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Neurophysiological Effects of Intermittent Hypoxia on Brain Activity: A Systematic Review

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Abstract: Intermittent hypoxia modulates brain function, producing adaptive and maladaptive neurophysiological effects depending on oxygen deprivation characteristics. Quantitative electroencephalography detects hypoxia-induced cortical alterations, but evidence remains fragmented. This systematic review synthesized evidence on intermittent hypoxia's neurophysiological effects on brain activity assessed by electroencephalography. The review followed PRISMA 2020 guidelines. PubMed, Scopus, Embase, and Web of Science were systematically searched from June 1 to June 3, 2026. Eligible human studies evaluated intermittent hypoxia using quantitative electroencephalographic measures. Two reviewers independently performed study selection, data extraction, and methodological quality assessment using the Joanna Briggs Institute Critical Appraisal Tools. Owing to methodological heterogeneity, findings were synthesized narratively. Five studies involving 258 participants met the eligibility criteria. Intermittent hypoxia was associated with altered cortical oscillatory activity, including increased delta and theta power and reduced alpha activity. Controlled hypoxic interventions generally enhanced functional connectivity, whereas chronic pathological hypoxia in obstructive sleep apnea was associated with impaired network connectivity. Methodological quality ranged from moderate to high; however, the small number of heterogeneous studies warrants cautious interpretation. Quantitative electroencephalography is a promising biomarker, but larger standardized studies are needed before clinical translation.

1. INTRODUCTION

Intermittent hypoxia (IH) refers to repeated episodes of reduced oxygen availability alternating with periods of normoxia and occurs in both pathological conditions, such as obstructive sleep apnea (OSA), and controlled therapeutic interventions, including intermittent hypoxic training and hypoxic conditioning [1]. IH has emerged as an important neuromodulatory stimulus because its physiological effects depend on the severity, duration, and frequency of hypoxic exposure [2]. While prolonged or severe IH has been associated with cognitive impairment, oxidative stress, and neuronal injury, appropriately dosed hypoxic exposure may promote neuroplasticity and adaptive neural responses [3].

The neurophysiological effects of IH are mediated through complex molecular and cerebrovascular mechanisms involving hypoxia-inducible factor-1α (HIF-1α), brain-derived neurotrophic factor (BDNF), vascular endothelial growth factor (VEGF), and other oxygen-sensitive signaling pathways that regulate neuronal adaptation [4]. Excessive hypoxic exposure may also induce neuroinflammation, mitochondrial dysfunction, and impaired cerebral autoregulation, ultimately disrupting cortical

function [5]. These biological alterations are reflected in changes in neuronal oscillatory activity, cortical excitability, and large-scale brain network synchronization, which may influence attention, memory, executive function, and sensorimotor processing [6]. Quantitative electroencephalography (qEEG) provides a sensitive, non-invasive approach for detecting these neurophysiological changes by objectively measuring spectral power, frequency distribution, coherence, and functional connectivity with high temporal resolution [7].

Despite growing interest in the neurological consequences of IH, current evidence remains heterogeneous because studies employ diverse hypoxic protocols, participant populations, and qEEG outcome measures [8]. Previous investigations have reported alterations in delta, theta, alpha, and beta oscillations as well as changes in cortical connectivity; however, the reported findings remain inconsistent across experimental and clinical settings [9]. To date, no systematic review has specifically synthesized quantitative EEG evidence describing the neurophysiological effects of intermittent hypoxia on brain activity [10]. Therefore, this systematic review aims to comprehensively evaluate qEEG findings related to intermittent hypoxia, summarize changes in electrophysiological brain activity and functional connectivity, and identify current knowledge gaps to guide future neuroscience and clinical research.

2. MATERIALS AND METHODS

2.1 Study Design

This systematic review was conducted to synthesize the available evidence regarding the neurophysiological effects of intermittent hypoxia on brain activity assessed using qEEG. The review methodology was developed and reported in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 statement to ensure methodological transparency, reproducibility, and comprehensive reporting throughout the review process. Owing to the anticipated heterogeneity in study design, participant characteristics, hypoxic protocols, and electrophysiological outcome measures, a narrative synthesis was planned rather than a quantitative meta-analysis.

