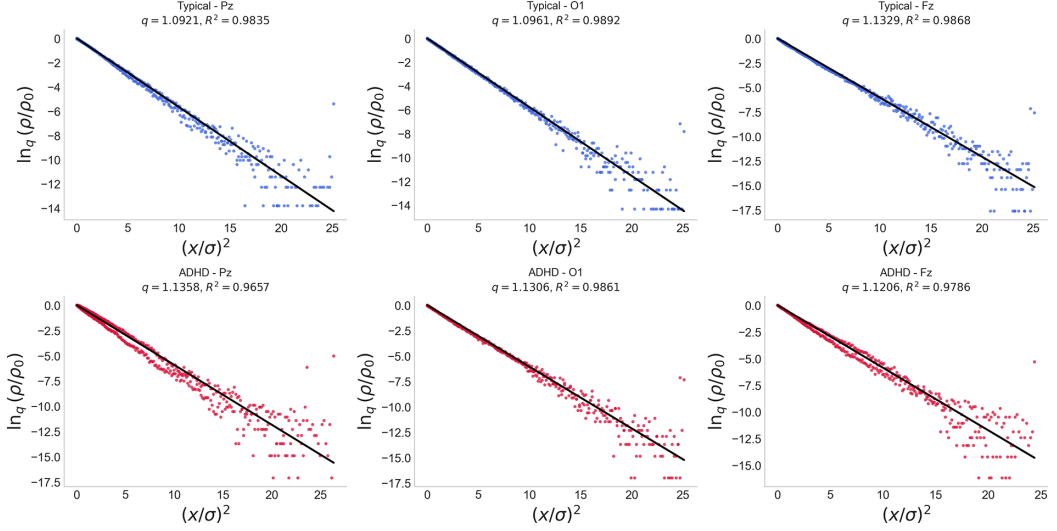


FIG. 2. Curves of $\ln_q(\rho/\rho_0)$ vs $(x/\sigma)^2$ for the Pz, O1, and Fz channels, where σ is the standard deviation used to normalize the signal. The choices of the channel and the subject were arbitrary and made only for illustration. The dimensionless variable x/σ was used only for better visualization.

by mapping the problem to a weighted linear regression. Specifically, we defined the transformed variable $y = \ln_q(\rho/\rho_0)$ and performed a weighted least-squares regression of y versus x^2 through the origin, so that the fitted slope yields $-\beta$. The corresponding entropic index q was determined by maximizing the coefficient of determination R^2 of this linear relation over a wide interval $q \in [q_{\min}, q_{\max}]$, using a combination of grid search and local refinement. The fitted parameters (q, β) were recorded for each channel and subject, enabling subsequent statistical comparison between typical and ADHD groups.

III. RESULTS

In Fig. 2, we arbitrarily illustrate some probability distributions on the scale $\ln_q(\rho/\rho_0)$ versus $(x/\sigma)^2$, which clearly exhibit linear behavior. This representation was intentionally chosen because $\ln_q(e_q^x) = x$; therefore, since $\ln_q(1) = 0$, we obtain exactly $\ln_q(\rho/\rho_0) = -\beta x^2$.

To investigate the empirical dependency between the model parameters, the scaling exponent γ was determined independently for each channel by pooling the data from both groups. Specifically, γ was optimized within the interval $[0.01, 5.0]$ to maximize the coefficient of determination (R^2) for the linear relation $q \sim c_0 + c_1/\beta^\gamma$ through a grid search approach. The numerical uncertainties of the optimized exponents were estimated from the curvature of the R^2 landscape around its maximum.

TABLE I. Mean values of q and $\beta^{-\gamma}$ over $N = N_p \times N_{\text{ch}}$, where $N_p = 19$ and $N_{\text{ch}} = 17$ represent the numbers of patients and channels, respectively. The mean values are pooled over all channels and subjects for descriptive purposes.

