Neuroscience underlying rehabilitation: what is neuroplasticity?

Neuroplasticity generally refers to the capacity of neurons and neural networks to change their connections and behaviour in response to experience. However, can therapy induce neuroplasticity? And does neuroplasticity contribute to developmental rehabilitation? Affirmative answers to these questions have become the tenets of therapy and rehabilitation, for which they provide a mechanistic understanding, research framework, and thereby, growing evidence base that can justify specific practices. Various behavioural, neurophysiological, and neuroimaging methods can document changes in the nervous system. Anatomical, histological, biochemical, and gene expression evidence is also available. But the resulting picture is blurred, owing to the variety of phenomena termed 'neuroplasticity', and lack of a clear definition and general theory.

It may be useful to distinguish between functional and structural neuroplasticity. The former relates to changes in physiological functioning of neurons, for example, in excitability, response level, or synchronicity within neural networks. But the duration of the effect suggests that not all functional plasticity has direct relevance to rehabilitation. Short-term synaptic plasticity corresponds to an increased or decreased probability of neurotransmitter release of less than a few minutes (possibly just tens of milliseconds), through neural facilitation, synaptic augmentation, or synaptic fatigue or depression. Other mechanisms result in long-term synaptic potentiation or depression lasting for hours or weeks, possibly even years in selected cell populations. Synaptic efficacy can depend on the history of their activity, relating to experience in daily life (potentially enhanced by rehabilitation), whether physiological or pathological (as in dystonia or epilepsy). This contributes to storing information in the nervous system. For example, cortical representations of body parts can be altered by experience. Such remapping can potentially be facilitated by rehabilitation or brain stimulation.[1,2]

Intense synaptic plasticity occurring in dendritic spines establishes an important link between functional and structural neuroplasticity. Dendritic spines thus shape developmental trajectories, learning and adapting to existing or new conditions.[3] Early in development, dendrites have relatively few spines. Subsequent molecular processes result in the formation of many spines, which then undergo changes in structure and function, sculpting the individual's nervous system connectivity, largely as a result of synaptic use through their experiences.

On a wider scale, structural neuroplasticity involves regional volume changes or the formation of new neural pathways, through synaptogenesis, axonal or dendritic sprouting, changes in myelin, or production of new neurons. The latter was long held to be impossible in the human brain beyond infancy (in contrast to other mammals), but the generation of neurons, whose function remains to be further clarified, has now been documented in several parts of the adult human brain.[4]

Yet, more relevant mechanisms to neurodevelopmental disorders and rehabilitation likely involve strengthening of previously silent, hardly-used connections (e.g. ipsilateral corticospinal tract spinal projections), through long-term synaptic potentiation, production of new synapses, fibre sprouting, and myelin changes.[5] Current understanding of neuroplasticity implies that synapses, fibres, and cells are overproduced early in maturation and subsequently compete for survival and efficacy within functional networks. Local (in)activity induces local plasticity and also widespread network changes, as predicted by an activity-based model, which also provides arguments for promotion of self-initiated activities, experiences, and intervention provided closer to the time of injury and during sensitive periods.[1,3,5] Alternative pathways after brain injury are typically less efficient, though therapy can help strengthen them and enhance their function.

But what makes an experience biologically meaningful? How does experience induce modifications at different levels of neural (and neuroglial) organization? What is the potential for long-term changes to refine therapy and engineer signalling devices to effectively promote improved functioning through neuroplasticity? Answers to these questions are required for further understanding of how neuroplasticity is achieved, and can help rehabilitation.

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