2.2 Eligibility Criteria

Study eligibility was established according to the Population, Exposure, Comparator, Outcomes, and Study Design (PECOS) framework. Eligible studies included human participants exposed to intermittent hypoxia, whether experimentally induced, administered as intermittent hypoxic exposure or intermittent hypoxic training, or occurring secondary to clinical conditions such as obstructive sleep apnea. Studies involving both healthy individuals and clinical populations were considered eligible.

Studies were included if they evaluated brain activity using quantitative EEG analyses, including spectral power analysis, frequency-domain analysis, coherence, functional connectivity, phase synchronization indices, or other quantitative electrophysiological parameters. Eligible study designs comprised randomized controlled trials, crossover trials, prospective and retrospective cohort studies, case-control studies, cross-sectional studies, and quasi-experimental studies. Only full-text articles published in peer-reviewed journals and written in English were included.

Animal experiments, in vitro studies, conference abstracts, editorials, letters, commentaries, study protocols, narrative reviews, systematic reviews, meta-analyses, and case reports were excluded. Studies were also excluded if they did not involve intermittent hypoxia, did not evaluate brain activity using EEG-based techniques, or failed to report quantitative electrophysiological outcomes.

2.3 Search Strategy

A comprehensive literature search was performed in PubMed, Scopus, Embase, and Web of Science between June 1 and June 3, 2026. These databases were selected to maximize the retrieval of biomedical, neuroscience, and multidisciplinary studies investigating the effects of intermittent hypoxia on brain electrophysiology. No restriction on publication year was applied to ensure comprehensive evidence retrieval, whereas eligibility was limited to English-language full-text publications.

The electronic search strategy combined controlled vocabulary and free-text terms related to intermittent hypoxia and electrophysiological brain activity. The core search syntax was as follows: ("intermittent hypoxia" OR "acute intermittent hypoxia" OR "intermittent hypoxic exposure" OR "intermittent hypoxic training" OR "rhythmic intermittent hypoxia") AND (EEG OR electroencephalography OR qEEG OR "quantitative EEG" OR "spectral power" OR "brain oscillations" OR coherence OR connectivity). Database-specific adaptations were applied where appropriate while preserving the same

conceptual framework. The search identified 885 records, including 243 from PubMed, 387 from Scopus, 198 from Embase, and 57 from Web of Science. Reference lists of all included articles were also manually screened to identify additional relevant studies not captured during the electronic search. The complete database-specific search strategies are provided in the Supplementary Material.

2.4 Study Selection

All retrieved records were exported into reference management software, and duplicate publications were removed before screening. Study selection was conducted independently by two reviewers using a two-stage process. First, titles and abstracts were screened to identify potentially eligible studies according to the predefined eligibility criteria. Articles considered potentially relevant subsequently underwent full-text assessment.

During full-text review, studies were evaluated against all inclusion and exclusion criteria, and reasons for exclusion were documented. Any disagreement between reviewers regarding study eligibility was resolved through discussion and consensus. When consensus could not be achieved, a third reviewer made the final decision. The complete selection process was documented using the PRISMA 2020 flow diagram.

2.5 Data Extraction

Data extraction was independently performed by two reviewers using a standardized extraction form developed specifically for this review. Discrepancies were resolved through discussion until consensus was reached. The extracted information included the first author, publication year, study design, participant characteristics, sample size, intermittent hypoxia protocol, electrophysiological assessment methods, outcome measures, and principal findings. Particular attention was given to quantitative electrophysiological variables, including spectral power across delta, theta, alpha, beta, and gamma frequency bands, coherence, functional connectivity, phase synchronization, and other reported neurophysiological parameters. Extracted data were summarized in evidence tables before narrative synthesis.

2.6 Quality Assessment

The methodological quality of the included studies was independently assessed by two reviewers using the Joanna Briggs Institute Critical Appraisal Tools. The appropriate JBI checklist was selected according to each study design, including instruments for randomized controlled trials, cohort studies, case-control studies, and quasi-experimental studies. Each study was evaluated across domains addressing participant selection, exposure assessment, outcome measurement, management of confounding variables, follow-up completeness, and statistical analysis. Individual checklist items were rated as "Yes," "No," "Unclear," or "Not Applicable" according to JBI guidance. For descriptive interpretation, the proportion of applicable items rated "Yes" was used to classify studies as high quality (≥80%), moderate quality (50–79%), or low quality (<50%). These classifications were used to contextualize confidence in the evidence, not as numerical scores for weighting studies or excluding them from the narrative synthesis. Any discrepancies between reviewers were resolved through consensus before final quality classification.

