

## **Webinar**

### **Molecular strategies to promote Spinal Cord repair: Barriers and Solutions**

**Ishwariya Venkatesh**

**Marquette University, WI, USA**

Embryonic and peripheral neurons respond to Spinal Cord Injury with activation of transcriptional networks conducive to re-growth, achieving full recovery. In contrast, injured mature CNS neurons fail to re-induce appropriate transcriptional networks, resulting in failed regeneration and permanent damage. We have previously shown that forced re-expression of single transcription factors (TFs) such as KLF6 partially re-activates pro-growth transcriptional networks and promotes axon outgrowth following injury. While this is a promising strategy for therapeutic neural repair, full functional recovery requires an increase in overall number and regenerative speed of axons. What could be preventing full efficacy of transcription factor treatments? I have identified three neuron-intrinsic barriers that dampen effects of transcription factor treatments preventing full recovery. These barriers are (1) Lack of availability of activating TFs (2) Presence of repressive TFs (3) Epigenetic constraints. By reconstructing the transcriptional regulatory landscape in regeneration competent embryonic neurons, I have identified targeted gene candidates to override intrinsic barriers and achieve full recovery in injured mature CNS neurons. These gene candidates will be screened in single-cell assays of gene expression and chromatin accessibility followed by functional testing in in-vivo mouse models of Spinal Injuries. Overall this research is expected to identify novel therapeutic targets to improve regenerative outcomes following Spinal Cord Injury.

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***5:30 PM***