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Aperiodic dynamics of neural signals as a biomarker in deep brain stimulation: a systematic review

Dinâmica aperiódica de sinais neurais como biomarcador na estimulação cerebral profunda: revisão sistemática

Letícia Souza Mangrich Pereira[1], Ana Luiza Souza Tavares[2], Anita Maria da Rocha Fernandes[1], Bruno Duarte Gomes[2]

ORCID: 0009-0004-2129-9192 · 0009-0006-1180-2967 · 0000-0002-2986-5353 · 0000-0002-3646-7847

[1] Universidade do Vale do Itajaí - UNIVALI, Itajaí (SC), Brasil.

[2] Universidade Federal do Pará - UFPA, Belém (PA), Brasil.

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CORRESPONDING AUTHOR
Letícia Souza Mangrich Pereira
leticiamangrich@edu.univali.br

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ABSTRACT

Objective: To investigate how aperiodic neural signal components contribute to surgical mapping and correlate with clinical assessments in patients undergoing Deep Brain Stimulation. **Method:** A Systematic Literature Review was performed following PRISMA 2020 guidelines across BVS, CAPES, IEEE Xplore, and PubMed (2021–2026) for studies on spectral parameterization, with methodological quality assessed using an adapted Newcastle-Ottawa Scale. **Results:** Out of 90 records, 25 studies were included. Aperiodic parameters provided complementary information to oscillatory metrics in anatomical and functional characterization. Changes in the exponent were associated with medication state and Deep Brain Stimulation across multiple studies, consistent with excitation/inhibition balance modulation, though correlation with clinical scales was not observed uniformly across the corpus. **Conclusion:** Aperiodic dynamics represent a promising biomarker of neural excitability in Deep Brain Stimulation, with preliminary evidence of generalization across pathologies that warrants confirmation through multicenter, longitudinal studies.

Keywords: Electrophysiology; Biomarkers; Deep Brain Stimulation.

RESUMO

Objetivo: Investigar a contribuição dos componentes aperiódicos do sinal neural no mapeamento cirúrgico e sua correlação clínica em pacientes submetidos à Estimulação Cerebral Profunda. **Método:** Revisão Sistemática da Literatura conforme PRISMA 2020 nas bases BVS, CAPES, IEEE Xplore e PubMed (2021–2026), selecionando estudos sobre parametrização espectral, com avaliação da qualidade metodológica pela escala de Newcastle-Ottawa adaptada. **Resultados:** Foram selecionados 25 artigos de 90. Parâmetros aperiódicos forneceram informação complementar às métricas oscilatórias na caracterização anatômica e funcional. Alterações no expoente associaram-se à medicação e à Estimulação Cerebral Profunda em múltiplos estudos, de forma consistente com o balanço excitação/inibição, embora a relação com escalas clínicas nem sempre tenha sido observada. **Conclusão:** A dinâmica aperiódica constitui um biomarcador promissor da excitabilidade neural em Estimulação Cerebral Profunda, com evidência inicial de generalização entre patologias, ainda a ser confirmada por estudos multicêntricos e longitudinais.

Descritores: Eletrofisiologia; Biomarcadores; Estimulação Encefálica Profunda.

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1 INTRODUCTION

The analysis of neurophysiological activity has historically been guided by the identification of periodic oscillatory components, grounded since the earliest electroencephalography (EEG) recordings [1]. These oscillations are traditionally segmented into frequency bands and correlated with various cognitive functions and pathological states [2]. However, the decomposition of neural signals reveals that a substantial portion of brain activity is aperiodic in nature [3].

Stochastic processes in the brain give rise to a power spectral density (PSD) that exhibits a characteristic broadband decay, known as the $1/f^{\chi}$ component [4]. Although often dismissed as instrumental noise, recent studies demonstrate that the aperiodic exponent is a robust indicator of the synaptic excitation/inhibition (E/I) balance [3,5]. This dynamic reflects the neural excitability infrastructure and varies consistently across recording modalities: local field potentials (LFP) signals exhibit Brown noise behavior ($1/f^2$), while spiking recordings approach white noise (1/f), reflecting the different scales of spatial integration and the inherent dynamics of cortical and subcortical activity [6,7].

