

Neuroplasticity of the brain and recovery after central nervous system damage

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Annotation:

Neuroplasticity is the central biological mechanism supporting functional recovery after damage to the central nervous system (CNS), including stroke, traumatic brain injury, spinal cord injury, and demyelinating disorders. Following injury, the brain and spinal cord undergo multi-level reorganization: synaptic efficacy changes (LTP/LTD), structural remodeling of dendrites and axons, and large-scale network and cortical map reweighting. Recovery is influenced by time-dependent “windows” of heightened plastic potential, especially during the subacute period, when rehabilitation can most effectively shape adaptive rewiring. However, plasticity is not always beneficial; maladaptive changes may contribute to spasticity, chronic pain, compensatory overuse, and learned non-use.

Keywords

neuroplasticity; central nervous system injury; stroke recovery; traumatic brain injury; spinal cord injury; synaptic plasticity;

Neuroplasticity is the brain’s and spinal cord’s ability to change their structure and function in response to experience, learning, or injury. After central nervous system (CNS) damage—such as stroke, traumatic brain injury, or spinal cord injury—recovery depends not only on healing of the damaged tissue (which is often limited), but also on the nervous system’s capacity to reorganize surviving circuits and build alternative pathways for lost functions. Evidence supports

rehabilitation strategies that are intensive, task-specific, and meaningful (salient), using repetition and progressive challenge to drive use-dependent circuit strengthening. Emerging neurotechnology's—robotic therapy, virtual reality, and non-invasive brain stimulation—aim to amplify training-induced plasticity, but clinical outcomes depend on patient factors (lesion location/size, age, comorbidities), therapy dose, and engagement. Understanding the mechanisms and limits of neuroplasticity helps design personalized rehabilitation protocols that maximize functional gains while minimizing maladaptive outcomes. This reorganization happens at multiple levels: synapses can become stronger or weaker, networks can “rewire” to recruit new regions for a task, and surviving neurons can form new structural connections to compensate for disrupted pathways.

Conclusion

Neuroplasticity provides the neurobiological foundation for recovery after CNS damage through synaptic, structural, and network-level reorganization. The greatest improvements typically occur when rehabilitation leverages time-sensitive plasticity, emphasizing high-repetition, task-specific, and motivating practice. Because plasticity can be adaptive or maladaptive, effective recovery requires guiding reorganization toward functional patterns and preventing inefficient compensations. Future progress will likely come from personalized rehabilitation dosing combined with targeted neurotechnology and biomarker-informed monitoring of plastic change.