2.7 Data Synthesis

Given the substantial methodological heterogeneity across the included studies with respect to participant populations, hypoxic exposure protocols, EEG acquisition methods, analytical approaches, and reported electrophysiological outcomes, quantitative pooling of results was considered inappropriate. Consequently, findings were synthesized narratively by grouping studies according to major neurophysiological domains, including spectral power alterations, oscillatory activity, cortical excitability, coherence, functional connectivity, and other electrophysiological markers. The methodological quality of individual studies was considered during interpretation to provide a balanced assessment of the current evidence and to identify priorities for future research.

3. RESULTS AND DISCUSSION

3.1 Study Selection

The database search identified 885 records, comprising 243 from PubMed, 387 from Scopus, 198 from Embase, and 57 from Web of Science. After removing 247 duplicate records, 638 articles remained for title and abstract screening. Of these, 607 records were excluded because they did not meet the predefined eligibility criteria. Thirty-one full-text articles were assessed

for eligibility, of which 26 were excluded due to the absence of quantitative EEG outcomes, lack of intermittent hypoxia exposure, non-human study design, review publication type, conference abstract format, or insufficient electrophysiological data. Ultimately, five studies fulfilled all eligibility criteria and were included in the qualitative synthesis (Figure 1).

3.2 Study Characteristics

The five included studies were published between 2001 and 2024 and collectively involved 258 participants. The evidence base included healthy volunteers exposed to controlled hypoxia and patients with obstructive sleep apnea, with individual study samples ranging from 10 healthy participants to 161 participants in the largest prospective study. Included study designs comprised one randomized controlled trial, one single-blind randomized crossover trial, one prospective cohort study, one matched case-control pilot study, and one experimental cohort study.

Considerable methodological heterogeneity was observed across studies. Two investigations evaluated controlled normobaric hypoxia, one compared normobaric with hypobaric hypoxia, and two examined chronic intermittent hypoxia associated with obstructive sleep apnea. EEG outcomes also varied, encompassing spectral power, induced and evoked oscillatory power, coherence, phase lag index, and other functional-connectivity measures. Accordingly, the findings should be interpreted by hypoxic paradigm and outcome domain rather than as a single uniform effect of intermittent hypoxia (Table 1).