Group	$\langle q \rangle - 1$	σ_q	$\sigma_q/\langle q \rangle$	$\langle \beta^{-\gamma} \rangle$	$\sigma_{\beta^{-\gamma}}$	$\sigma_{\beta^{-\gamma}}/\langle \beta^{-\gamma} \rangle$	N
ADHD	0.38	0.30	0.22	0.93	0.32	0.34	323
Typical	0.23	0.15	0.12	1.08	0.13	0.12	323

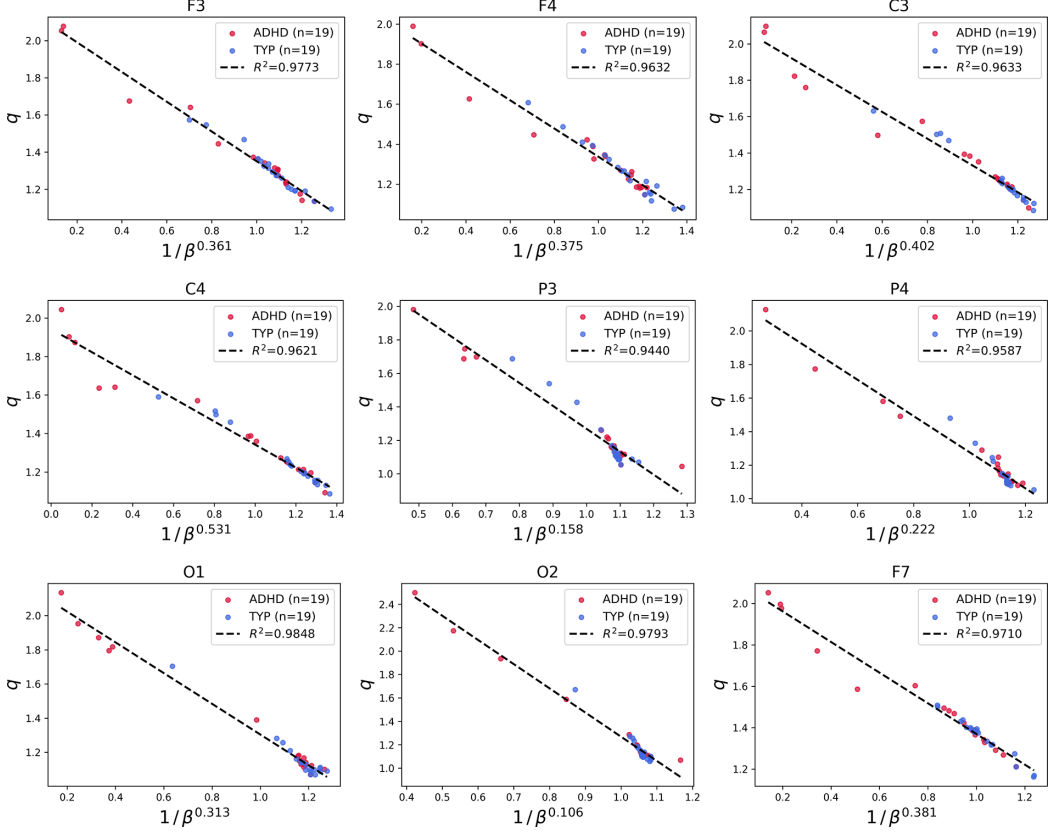


FIG. 3. Plot of q vs $1/\beta^\gamma$ for the channels F3, F4, C3, C4, P3, P4, O1, O2, and F7 for all patients. Each subfigure presents the fitting of individual channels for typical individuals (blue) and those with ADHD (red). Similar results have been obtained for the remaining channels.

The resulting group statistics from this optimization are summarized in Table I. The ADHD group exhibits a slightly higher mean q than the typical group, accompanied by a larger standard deviation and a higher relative deviation $\sigma_q/\langle q \rangle$. An identical pattern is observed for the scaling variable $\beta^{-\gamma}$, evaluated via the channel-specific exponents γ derived from the joint optimization framework.