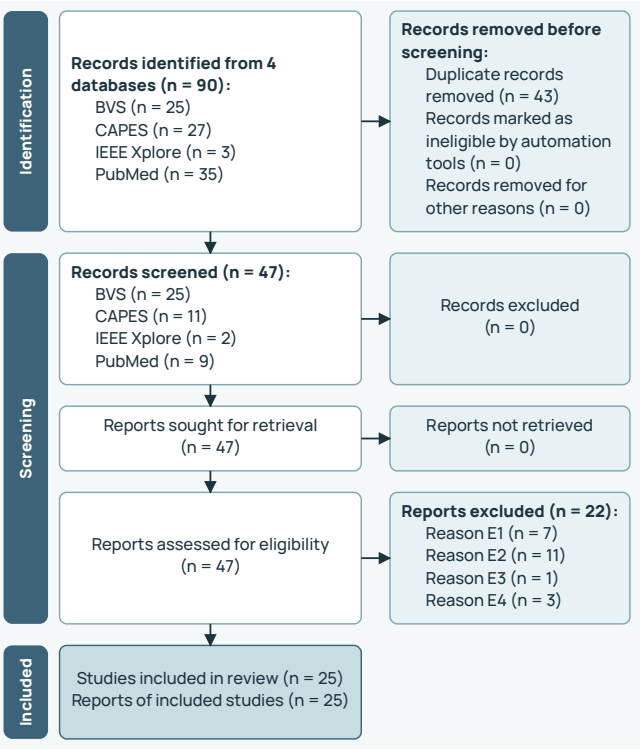
Deep Brain Stimulation (DBS) is an established neuromodulation therapy involving surgical implantation of electrodes to deliver electrical impulses to specific subcortical targets. This implantation procedure enables concurrent acquisition of multiple electrophysiological signals, offering a comprehensive view of the basal-cortical network [8]. In this context, aperiodic components emerge as promising tools for surgical mapping and therapeutic monitoring in pathologies such as Parkinson's disease (PD), dystonia, and depression [5,9,10]. Algorithms such as Specparam (also known as FOOOF) [3] and the Irregular Resampling Auto-Spectral Analysis (IRASA) [11] enable precise separation of these components, allowing a more faithful analysis of the neural infrastructure.

This work is justified by the need to consolidate the state of the art on aperiodic dynamics in the treatment of conditions such as PD. The general objective of this systematic literature review (SLR) is to investigate how aperiodic components of neural signals contribute to the accuracy of surgical mapping and whether they correlate with clinical assessment scales. Specifically, it aims to assess whether aperiodicity behaves as a stable candidate biomarker and to examine whether the neurophysiological response to DBS modulation is shared across pathologies or is pathology-specific.

2 METHODS

This systematic literature review was conducted and reported in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 guidelines, whose three phases (identification, screening, and inclusion) are illustrated in Figure 1.

Figure 1. PRISMA 2020 flowchart for the study selection process



Source: Prepared by the authors (2026).

To ensure rigor and replicability, the scope of the search and eligibility criteria were delimited using the Population, Intervention, Comparison, Outcomes, and Study Design (PICOS) framework, structured around four pre-specified research questions seen in Table 1. The population of interest consisted of patients diagnosed with movement disorders or neuropsychiatric disorders undergoing DBS. The intervention of interest was DBS combined with quantitative neurophysiological analysis of aperiodic (1/f) signal components; this was compared against the relative performance of conventional periodic (band-power) metrics. Outcomes of interest encompassed intraoperative surgical mapping accuracy, electrode contact localization, and correlation with validated clinical assessment scales. Eligible study designs were restricted to primary quantitative investigations characterizing aperiodic neural dynamics in the DBS context.

For clarity, we define aperiodic parameters as the spectral exponent (χ) and offset derived from parameterization algorithms such as FOOOF/Specparam or IRASA. Some included studies additionally examined complementary electrophysiological phenomena (such as evoked potentials, neural avalanches, and EEG microstates) that are conceptually distinct from these core parameters but

were retained within scope when explicitly linked by the primary studies to aperiodic or scale-free dynamics (e.g., avalanche statistics as an empirical signature of near-critical network states).

