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Electrophysiological indicators of neuroinflammation in multiple sclerosis: Emerging evidence.

Sofia Valdez*

Department of Neurophysiology, Pontificia Universidad Catolica de Chile, Chile.

***Correspondence to:** Sofia Valdez**,** Department of Neurophysiology, Pontificia Universidad Catolica de Chile, Chile, E-mail: s.valdez@puc.edu

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Introduction

Multiple sclerosis (MS) is a chronic autoimmune and neurodegenerative disorder characterized by demyelination and axonal damage within the central nervous system. One of the key pathological hallmarks of MS is neuroinflammation, which initiates and sustains the destruction of myelin sheaths and disrupts synaptic communication. Traditionally, magnetic resonance imaging (MRI) has been the gold standard for detecting MS lesions and monitoring disease progression. However, MRI is limited in its ability to provide real-time functional insights into the neurophysiological consequences of inflammation. In recent years, electrophysiological techniques such as electroencephalography (EEG) and event-related potentials (ERPs) have emerged as valuable tools for capturing subtle, dynamic changes in neural activity linked to neuroinflammation, thereby complementing structural imaging and enhancing diagnostic precision [1].

The presence of neuroinflammation in MS can alter neural oscillatory patterns and impair synaptic plasticity, both of which are reflected in EEG signals. Studies have shown that individuals with MS often exhibit increased theta and delta power and reduced alpha and beta activity, suggesting a general slowing of cortical rhythms. These abnormalities are believed to reflect disrupted thalamocortical communication, which is sensitive to inflammatory changes and demyelination. Moreover, the degree of alteration in resting-state EEG activity has been correlated with cognitive impairment, fatigue, and disease severity in MS patients. Importantly, these changes can occur even in the absence of new lesions on MRI, highlighting the potential of electrophysiological biomarkers to detect ongoing, subclinical neuroinflammatory processes [2].

Event-related potentials offer a more task-specific window into neuroinflammation-related functional deficits. Components such as the P300, N100, and mismatch negativity (MMN) have been used extensively to study sensory, attentional, and cognitive processes in MS. Among these, the P300 has shown particular promise as an indicator of cognitive dysfunction in MS patients. Reduced P300 amplitude and prolonged latency have been consistently reported in tasks involving attention and working memory, reflecting slowed information processing and diminished neural efficiency due to inflammation-related demyelination. Similarly, the MMN component, which reflects automatic detection of auditory changes, is often attenuated in MS, suggesting deficits in pre-attentive processing that

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may stem from altered neurotransmission and glial activation in auditory pathways [3].

Magnetoencephalography (MEG), with its superior spatial resolution compared to EEG, has further substantiated the role of electrophysiological markers in detecting neuroinflammatory changes in MS. MEG studies have demonstrated abnormal coherence and connectivity between brain regions, particularly in networks associated with sensorimotor and cognitive functions. Functional connectivity analyses have revealed that MS patients often display both hyperconnectivity and hypoconnectivity depending on disease stage and region of interest. These connectivity disruptions are thought to reflect compensatory mechanisms in early disease and eventual network breakdown in progressive stages. Importantly, alterations in MEG-derived network parameters such as node strength and clustering coefficient have been linked to levels of pro-inflammatory cytokines, thereby directly associating neurophysiological changes with the underlying inflammatory milieu [4].

In addition to providing insights into the pathophysiological mechanisms of MS, electrophysiological techniques are increasingly being evaluated for their prognostic and therapeutic monitoring capabilities. Longitudinal EEG and ERP studies have demonstrated that changes in electrophysiological markers can precede clinical relapses and may serve as early indicators of treatment response. For example, normalization of P300 latency and partial recovery of alpha power have been observed in patients responding to disease-modifying therapies, suggesting that electrophysiological indicators are sensitive to reductions in inflammatory activity. Furthermore, portable EEG systems are now being explored as tools for continuous home-based monitoring, potentially allowing for real-time assessment of disease dynamics and individualized treatment adjustments [5].

Conclusion

The growing body of evidence supporting electrophysiological indicators of neuroinflammation in multiple sclerosis underscores their potential as accessible, real-time, and functionally sensitive biomarkers. From detecting subtle changes in cortical rhythms to assessing cognitive and sensory processing deficits, EEG and MEG provide complementary insights into the neural disruptions caused by inflammatory processes. Their ability to capture dynamic changes over time makes them invaluable tools for early diagnosis, prognostication, and therapeutic monitoring. As technology advances and our understanding of neuroinflammatory mechanisms deepens, electrophysiological assessments are poised to become integral components of personalized care strategies in the management of MS.

References

1. Crozier S, Robertson N, Dale M. The psychological impact of predictive genetic testing for Huntington′ s disease: a systematic review of the literature. J Genet Couns. 2015;24:29-39.
2. Li HL, Zhang YB, Wu ZY. Development of research on Huntington disease in China. Neurosci Bull. 2017;33:312-6.
3. Li S, Lei Z, Sun T. The role of microRNAs in neurodegenerative diseases: A review. Cell Biol Toxicol. 2023;39(1):53-83.
4. Patil RS, Vyas SG, Quazi WT, et al. The gut microbiome in Huntington disease: A review. GSC biol pharm Sci. 2021;15(3):317-26.
5. Zhao T, Hong Y, Li XJ, et al. Subcellular clearance and accumulation of Huntington disease protein: a mini-review. Front Mol Neurosci. 2016;9:27.

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