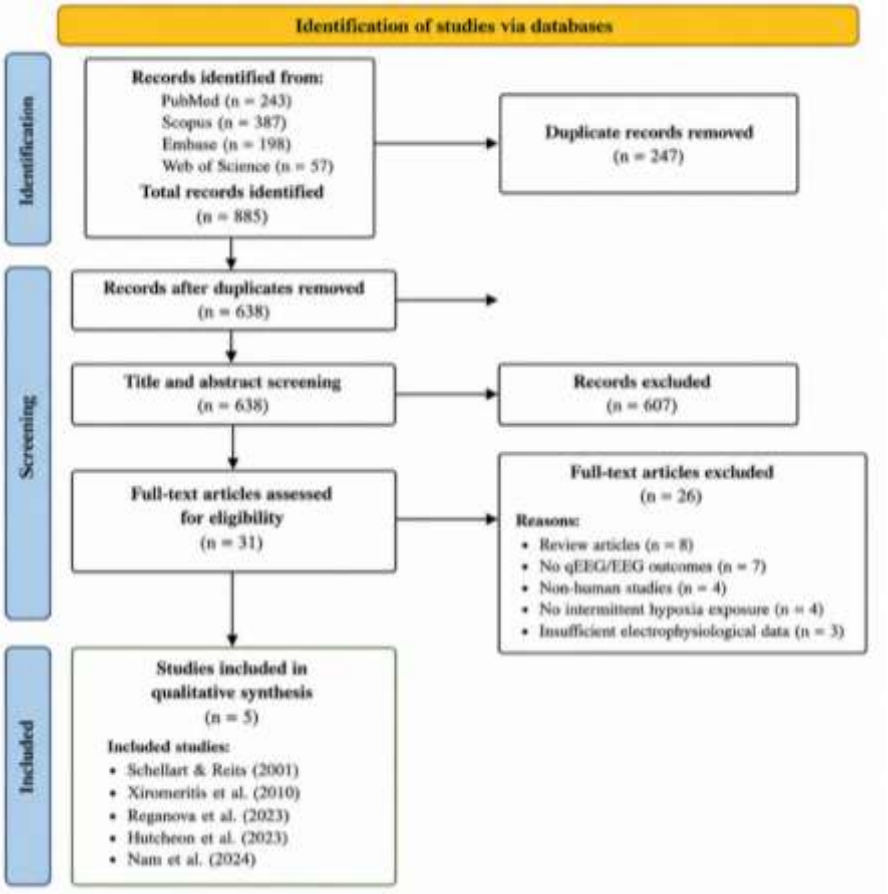


Figure 1. PRISMA 2020 flow diagram summarizing the identification, screening, eligibility assessment, and inclusion of studies in the systematic review.

3.3 Methodological Quality

Methodological quality was assessed using the Joanna Briggs Institute Critical Appraisal Tools according to study design. Two studies were classified as high quality, whereas three were considered of moderate quality because some applicable domains were rated "No" or "Unclear." The classifications indicate the transparency and completeness of reported methods within each design; they do not establish that the studies are directly comparable or that any individual finding is definitive. Quality appraisal

was therefore used to inform the certainty of the narrative interpretation, alongside the substantial variability in study design, hypoxic protocols, participant characteristics, and electrophysiological measurements (Table 2).

3.4 Neurophysiological Effects of Intermittent Hypoxia

Changes in EEG Spectral Power

Alterations in EEG spectral power represented the most consistently reported neurophysiological finding. Four of the five included studies reported significant changes across one or more frequency bands following intermittent hypoxia exposure. Overall, chronic or recurrent hypoxia was characterized by increased slow-wave activity, particularly within the delta and theta bands, whereas alpha activity generally decreased. Experimental hypoxia also produced reductions in alpha power accompanied by increases in beta activity under specific exposure conditions. Although the magnitude of these changes differed across protocols, the overall pattern suggests that intermittent hypoxia consistently modulates cortical oscillatory dynamics by shifting activity toward lower-frequency oscillations.

Changes in Functional Connectivity

Three studies further demonstrated that intermittent hypoxia affected large-scale functional brain networks. Controlled intermittent hypoxic training was consistently associated with increased functional connectivity, reflected by higher coherence and phase lag index values, suggesting enhanced synchronization between cortical regions. In contrast, chronic intermittent hypoxia secondary to obstructive sleep apnea was associated with reduced frontal delta connectivity despite increased local delta power, indicating that prolonged pathological hypoxia may impair network-level communication while simultaneously increasing regional slow-wave activity.

Table 1. Characteristics of the Included Studies

Study	Study Design	Participants	Hypoxia Type	EEG/qEEG Measures	Main Findings
Schellart & Reits (2001)	Experimental cohort study	10 healthy participants	Normobaric hypoxia (7–13% O_2)	Spectral power analysis (FFT)	Increased beta, theta, and delta power, accompanied by decreased alpha power during the eyes-closed condition.
Xiromeritis et al. (2010)	Prospective study	131 patients with obstructive sleep apnea and 30 healthy controls	Chronic intermittent hypoxia associated with obstructive sleep apnea	Relative spectral power (delta, theta, alpha, and beta)	Severe obstructive sleep apnea was associated with increased theta and delta activity in the occipital, temporal, and parietal regions. EEG slowing correlated with the severity of hypoxemia.
Reganova et al. (2023)	Randomized controlled trial	47 healthy men (hypoxia group, $n = 15$)	Normobaric intermittent hypoxic training	Functional connectivity (phase lag index and coherence)	Intermittent hypoxic training significantly increased functional connectivity compared with the control condition.
Hutcheon et al. (2023)	Single-blind randomized crossover trial	13 healthy participants	Normobaric hypoxia and hypobaric hypoxia	Induced and evoked oscillatory power (source-space EEG analysis)	Induced alpha power decreased during both normobaric and hypobaric hypoxia, whereas hypobaric hypoxia was associated with higher beta

					power than normoxia and normobaric hypoxia.
Nam et al. (2024)	Matched case-control pilot study	17 patients with obstructive sleep apnea and 17 healthy controls	Intermittent hypoxia associated with obstructive sleep apnea	Absolute and relative spectral power; functional connectivity (iCoherence)	Increased frontal delta power and reduced frontal delta-band functional connectivity were observed in patients with obstructive sleep apnea.