Furthermore, considering the ethical aspects of this research, since this is an SLR using exclusively publicly available data from open-access scientific articles, submission to a Research Ethics Committee (REC) was not required.

2.1 Research Questions

In order to guide the data collection and analysis process, research questions were established to delimit the scope of this review. These questions aim to investigate the state of the art of aperiodic dynamics analysis in neural signals applied to the DBS context. The formulated questions, along with their respective objectives, are presented in Table 1.

Table 1. Research Questions

Research Question	Objective
RQ1. What is the efficacy of the aperiodic metric in differentiating clinical states compared to traditional frequency bands?	To assess whether aperiodicity is a more stable and sensitive biomarker than periodic oscillations.
RQ2. Is there a statistically significant correlation between aperiodic parameters and clinical assessment scales in patients with movement disorders?	To evaluate the viability of aperiodic components as physiological markers with clinical utility.
RQ3. How do aperiodic components of the neural signal contribute to the accuracy of surgical mapping and DBS electrode localization in the subthalamic nucleus (STN)?	To evaluate the robustness of aperiodicity as a guide for precise device placement.
RQ4. Does aperiodic dynamics respond to modulation in a pathology- and target-specific way, or does it constitute a shared neurophysiological response across targets, pathologies, and modulation modalities?	To examine whether the analysis of aperiodic dynamics in neural signals reveals fundamental network properties, regardless of the specific pathology.

Source: Prepared by the authors (2026).

2.2 Search Strategy

The identification of primary studies was conducted through automated searches in four electronic databases: BVS, CAPES, IEEE Xplore, and PubMed. The foundational search string was syntactically adapted for each database to utilize native indexing features while maintaining conceptual consistency across all queries. The core conceptual string applied across all platforms was:

(("Deep Brain Stimulation" OR "DBS" OR "Subthalamic Nucleus" OR "STN") AND ("Tsallis" OR "Non-extensive" OR "q-Gaussian" OR "Superstatistics" OR "Aperiodic" OR "Heavy-tail" OR "Non-ergodic" OR "Scale-free") AND ("Parkinson" OR "Microelectrode recording" OR "MER" OR "LFP" OR "EEG" OR "SPK" OR "ECoG" OR "sEEG"))

The second concept block was deliberately built around scale-free and non-extensive statistical terminology rather than around specific parameterization tools, in order to capture the broad theoretical family of methods that quantify aperiodic dynamics without presupposing a single algorithm. We acknowledge as a limitation that implementation-specific terms (e.g., "FOOOF," "specparam," "IRASA") were not used as explicit search terms; the broad conceptual terms "Aperiodic" and "Scale-free," combined with backward citation screening of the included studies, were relied upon to reduce the risk of omitting relevant records. This limitation is addressed further in Section 3.3.

2.3 Selection Criteria

The inclusion and exclusion criteria applied during screening are detailed in Table 2.

Table 2. Selection criteria for the SLR

Category	Criterion
Inclusion	I1. Primary studies analyzing the aperiodic (1/f) or non-oscillatory component of electrophysiological signals.
	I2. Investigation of movement disorders or psychiatric disorders in the context of DBS.
	I3. Use of complexity, fractality, entropy, or non-extensive statistics metrics to characterize brain dynamics.
	I4. Studies published between 2021 and 2026.
Exclusion	E1. Studies with interventions outside the scope of conventional DBS, purely clinical studies without quantitative neural signal analysis, or purely mathematical studies without application to real data.
	E2. Secondary literature (reviews, editorials), conference abstracts, oral presentations, or preprints not peer-reviewed.
	E3. Single-case reports (N=1).
	E4. Experimental models used strictly in animals without direct correlation with human biomarkers.

Source: Prepared by the authors (2026).

2.4 Data Extraction

A two-phase data extraction protocol was executed by one independent investigator (L.S.M.P.). Initial screening, encompassing study identification and the application of eligibility criteria, was conducted using the Parsif.al platform. This system documented all inclusion and exclusion decisions, along with their corresponding justifications, as outlined in Section 2.3. Following this screening phase, the 25 eligible studies were exported to a custom spreadsheet for comprehensive data retrieval.