To ensure that the observed non-Gaussian signatures are not artifacts of the preprocessing pipeline, a sensitivity analysis was performed by varying the amplitude clipping threshold across $z = 4.0, 5.0$, and 6.0 . Relaxing the threshold leads to a marginal increase in the estimated entropic index due to the inclusion of extreme tail fluctuations, with group averages shifting by at most 0.05 for typical individuals ($\langle q \rangle_{z=4} = 1.20 \pm 0.16$; $\langle q \rangle_{z=6} = 1.25 \pm 0.14$) and 0.03 for the ADHD group ($\langle q \rangle_{z=4} = 1.36 \pm 0.32$; $\langle q \rangle_{z=6} = 1.39 \pm 0.30$). The strict ordering of the group means ($\langle q \rangle_{\text{ADHD}} > \langle q \rangle_{\text{typ}}$) and the overall population dispersion are structurally preserved at every threshold, demonstrating that the qualitative group-level dynamics are highly robust to the specific choice of the z -score cutoff.

In Fig. 3, we observe that $q = q(\beta)$ varies as an affine function of $1/\beta^\gamma$, namely, $q_i(\beta) = c_{0i} + c_{1i}/\beta^{\gamma_i}$ ($i = 1, 2, \dots, 17$), where the coefficients (c_0, c_1, γ_i) differ slightly across channels. This systematic relation between q and β indicates that the shape and scale of the EEG amplitude distributions are coupled rather than independent. The parameter q controls the relative weight of large-amplitude fluctuations, while β sets the characteristic scale of the distribution. In standard

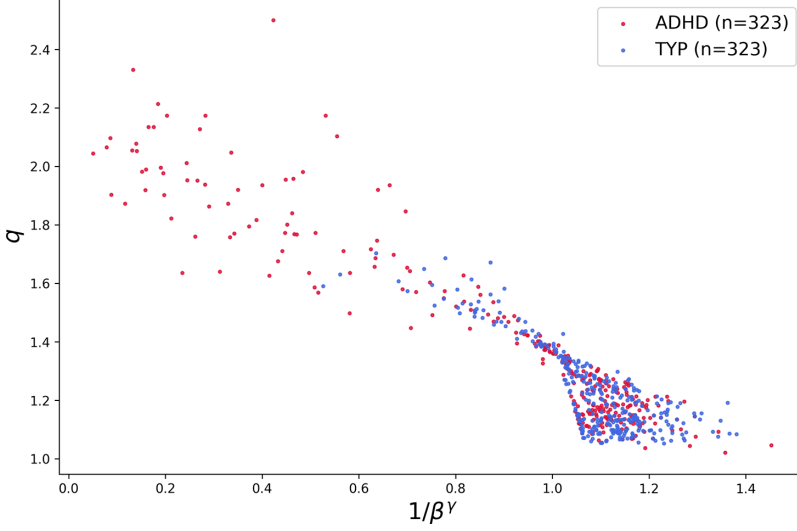


FIG. 4. Plot of q vs $1/\beta^\gamma$ for all 17 channels and all 38 patients.

Gaussian or uncorrelated processes, shape and scale are decoupled. Here, each empirical subject-channel realization is associated with a unique fitted pair (q, β) lying on an equation-of-state-like scaling manifold. An analogous situation was reported in Ref. [39], in which each observation scale selected a unique (q, β) pair and the monotonic relation was interpreted as evidence of criticality. Given that the q -Gaussian distribution decays asymptotically as $\rho \sim A x^{-2/(q-1)}$, where A is a constant, the power-law exponent can be expressed as $2/(c_{0i} - 1 + c_{1i}/\beta^\gamma)$. Notice that in the limit $\beta \rightarrow \infty$, this expression reduces to $2/(c_{0i} - 1)$. From the empirical results in Fig. 3, the asymptotic index $q_i(\infty) = c_{0i}$ lies between 1.9 and 2.5 ($1.9 < c_{0i} < 2.5$), which corresponds to an asymptotic exponent $2/[q_i(\infty) - 1]$ approximately within the range $[4/3, 2.22]$.

Despite the γ values differing across channels for both typical and ADHD individuals, the graph of q versus $\beta^{-\gamma}$ for all channels shows a clear collapse (see Fig. 4). Physically, this shape-scale coupling can be understood through two complementary mechanisms: Under multiscale dynamics, high-intensity fluctuations (small β) engage cross-scale neural synchronization, producing intermittent bursts that systematically heavy tail the distribution (higher q); alternatively, under nonstationary slow modulation, slow temporal drifts in background neural arousal or local network gain modulate the local variance, generating superstatistical mixtures where scale fluctuations directly dictate overall tail geometry. The common curve observed across typical and ADHD groups is therefore best described as a common empirical scaling relation rather than proof that the groups belong to the same universality class.

