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A NOVEL SENSORY WAVE (P25) IN MYELIN OLIGODENDROCYTE GLYCOPROTEIN-INDUCED EXPERIMENTAL AUTOIMMUNE ENCEPHALOMYELITIS MURINE MODEL

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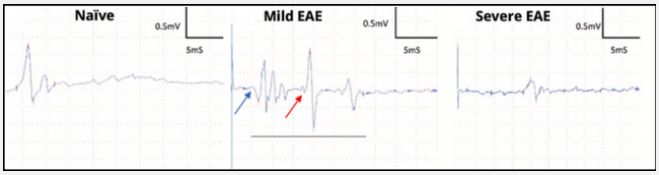
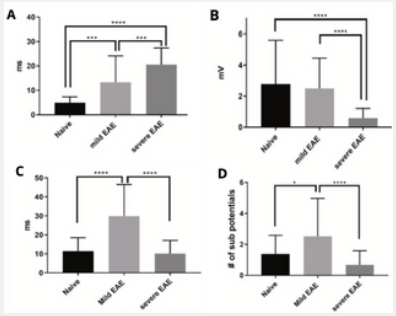
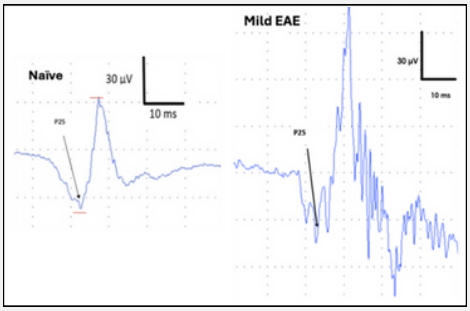
RESULTS



Motor evoked potentials: Characterization of the main tcMEP event in naïve and EAE diseased models.



Motor evoked potentials: (A) Mean group latency to the main event in naïve, mild disease, and severe disease animals. (B) Amplitudes of main event. (C) Mean group duration of the total event signal. (D) Mean group number of subpotentials recorded.



Sensory evoked potentials: The P25 amplitudes of MOG-induced EAE mice increased with mild disease (right) compared with naïve mice (left) indicating hypersensitivity at this stage of the disease.

OBJECTIVES

The myelin oligodendrocyte glycoprotein (MOG)-induced experimental autoimmune encephalomyelitis (EAE) model is characterized by chronic and progressive demyelination, leading to impairment of motor function and paralysis. While the outcomes of the disease, including impaired motor function and immunological changes, are well-characterized, little is known about the impact of EAE on the electrophysiology of the motor and sensory systems.

METHODS

In this study, we assessed evoked potentials as a quantitative marker for in vivo monitoring of nervous system damage. Motor-evoked potentials (MEPs) and sensory-evoked potentials (SEPs) were first standardized in naïve C57BL mice and studied thoroughly in EAE mice.

CONCLUSIONS

A sensory-evoked potential wave was identified in naïve animals that significantly increased in MOG-induced EAE animals with no or mild symptoms. The wave occurred 25 milliseconds post-stimulation. P25 was correlated with increased vocalization and was also reduced in amplitude following treatment with morphine. The results demonstrated that desynchronized neural motor activity, along with hypersensitivity in the early stages of EAE, leads to a complete loss of motor and sensory functions in the late stages of the disease.

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