All included papers were further categorized according to the four foundational research questions and assessed for methodological quality using the modified Newcastle-Ottawa Scale, detailed in Section 2.5. The resulting dataset informs the qualitative synthesis in

Section 3.1 and the itemized bias assessment in Table 3. Because this process was managed by a single researcher, it was not possible to evaluate inter-rater agreement; this limitation is addressed in Section 3.3.

2.5 Quality Assessment

Risk of bias was assessed using the Newcastle-Ottawa Scale (NOS), applied by the same investigator (L.S.M.P.) who conducted data extraction. Given the methodological diversity of the included studies, the NOS cohort version was adapted to the specific technical requirements of DBS electrophysiology. Studies were scored up to nine stars across three domains, classifying quality as low (0–3), moderate (4–6), or high (7–9).

In the Selection domain (maximum 4 stars), one star was awarded for each of the following: adequate clinical profiling; precise electrode anatomical verification; robust aperiodic signal extraction with reported fit metrics; and an explicitly defined resting-state baseline with documented artifact rejection. The Comparability domain (maximum 2 stars) awarded points for strict control of primary physiological or pharmacological states and for standardization of task execution or stimulation parameters. The Outcome domain (maximum 3 stars) evaluated the use of validated, disease-specific clinical scales; adequate temporal framing (requiring ≥3 months of follow-up for neuroplasticity claims, or explicit acknowledgment of acute-study limits); and overall data transparency, including reported sample sizes, exclusions, and artifact rejection rates.

3 RESULTS AND DISCUSSION

The selection process resulted in 25 included articles. LFP was analyzed in all studies (n=25), with additional modalities including MER/Spiking (n=4), EEG (n=3), SPK (n=2), ECoG (n=1), and sEEG (n=1). Spectral parameterization was performed predominantly via FOOOF/Specparam (n=21), complemented by IRASA (n=3), direct PSD analysis (n=1), and neural avalanche metrics (n=1).

The clinical sample focused primarily on PD (n=20), followed by dystonia (n=3), epilepsy (n=2), essential tremor (n=1), treatment-resistant depression (TRD, n=1), and bipolar/unipolar depression (n=1). The STN was the most frequent anatomical target (n=20), followed by GPi/GPe (n=3), thalamus (n=3), SNr (n=2), cortex (n=2), habenula (n=1), and the BNST-NAc circuit (n=1); totals exceed the article count since several studies compared multiple targets.

Clinical outcomes were assessed mainly via the UPDRS-III/MDS-UPDRS-III (n=20), with dystonia-specific scales (TWSTRS, BFMDRS, JRS) and psychiatric scales (HAMD-17, HAMA) used elsewhere; two studies used non-standard outcomes: a retrospective efficacy ranking and seizure-count reduction. The synthesis below follows the research-question structure summarized in Table 3.

3.1 Analysis of Research Questions

RQ1. What is the efficacy of the aperiodic metric in differentiating clinical states compared to traditional frequency bands?

Several studies reported that aperiodic parameters carried information not captured by traditional band-power measures. Bush *et al.* found a marked difference between cortical and subcortical exponents (approximately 3.2 versus 1.3, respectively), a pattern reproducible across patients, centers, and diagnoses [6]. Opri *et al.* reported that the DBS-induced local evoked potential (DLEP), linked to aperiodic dynamics, identified the optimal STN contact with higher accuracy (100%) than the beta metric (89.74%) in their cohort [12]. Complementing this, Pardo-Valencia *et al.* showed that raw beta power can conflate oscillatory and aperiodic background activity, supporting the view that component separation is a useful methodological step for interpreting band-limited measures [13], though this has not been formally tested across the full range of clinical contexts represented in this review.

RQ2. Is there a statistically significant correlation between aperiodic parameters and clinical assessment scales in patients with movement disorders?

