**ELECTROPHYSIOLOGIC-PATHOLOGIC CORRELATIONS IN SEVERE MURINE EXPERIMENTAL AUTOIMMUNE NEURITIS**

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**ABSTRACT**

Introduction: Severe murine experimental autoimmune neuritis (sm-EAN) is a reliable animal model of Guillain-Barré Syndrome (GBS). Electrophysiological data and their correlation with neuromuscular severity scores are unknown. The proinflammatory chemokine expression is also unknown.

Methods: Sm-EAN was induced in 8-12 week old female SJL/J mice using bovine peripheral nerve myelin. Motor nerve conduction studies (MNCS; right dorsal caudal tail (DCT) and bilateral sciatic nerves) were performed on days 10, 30 (peak), 37 and 62 post-induction (p.i.). Sciatic nerves were harvested for histology, with chemokine ligand/receptor expression determined using immunohistochemistry and/or polymerase chain reaction.

Results: MNCS showed statistically significant reductions in mean compound motor action potential (CMAP) amplitudes and conduction velocities and increases in mean CMAP durations in both nerves compared to controls. Regression analyses demonstrated correlations ($r^2=0.78-0.99$) between neuromuscular severity scores and the above parameters, with stronger correlations seen with the DCT nerve. Severe multifocal or diffuse demyelination with some axonal loss was associated with F4/80+ macrophages, CD3+ CD4+ T-cells and CD19+ B-cells. There was increased expression of CCL2-CCR2, CCL5-CCR1, CCR5 and CXCL10-CXCR3 relative to controls at peak severity.

Conclusions: The electrophysiological and pathologic features of sm-EAN correlate and significantly mimic GBS.