

ACADEMIC LECTURE

RECENT KNOWLEDGE ON NEUROPLASTICITY

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Treatment of neurological disease has traditionally focused on neuroprotection, reducing lesions of the central nervous system (CNS). To a large extent, this approach has failed and few if any neuroprotective treatments are clinically available for neural injury and neurodegenerative disease. A far more promising approach would be to target, not the lesion, but the intact CNS, with the objective to remodel the CNS tissue so as to compensate for the CNS injury or degenerative disease. After a neural injury, intrinsic CNS restorative mechanisms are activated, but they infrequently are capable of completely restoring neurological function. Here, I will describe ways to amplify endogenous restorative processes post neural injury, using cell-based and pharmacological therapies. I will describe the means by which neurorestorative therapeutic approaches amplify angiogenesis, neuroge-

nesis, neurite outgrowth, and oligodendrogenesis, and how these restorative events interact and enhance neurological outcome post injury. For ease and clarity of description, I will describe our work on neurorestoration using cell-based therapy in experimental stroke as the model of injury. Data will also be provided demonstrating that exogenously administered cells activate parenchymal cells, primarily astrocytes, to express trophic factors as well as select proteases that promote neurological recovery. In addition, I will describe a novel molecular pathway by which the administered exogenous cells communicate with and alter the surrounding parenchymal cells to activate restorative events. This presentation will thereby emphasize our ability to stimulate endogenous restorative processes, present even in the aged brain, and thereby to enhance neurological function.