Multiple studies reported correlations between aperiodic parameters and UPDRS-III/MDS-UPDRS-III scores, though findings were not fully consistent across the corpus. Gerster *et al.* reported that broadband aperiodic power tracked individual bradykinesia severity in real time, with mid-gamma (45–60 Hz) activity of aperiodic origin functioning as a candidate proxy in their sample [14]. Sayfulina *et al.* found that levodopa-induced change in the 1/f exponent correlated with improvement in bradykinesia ($p=0.005$) and rigidity ($p=0.007$) [15]. Clark *et al.* reported that combining periodic beta with the aperiodic slope improved prediction of clinical response to DBS at 12 months relative to beta power alone [16]. Outside movement disorders, Sonkusare *et al.* reported a correlation between the aperiodic exponent of the left habenula and HAMD scores in patients with treatment-refractory bipolar or unipolar depression ($r=0.88$), a single-study finding suggestive of clinical relevance in psychiatric contexts that warrants replication [9]. Notably, other studies did not find direct correlations between the exponent and UPDRS sub-scores, including Shi *et al.* [17] and Wiest *et al.* [5], indicating that the aperiodic-clinical relationship may depend on disease context and may be more reliably detected when combined with periodic metrics rather than used in isolation.

Table 3. Articles selected in the systematic review

Authors (year)	Signal	Metric	Target	Pathology	NOS	Main Finding
Bush et al. (2024) [6]	LFP, ECoG, sEEG	Specparam (custom Lorentzian fit)	Cortex, STN, GPi, SNr, Thalamus	PD, essential tremor, epilepsy	8/9	Subcortical exponent (≈1.3) lower than cortical (≈3.2); cortical exponent tracks ON-state motor severity.
Clark et al. (2023) [16]	LFP	FOOOF	STN	PD	6/9	Beta + aperiodic slope predicts 12-month DBS response; beta alone cannot.
Balachandar et al. (2026) [18]	LFP	FOOOF	STN	PD	6/9	Chronic DBS: higher beta, lower aperiodic exponent/offset; tracks long-term network change.
Gerster et al. (2025) [14]	LFP	FOOOF	STN	PD	8/9	Mid-gamma aperiodic power tracks bradykinesia severity across ON/OFF states.
Shi et al. (2025) [17]	LFP, MER	PSD, FOOOF	SNr	PD, dystonia	5/9	Reduced SNr exponent in PD vs. dystonia reflects hyperexcitability; no direct clinical correlation.
Opri et al. (2025) [12]	LFP via MER	IRASA, DLEP	STN	PD	7/9	DLEP identified optimal STN contact more accurately than beta (100% vs. 89.74%).
Joshi et al. (2025) [19]	LFP	FOOOF, IRASA	STN	PD	5/9	Cycling increased dorsal STN exponent, suggesting neuroplasticity; tremor improved.
Belova et al. (2023) [20]	LFP (macro)	FOOOF	STN	PD	5/9	Alpha-beta peaks (8–15 Hz), visible only post-decomposition, mark disease duration/severity.
Joshi et al. (2026) [21]	LFP	FOOOF, iCOH, PSI	STN	PD	6/9	One month of exercise increased dorsal STN aperiodic exponent/offset; oscillations unchanged.
Agouram et al. (2025) [22]	EEG, LFP	Avalanches, ATM	STN, motor cortex	PD	5/9	Levodopa increased avalanche propagation; aperiodic burst dynamics correlated with improvement.
Lamoš et al. (2026) [23]	LFP	FOOOF	STN	PD	7/9	DBS ON increased aperiodic slope/offset; stable at 1 year; tracked motor, not tremor, severity.
Darmani et al. (2023) [24]	LFP	IRASA	STN	PD	7/9	Exponent/offset rose over 18 months post-DBS regardless of medication, tracking neuroplasticity.
Stam et al. (2025) [25]	LFP	FOOOF	STN	PD	6/9	Aperiodic metrics increased with DBS ON; moderate reliability as supplementary marker.
Pardo-Valencia et al. (2024) [13]	LFP	FOOOF, Sharpness	STN	PD	8/9	Raw beta conflates aperiodic noise; spectral sharpness is a cleaner oscillatory biomarker.
Semenova et al. (2021) [26]	LFP	1/f slope (log-log fit, 30–70 Hz)	GPe, GPi	Cervical dystonia	3/9	Inter-hemispheric GPi slope asymmetry reflects tonic E/I imbalance in cervical dystonia.
Sayfulina et al. (2025) [15]	LFP	FOOOF	STN	PD	6/9	Levodopa-induced exponent increase correlated with bradykinesia and rigidity improvement.
Wu et al. (2023) [27]	LFP via MER	FOOOF, PAC	STN	PD	6/9	Early-onset PD showed lower aperiodic parameters, higher low-beta, than late-onset.
Sonkusare et al. (2022) [9]	LFP, EEG	1/f slope, ERSP	Habenula	Bipolar/unipolar depression	4/9	Left habenula exponent correlated with depression severity (r=0.88).
Yang et al. (2023) [28]	LFP	Spectral decomposition (Donoghue-based)	ANT, CM	Epilepsy	6/9	Aperiodic slope was the sole biomarker distinguishing ictal from inter-ictal states.
Wiest et al. (2023) [10]	LFP	FOOOF	STN, VIM, ZI	Dystonia	5/9	STN-DBS increased exponent in dystonia identically to PD, suggesting a disease-nonspecific response.
Peng et al. (2024) [29]	LFP	FOOOF	STN	PD	5/9	Exponent peaks during hyperacute microlesion phase; levodopa mimics this effect.
Liu et al. (2024) [7]	LFP, SPK	FOOOF	STN	PD	5/9	SPK aperiodic offset identified STN entry; combined features outperformed demographics.
Tani et al. (2026) [30]	LFP	FOOOF	STN	PD	7/9	Periodic beta is maximal dorsally; aperiodic exponent is greater ventrally, a spatial dissociation useful for adaptive DBS contact selection.
Wiest et al. (2023) [5]	LFP, SPK	FOOOF	STN, GPe	PD (rat + human)	8/9	Cross-species: exponent reflects synaptic E/I balance; levodopa/DBS both increased it.
Ye et al. (2025) [31]	LFP, EEG	FOOOF, microstates	BNST-NAc	TRD	6/9	NAc aperiodic gamma bursts triggered microstate transitions correlated with improvement (r>0.73).