This suggests two possible explanations: (1) ADHD and typical subjects belong to the same universality class, thus exhibiting no significant differentiation, or (2) some of the 19 patients diagnosed with ADHD are in fact false positives. Explanation (1) is depicted in Figs. 3 and 4, where the criticality occurs along the same straight lines for both ADHD and typical results. This outcome suggests that they share the same exponents, and therefore belong to the same universality class. Explanation (2) can be more clearly seen in Fig. 4, where for all channels it is very difficult to differentiate the ADHD points from the typical ones. If explanation (1) were incorrect, we would expect to observe a cluster or at least some differentiation among patients, which does not occur in this case. Let us clarify that if explanation (1) is correct, then explanation (2) might also hold true. However, if explanation (2) is correct, the lack of knowledge about the diagnosis does not allow any definitive conclusion regarding explanation (1).

TABLE II. Values of γ corresponding to the channels F (frontal), C (central), O (occipital), P (parietal), and T (temporal).

Channel	$\gamma \pm \sigma_\gamma$	R^2	N_{ADHD}	N_{TYP}
F3	0.36 ± 0.009	0.98	19	19
F4	0.38 ± 0.012	0.96	19	19
C3	0.40 ± 0.013	0.96	19	19
C4	0.53 ± 0.018	0.96	19	19
P3	0.16 ± 0.006	0.94	19	19
P4	0.22 ± 0.008	0.96	19	19
O1	0.31 ± 0.006	0.98	19	19
O2	0.11 ± 0.003	0.98	19	19
F7	0.38 ± 0.011	0.97	19	19
F8	0.44 ± 0.015	0.96	19	19
T3	0.27 ± 0.009	0.96	19	19
T4	0.24 ± 0.008	0.96	19	19
T5	0.18 ± 0.008	0.93	19	19
T6	0.10 ± 0.003	0.97	19	19
Fz	0.29 ± 0.007	0.98	19	19
Cz	0.25 ± 0.009	0.96	19	19
Pz	0.21 ± 0.006	0.97	19	19

Table II presents the γ values for each channel along with their numerical uncertainties (σ_γ) obtained from the optimization landscape. Notice that the channels F3, O1, O2, and Fz have the highest R^2 values. Although F3, O1, and Fz share similar γ values, the channel O2 exhibits an exponent γ that is markedly different from those of the other channels, and it can be compared to the exponents of P3 and T6. The highest γ value is associated with the C4 channel.

In Fig. 5, we present the channel indices q for both typical and ADHD individuals. Figure 6 compares the subject-level channel averages $\langle q \rangle_{\text{ch}}$, with each participant contributing a single value. The subject-level mean is $\langle q \rangle_{\text{ch}} = 1.23$ for the 19 typical participants and 1.38 for the 19 participants with ADHD. Thus, ADHD participants tend to exhibit larger $\langle q \rangle_{\text{ch}}$ values, corresponding within the q -Gaussian framework to heavier-tailed and more heterogeneous EEG amplitude fluctuations. In terms of the entropic index q , which characterizes complexity within nonextensive statistical mechanics, this reflects a higher neural complexity. However, this tendency is not uniform across the ADHD group, as the two distributions overlap substantially; the group mean difference is strongly influenced by a subset of participants with the largest q values. This result may be consistent with stronger intermittency, dynamical heterogeneity, or multiscale modulation.

For the typical group, no strong anomalies are observed. For the ADHD group, four patients exhibit channel indices whose q values are sensibly larger than the values observed for typical patients. These results neatly exhibit that the ADHD behavior is associated with a complexity higher than the normal one.