Overall Neurophysiological Pattern

Across all included studies, several common neurophysiological patterns emerged despite substantial methodological heterogeneity. Intermittent hypoxia consistently altered cortical oscillatory activity, characterized by increased delta and theta power together with suppression of alpha rhythms. At the network level, the direction of connectivity changes appeared to depend on the hypoxic paradigm, with controlled exposure generally promoting adaptive network synchronization, whereas chronic pathological hypoxia was associated with disrupted functional connectivity. Collectively, these findings indicate that intermittent hypoxia modifies brain function through complementary changes in oscillatory activity and large-scale cortical network organization, supporting the utility of qEEG as a sensitive tool for detecting hypoxia-induced neurophysiological alterations.

Table 2. Methodological Quality Assessment of the Included Studies Using the Joanna Briggs Institute Critical Appraisal Tools

Study	Study Design	JBI Assessment Tool	Yes	No	Unclear	Overall Quality
Reganova et al. (2023)	Randomized controlled trial	JBI Critical Appraisal Checklist for Randomized Controlled Trials	7	2	3	Moderate
Hutcheon et al. (2023)	Randomized crossover trial	JBI Critical Appraisal Checklist for Randomized Controlled Trials	5	2	2	Moderate
Nam et al. (2024)	Case-control study	JBI Critical Appraisal Checklist for Case-Control Studies	9	0	0	High
Schellart & Reits (2001)	Experimental cohort study	JBI Critical Appraisal Checklist for Cohort Studies	5	2	0	Moderate
Xiromeritis et al. (2010)	Prospective cohort study	JBI Critical Appraisal Checklist for Cohort Studies	7	0	0	High

3.5 Discussion

This systematic review synthesized evidence from five studies evaluating the neurophysiological effects of intermittent hypoxia on brain activity using qEEG. Despite substantial heterogeneity in participant characteristics, hypoxic paradigms, and electrophysiological analyses, the available evidence consistently demonstrated that intermittent hypoxia modifies cortical oscillatory activity and large-scale functional brain organization. The most reproducible findings were increased slow-wave activity, particularly within the delta and theta frequency bands, accompanied by reduced alpha activity, while changes in functional connectivity appeared to depend on the characteristics of hypoxic exposure. These findings are consistent with the growing recognition that intermittent hypoxia exerts both adaptive and maladaptive effects on the central nervous system depending on exposure intensity, duration, and physiological context [11].

The predominance of increased delta and theta activity together with alpha suppression suggests a shift toward reduced cortical efficiency and altered neuronal excitability under hypoxic conditions. Similar electrophysiological patterns have been described by Wong et al. (2023), who reported that oxygen deprivation preferentially disrupts higher-frequency oscillations while enhancing slow-wave activity through impaired oxidative metabolism and altered synaptic transmission [12]. Likewise, Zhang et al. (2023) demonstrated that cerebral hypoxia induces characteristic EEG slowing, reflecting reduced neuronal firing efficiency and compensatory metabolic adaptation [13]. These mechanisms are biologically plausible because neuronal activity is highly dependent on continuous oxygen delivery, and even transient reductions in cerebral oxygenation can alter membrane excitability and synaptic signaling.

An important finding of this review is that changes in functional connectivity differed according to the nature of hypoxic exposure. Controlled intermittent hypoxic interventions generally enhanced network synchronization, whereas chronic pathological hypoxia associated with obstructive sleep apnea disrupted large-scale cortical communication. These observations are in agreement with Micaux et al. (2025), who proposed that appropriately dosed intermittent hypoxia may induce adaptive neuroplastic remodeling, whereas repetitive nocturnal hypoxemia promotes network disorganization through cumulative oxidative stress and neuroinflammatory processes [7]. This apparent duality supports the concept of intermittent hypoxia as a biological stimulus with hormetic properties, in which moderate exposure may promote neural adaptation while excessive or prolonged exposure results in functional impairment.