Source: Prepared by the authors (2026).

RQ3. How do aperiodic components of the neural signal contribute to the accuracy of surgical mapping and DBS electrode localization in the subthalamic nucleus?

Aperiodic parameters showed potential value as an intraoperative surgical guide through the identification of anatomical transitions and functional subdomains, though this evidence is based on a small number of studies. Liu *et al.* reported that the aperiodic offset of the spiking signal was greater within the STN than in the pre-STN region, distinguishing the dorsolateral oscillatory region (DLOR, motor) from the ventromedial non-oscillatory region (VMNR, non-motor) in their cohort [7]. Tani *et al.* identified a spatial dissociation in which periodic beta best characterized the dorsal motor zone while the aperiodic exponent showed a distinct, more ventral topography [30], a pattern that may inform adaptive DBS programming pending further validation.

RQ4. Does aperiodic dynamics respond to modulation in a pathology- and target-specific way, or does it constitute a shared neurophysiological response across targets, pathologies, and modulation modalities?

The available evidence is consistent with, though not conclusive proof of, a transdiagnostic effect of DBS on aperiodic dynamics. Wiest *et al.* reported that STN-DBS produced broadband suppression and an increase in the aperiodic exponent in dystonia patients similar to patterns observed in PD [10], interpreting this as evidence of a disease-nonspecific response, though this interpretation is based on a single comparative study. Yang *et al.* extended this observation to epilepsy, reporting that the thalamic 1/f slope distinguished ictal from baseline states in their sample, in the absence of detectable oscillatory differences [28]. Ye *et al.* reported an aperiodic gamma response in the BNST-NAc during DBS for TRD, correlating with mood scale improvement ($r>0.73$) [31]. Wiest *et al.* also reported cross-species evidence, in both animal and human models, linking the 1/f exponent to synaptic E/I balance at the cellular level [5].

