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Ph.D. DISSERTATION DEFENSE

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Title: Uncovering the Mechanism of Electrical Stimulation in Schwann Cells for Nerve Injury Repair

Chairperson: Dr. Hongjun Wang, Department of Biomedical Engineering, School of Engineering and Science

Committee Members: Dr. Kenny Wong, Department of Chemistry and Chemical Biology, School of Engineering and Science
Dr. Marcin Iwanicki, Department of Chemistry and Chemical Biology, School of Engineering and Science
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ABSTRACT

Peripheral nerve injuries affect over 20 million people worldwide each year, often resulting in debilitating functional impairments due to the limited and inconsistent capacity for regeneration. Peripheral nerve regeneration relies critically on Schwann cells (SCs), which undergo a transient repair-state transition following injury to support axonal regrowth. Electrical stimulation (ES) has emerged as a promising strategy to enhance regeneration, yet the underlying cellular mechanisms remain poorly defined. Here, we developed a sterile, high-throughput electrical stimulation platform compatible with standard 96-well plates to deliver uniform, reproducible direct current (DC) electric fields in vitro. Using this system, we investigated how ES influences calcium signaling, gene expression, and migration. Live-cell imaging revealed rapid, voltage-dependent calcium influx in primary rat SCs, with responses approximately 65% higher at 200 mV/cm compared to sham controls, and dependent on intact microtubule dynamics. RNA sequencing identified ES-induced transcriptional programs related to cytoskeletal regulation, stress response, and injury signaling, including differential regulation of *Map1b*. Targeted qPCR further confirmed time-dependent expression of injury-responsive genes such as c-*Jun*, *Serpinf1*, *p75NTR*, *Atf3*, *Sox2*, *Thbs2*, and *Map1b*, consistent with transient repair-state activation. Functionally, ES enhanced SC migration in vitro, an effect inhibited by microtubule disruption but preserved when treated with a mitotic blocker, indicating a migration-driven rather than proliferation-driven response. Together, these findings demonstrate that ES activates calcium-dependent, cytoskeleton-mediated signaling pathways in Schwann cells, promoting a pro-regenerative repair phenotype. This work establishes a mechanistic in vitro framework for studying ES-driven nerve repair and highlights its therapeutic potential to improve regeneration outcomes.