Several biological mechanisms may explain these electrophysiological alterations. Oxygen-sensitive signaling pathways activated during hypoxia, particularly HIF-1α, regulate downstream mediators including VEGF and BDNF, thereby facilitating angiogenesis, synaptic plasticity, and neuronal survival during repeated oxygen fluctuations. Experimental evidence summarized by Wardaya et al. (2025) indicates that activation of these pathways contributes to adaptive cortical reorganization and may underlie the improved functional connectivity observed following intermittent hypoxic training [14]. Conversely, prolonged activation of hypoxia-responsive pathways may increase oxidative stress, mitochondrial dysfunction, and neuroinflammation, ultimately impairing neuronal communication and cortical network integrity. Similar mechanistic relationships between cerebral oxygen metabolism and electrophysiological alterations have also been described by Amaral-Silva and Santin (2023) and further supported by computational neurophysiological models reported by Iqbal et al. (2026) [8,15].

The observed alterations in spectral power also align with previous evidence indicating that slow-wave predominance represents a sensitive electrophysiological marker of impaired cortical metabolism. Ippolito et al. (2022) demonstrated that increased delta activity accompanies reduced cortical arousal and diminished information-processing capacity, whereas Zeng et al. (2022) reported that alpha suppression is consistently associated with hypoxia-induced reductions in cerebral oxygen availability [16,17]. Collectively, these findings support the interpretation that qEEG provides an objective surrogate marker of cerebral functional adaptation to intermittent hypoxia and may detect neurophysiological alterations before overt neurological dysfunction becomes clinically apparent.

Several limitations should be considered when interpreting these findings. First, only five studies met the inclusion criteria, and most individual studies enrolled relatively small samples; consequently, the evidence base is insufficient to support precise estimates of effect size or broad generalization. Second, substantial heterogeneity in participant populations, controlled versus pathological hypoxic exposure, EEG acquisition methods, analytical approaches, and outcome measures precluded quantitative meta-analysis and limits direct comparison across studies. Third, most evidence concerned short-term responses, whereas the long-term neurophysiological consequences of repeated intermittent hypoxia remain insufficiently investigated. Finally, the coexistence of healthy experimental populations and patients with obstructive sleep apnea means that observed differences may reflect distinct underlying physiological conditions as well as hypoxic exposure. These limitations require cautious interpretation and highlight the need for larger prospective studies using standardized protocols and harmonized qEEG outcomes.

Despite these limitations, this review provides the first focused synthesis of qEEG evidence describing the neurophysiological effects of intermittent hypoxia. The findings indicate that intermittent hypoxia consistently alters cortical oscillatory dynamics and functional brain network organization, although the direction and magnitude of these changes depend on the characteristics of hypoxic exposure. Future studies integrating qEEG with multimodal neuroimaging, molecular biomarkers, and cognitive assessment will be essential for clarifying the mechanisms underlying adaptive and maladaptive brain responses to intermittent hypoxia and for establishing electrophysiological biomarkers that may facilitate individualized therapeutic applications.

4. CONCLUSION

This systematic review demonstrates that intermittent hypoxia consistently induces measurable neurophysiological alterations in brain function, affecting both cortical oscillatory activity and large-scale functional network organization. The available evidence further suggests that these responses are highly dependent on the characteristics of hypoxic exposure, reflecting a continuum between adaptive neuroplasticity under controlled conditions and maladaptive changes associated with chronic pathological hypoxia. Overall, qEEG emerges as a sensitive and promising tool for characterizing hypoxia-induced brain responses and may contribute to future physiological monitoring, mechanistic investigations, and the development of targeted therapeutic strategies. Nevertheless, larger, well-designed studies employing standardized methodologies are required to strengthen the current evidence base and improve the clinical translation of these findings.

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AUTHOR CONTRIBUTIONS

SS: Conceptualization, Methodology, Literature Search, Data Curation, Formal Analysis, Writing – Original Draft, Visualization.

NM: Conceptualization, Methodology, Validation, Supervision, Writing – Review & Editing.

MIB: Methodology, Validation, Supervision, Writing – Review & Editing.

All authors have read and approved the final version of the manuscript.

CONFLICT OF INTEREST

The authors declare that they have no competing interests.

DATA AVAILABILITY STATEMENT

All data generated or analyzed during this study are included in this published article and its Supplementary Material.

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