IV. FINAL REMARKS

In this work, we have analyzed the statistical properties of EEG signal segments from both typical individuals and subjects diagnosed with ADHD within the framework of nonextensive statistical mechanics. Our results show that the distributions of EEG fluctuations are well described by q -Gaussians. Together with the intermittent large-amplitude events observed in the signals, these heavy-tailed statistics are consistent with multiscale organization and long-range correlations [36], with intermittency serving as a plausible generating mechanism [34,38]. Although nonstationary slow modulation presents an alternative route to heavy tails [36,37], the present analysis focuses

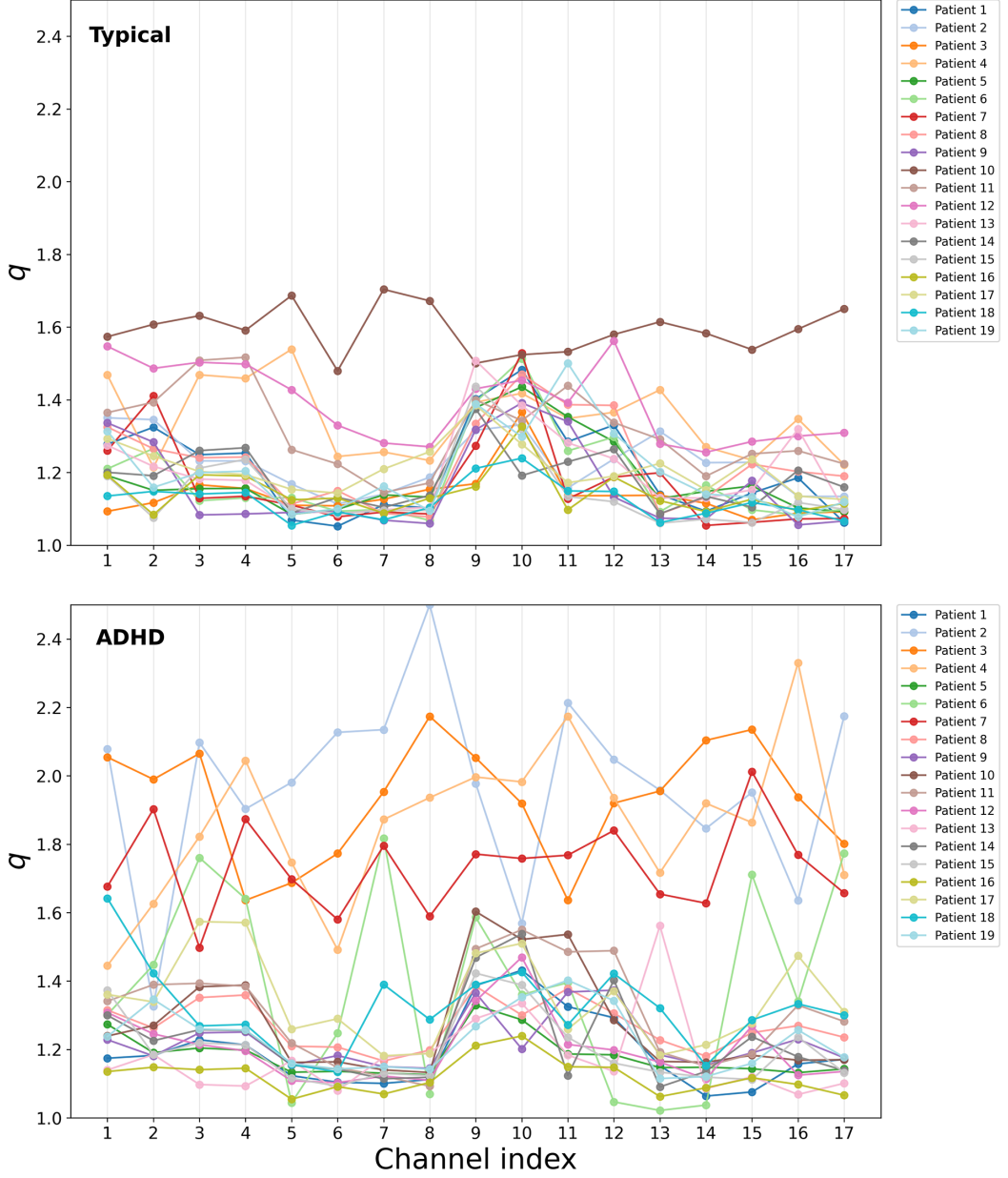


FIG. 5. Plot of q vs channel index for all 38 subjects. The channels are ordered as follows: F3, F4, C3, C4, P3, P4, O1, O2, F7, F8, T3, T4, T5, T6, Fz, Cz, Pz (17 channels in total).

on amplitude fluctuation dynamics, remaining complementary to direct spatiotemporal correlation measurements.

