

Module 9 – Neuroplasticity

Structural Plasticity

Structural plasticity is observed after an SCI in many locations. Axons that have been spared from injury can branch out and make connections with neurons they previously were not connected to, which can help restore motor abilities

Injured axons can sprout branches and connect to other neurons such as interneurons (with cell bodies located within the brainstem or spinal cord) and can form detours to reroute information around an SCI lesion.

Axon collaterals that form after an SCI can also make more connections onto the same original neuron it is already connected to, in order to provide a stronger signal, for example to make up for loss of other cells that provided input.

Remodelling of axons occurs throughout the neuroaxis after an SCI. Evidence of structural plasticity that results in functional adaptations has been observed above and below an SCI lesion, as well as within the brain and brainstem.

Below the spinal cord injury, axons from upper motor neurons and interneurons can form new connections to connect to targets that have lost input due to the SCI.

This can contribute to recovery. Similarly, axon sprouting between sensory neurons and interneurons can affect the perception of pain.

Rewiring of upper motor neurons onto interneurons above a lesion can form complicated relays that re-route descending information through networks of spared circuitry to aid in functional recovery. Finally, the connectivity of neurons in the brain undergoes reorganization to make better use of what circuitry remains. Plasticity at every level can potentially be enhanced for therapeutic gain.

Structural plasticity also occurs within the brain after an SCI. Two regions of the brain called the motor cortex and sensory cortex are essential to producing movement and interpreting sensory information.

After an SCI the ability to generate movement and feel below the injury become disrupted. Both the motor cortex and sensory cortex are specifically organized in a manner such that one region of the brain is responsible for controlling one muscle, or generating sensation of one area of the body. This organization is termed somatotopic organization

After an SCI the regions of the brain that no longer respond to sensation or cannot produce movement can become quiet and no longer in use. Further, other regions can become more active to compensate for new and unique functional demands that are required after an SCI.

Regions of the brain that now require more activity can reorganize to make use of the neuronal circuitry that would otherwise be dormant caused by SCI. This “cortical reorganization” rewires the brain to adapt circuitry to best improve functions.

However, just as maladaptive plasticity within the spinal cord can drive secondary complications such as pain, maladaptive cortical reorganization in the sensory cortex may also contribute to pain or phantom limb syndrome.

How neurons re-connect to one-another after an SCI, and the consequences of that re-arrangement, is still being investigated. Structural plasticity underlies many known positive adaptations that occur after SCI leading to improvements in sensory abilities and motor function.

However, plasticity is also thought to underly several maladaptive adjustments including the development of neuropathic pain, autonomic dysreflexia, and spasticity caused by sensory neurons branching and making inappropriate connections within the spinal cord.