3.2 Implications for Health Informatics and the Brazilian Context

Because the aperiodic exponent can be estimated automatically and in near real time, it is well suited to clinical decision-support systems and closed-loop DBS, where stimulation is adjusted from a computed biomarker rather than fixed parameters. This has direct relevance to the Sistema Único de Saúde (SUS), where DBS is offered at a limited number of specialized centers and access to surgical and programming expertise varies markedly by region. Low-cost, software-based biomarkers could help narrow this gap by reducing dependence on operator experience during targeting and programming. At the same time, chronic intracranial data collection by sensing-enabled devices raises data-governance and privacy questions under Brazil's Lei Geral de Proteção de Dados. Integrating aperiodic biomarkers into SUS-compatible, auditable, privacy-preserving infrastructure is therefore both a technical and a governance challenge.

3.3 Limitations

This review has several limitations. Study selection, extraction, and quality scoring were performed by a single reviewer, precluding assessment of inter-rater agreement. Although methodological quality was formally scored using the adapted NOS (Table 3), these scores were not used to exclude studies or to systematically adjust the emphasis given to individual findings in the synthesis above; they are reported for transparency and to support future meta-analytic use rather than as a formal weighting mechanism applied in this review. The search strategy relied on broad conceptual terms rather than implementation-specific algorithm names, which may have missed studies using idiosyncratic terminology.

The evidence base is also concentrated in PD and the STN, limiting generalizability to other targets and pathologies. Heterogeneity in spectral parameterization methods (FOOOF/Specparam vs. IRASA) introduces variability in how aperiodic components are defined and extracted, and most studies rely on small, single-center, cross-sectional cohorts, which precludes causal inference and limits external validation. These limitations point to the need for multicenter, longitudinal studies with standardized parameterization protocols, independent dual review, and formal quality-weighted synthesis to consolidate aperiodicity as a clinical-grade biomarker.

4 CONCLUSIONS

This systematic review gathered evidence from 25 primary studies investigating aperiodic dynamics in the neural signals of patients undergoing DBS. The results suggest that the aperiodic component, quantified primarily by the exponent and offset of the 1/f spectrum via FOOOF/Specparam, is a candidate biomarker with utility suggested across three clinical dimensions: state differentiation (RQ1), intraoperative surgical navigation (RQ3), and, more tentatively, correlation with standardized motor scales (RQ2), for which findings were less consistent across the corpus.

The evidence is broadly consistent with, though not sufficient to confirm, the interpretation that the aperiodic exponent indexes synaptic E/I balance within the basal-cortical network, potentially complementing beta oscillations' sensitivity to momentary dopaminergic tone rather than duplicating it. Aperiodic modulation by DBS and by levodopa was reported across PD, dystonia, epilepsy, and treatment-resistant depression (RQ4), but this transdiagnostic

interpretation rests on a still-limited and heterogeneous evidence base, drawn largely from single studies per condition, and warrants replication in larger, multicenter cohorts before it can be considered established.

Future research should prioritize standardized parameterization protocols and independent dual review, and could investigate neural criticality as an organizing principle for monitoring phase-transition states via the aperiodic component.

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Use of artificial intelligence. During the preparation of this work, the authors used Generative Artificial Intelligence tools to assist with grammatical revision, improvement of writing clarity, and logical structuring of the text in accordance with the template guidelines. Following the use of these tools, the authors critically reviewed and edited the content, assuming full responsibility for the scientific accuracy and integrity of the final version of the manuscript.

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EDITORIAL NOTES

The Journal of Health Informatics is a scientific journal maintained by the Brazilian Society of Health Informatics (Sociedade Brasileira de Informática em Saúde), a non-profit scientific association dedicated to the development of Health Informatics and Digital Health in Brazil. The editorial process is conducted with scientific independence, free from institutional, commercial, or sponsor interference, and follows international best practices in publication ethics.

This article was double-blind peer reviewed by specialists in the field. The editorial team used artificial intelligence tools, under human supervision, in the initial screening, in checking the text, references and numbers and, when needed, in redrawing figures, charts and tables, which the authors approved in the proof. The tools and safeguards are described in the journal's Policy on the use of artificial intelligence. Editorial decisions were made by people.

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