A central finding is that the parameters (q, β) are not independent, but instead exhibit a power-law-like relationship for both groups. This scaling behavior suggests the existence of an underlying organizing principle, compatible with the emergence of critical-like dynamics in brain activity [3,4]. Notably, although both typical and ADHD subjects follow similar functional trends, the corresponding parameter regimes differ, pointing to distinct dynamical signatures. A monotonic

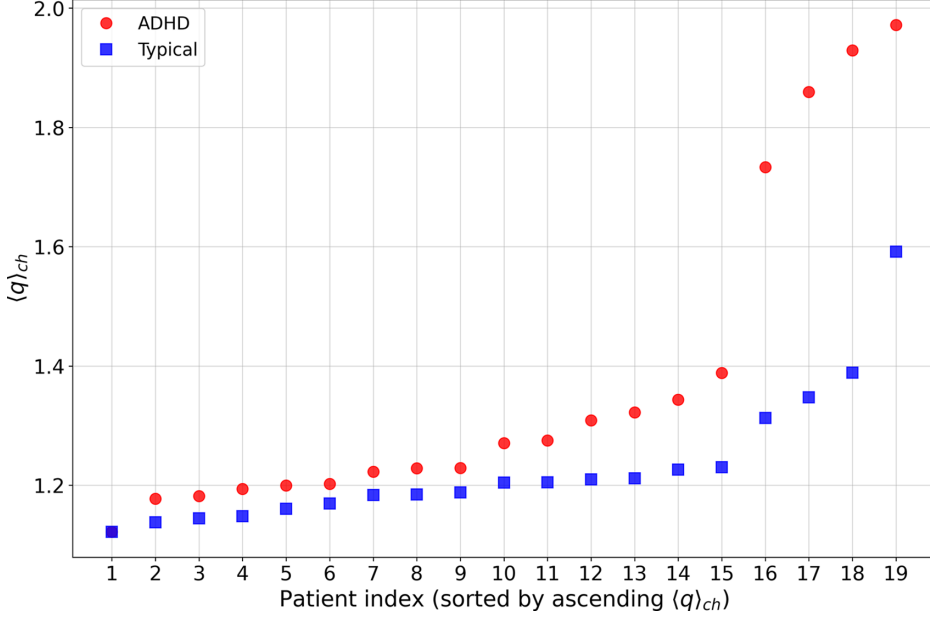


FIG. 6. Plot of $\langle q \rangle_{ch}$ (average over channels) vs patient index in ascending order. The indices of the first ADHD and first typical patients coincide.

dependence of q on β has been encountered in various complex systems [39–41], and it is in variance with that corresponding to the distribution of delays in British railways (see Fig. 5 of Ref. [42]).

These results support the idea that the q -generalized statistical approach provides a promising approach for estimating the neural complexity, from EEG signals [2,23–25]. More broadly, like other studies using different methodologies, our findings also point to neural complexity as a pathway for establishing quantitative and physically grounded markers for mental disorders. In particular, the eight individuals whose values of q distinctively are larger from their pairs (see subjects 16–19 in Fig. 6) indicate the heterogeneity of the populations classified as typical or ADHD, reflecting the variability across the diverse biological and cognitive dimensions of the subjects involved. The direct group-level observation is that ADHD participants tend to exhibit larger subject-level $\langle q \rangle_{ch}$ values than typical participants, corresponding within the present model to heavier-tailed amplitude fluctuations. Such behavior may reflect differences in intermittency, dynamical heterogeneity, or multiscale organization; however, the substantial overlap between groups and the influence of a small high- q subgroup require a cautious, exploratory interpretation.

Future work may extend this approach by exploring larger datasets of brain activity from different brain states in different human profiles clarifying through complexity measures (q -statistics) several mind disorders and neurodivergences, perhaps reaching statistical markers.

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DATA AVAILABILITY

There are no publicly available research data or software supporting this manuscript. Requests for further information or data should be sent to the authors.

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