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The role of exosome-mediated Adiponectin in Central Nervous system injury repair: investigating the molecular mechanisms of mRNA regulation in neuroregeneration

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**Abstract**: miR-133b is a microRNA that plays an important regulatory role in nerve regeneration. Exosomes are small vesicles produced by cells that can carry a variety of bioactive molecules, including miRNAs. Adiponectin is a multifunctional protein hormone that can be transmitted to damaged nerve tissue by exosomes. The relationship between these elements is related to the mechanism of action of miR-133b and adiponectin in nerve regeneration mediated by exosomes.

**Objective**: Nerve regeneration is a key process in the repair of central nervous system injury. The purpose of this study was to investigate the role of miR-133b, exosomes and adiponectin in nerve regeneration, focusing on their interrelationships and their effects on neuronal growth and connection reconstruction.

**Methods and Materials**: Animal models utilized rat neuronal cells. Exosomes were isolated from the culture medium via ultracentrifugation. Exosome morphology and size were validated using TEM and NTA, with surface markers confirmed by Western blotting. Rat neuronal cells were exposed to exosomes to assess impact on miR-133b expression and neuronal connectivity, analyzed through immunofluorescence and qRT-PCR.

**Results**: The exosomes were observed by TEM, and the diameter of the exosomes was 30-150 nm round doublelayer vesicles. NTA showed that the particle concentration of the exosomes was between 107 to 1013 Particles/ ml. The expression of CD63, CD81 and CD9 proteins was detected by Western blotting. qRT-PCR results showed that the expression level of miR-133b in serum exosomes in the model group was significantly lower than that in the normal group (P < 0.05). Immunohistochemical staining showed that the axonal myelin structure was clearly visible in the normal group, the neuronal cells in the abdominal horn of the spinal cord were arranged neatly, and the Nissl body were evenly distributed. In the model group, the neuronal axons were demyelinated, the neuronal cells were deformed and ruptured in the ventral horn of the spinal cord, and the number of Nissl body decreased. The difference quantity of Nissl body between the two groups was statistically significant (P<0.05). Immunofluorescence and Western blotting results showed that the expressions of neurofilament protein (NF), myelin basic protein (MBP) and glial fibrillary acidic protein (GFAP) in the model group were significantly lower than those in the normal group. The difference was statistically significant (P<0.05).

**Conclusion**: miR-133b, transferred by exosomes, along with the involvement of adiponectin, exerts a crucial regulatory role in neuronal growth and synaptic reconstruction during neuroregeneration. These findings offer potential avenues for research and therapeutic strategies in neural injury repair and regeneration.

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## Biography

Paul Dreschnack is a Plastic Surgeon in New York City interested in advanced cellular medicine research. Trained in reconstructive microsurgery and hand surgery, he authors papers on stem cell research and extracellular vesicle medicine research.

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