MicroRNA-338 stimulates nerve function recovery in rats with experimental autoimmune neuritis by inhibiting the conversion of Schwann cells into inflammatory cells

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Objective: To explore the mechanism of microRNA-338 on the recovery of neurological function in rats with experimental autoimmune neuritis

Methods: We used microRNA-338 coded lentivirus vectors in a Lewis rat EAN model, in conjunction with the P0 peptide 180-199 which was injected into the footpads of animals to induce immunization. Electrophysiology, ultrastructure and immunofluorescence histopathology were measured at the neuromuscular severity peak and recovery platform period(day 42) post-immunization. Cell-specific protein markers were used for immunofluorescence histopathology staining to characterize sciatic nerve cells: Iba-1, S100 (myelin), and neurofilament 200 (axon). All rats were weighed and scored daily until day 42 post-immunization(p.i.).

Results: The clinical scores of microRNA-338 and intravenous immunoglobulin (IVIg) groups were significantly superior to those of the untreated group at disease peak ($p < 0.05$). The clinical scores of microRNA-338 and IVIg groups were also significantly superior to those of the untreated group at disease plateau ($p < 0.05$). The nerve conduction velocity of microRNA-338 and IVIg groups increased significantly compared to that of the untreated group at disease peak ($p < 0.01$). The compound nerve action potential amplitude of microRNA-338 and IVIg groups increased significantly compared to that of the untreated group at disease peak ($p < 0.01$). At disease peak, myelin swelling, cavity formation, and lamellae separation showed improvement in microRNA-338 and IVIg groups compared to the untreated group. S100 and NF200 expression in microRNA-338 and IVIg groups increased

compared to that in the untreated group. Iba1 and S100 co-expression in Schwann cells in microRNA-338 and IVIg groups decreased compared to that in the untreated group, which was indicative of the reduced conversion of Schwann cells into inflammatory cells.

Conclusion: In conclusion, overexpression of microRNA-338 in sciatic nerves can alleviate symptoms and improve neuromuscular function in rats with EAN by inhibiting the conversion of Schwann cells into inflammatory cells.

Keywords: microRNA-338 • experimental autoimmune neuritis • Schwann cell • sciatic nerve • myelin ultrastructu