Editorial
Neuroplasticity in the Pathology of Neurodegenerative Diseases

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The phenomenon of neuroplasticity was first described about 50 years ago. Neuroplasticity is a primary property of the nervous system that has been widely observed in both physiological and pathological conditions. Neuroplasticity in physiological conditions includes developmental plasticity, learning and memory, compensatory plasticity, and repair of the adult brain. Neuroplasticity in pathological conditions include plasticity after injury and removal of brain tumor, stroke, epilepsy, and neurodegenerative diseases such as Alzheimer’s disease, Parkinson’s disease, and Huntington’s disease. The current special issue is primarily concerned with the pathogeneses of common neurodegenerative diseases and their effects on neuroplasticity.

It is known that the central changes that occur during a stroke vary widely. Regeneration after ischemic injury and recovery of lost functions can be attributed to the plasticity of neurons which enable their ability to reorganize and rebuild the brain. These neuroplastic changes which include gene expression and possession of specific genetic variants are influenced by environmental factors and epigenetic mechanisms. Neuro-, glio-, and angiogenesis processes, molecular changes occurring after stroke such as synaptic plasticity and axon growth, are discussed in the article by J. Dąbrowski et al.

Many rare diseases are due to disorders at the genetic level such as Waardenburg syndrome (WS). WS is one of the most common forms of deafness with heterogeneity of loci and alleles and variable expression of clinical features. In this issue, W. Li et al. described fourteen mutations in Chinese patients with WS, including c.808C>G, c.117C>A, c.152T>G, c.803G>T, c.793-3T>G, and c.801delT on PAX3; c.642_650delAAG on MITF; c.122G>T and c.127C>T on SOX10; c.230C>G and c.365C>T on SNAI2; and c.481A>G, c.1018C>G, and c.1015C>T on EDNRB. Importantly, three CNV variants were de novo and five EDNRB mutations were first associated with WS type 1 heterozygous, with a detection rate of 22.2%. These studies suggest that EDNRB mutations should be considered as another common pathogenic gene in WS type 1. More information can be found in the articles by W. Li et al.

At the cellular level, synaptic plasticity plays an important role in maintaining neuronal connections. Synaptic plasticity leads to changes in the structure of synapses and dendritic spines, synaptogenesis, and axonal modification. Disturbed contact between neurons leads to senile changes and elderly diseases such as dementia. Common causes of dementia include Alzheimer’s disease, vascular dementia, Parkinson’s disease dementia, dementia with Lewy
Neural Plasticity

Neuroplasticity not only rebuilds brain function but also repairs developmental disorders and is responsible for learning and memory. It appears in response to various internal and external insults and factors, including age-dependent neurodegenerative diseases and pharmacological treatment.

It is known that the repair of brain damage depends on the degree and extent of central lesions. This is an important consideration in the neurorehabilitation and neuropsychological treatment of patients with central nervous system disorders like Alzheimer’s disease. Recent studies suggest that intensive and cognitively demanding physical exercise programs are capable of inducing plastic brain changes in Parkinson’s disease. The article by E. Palasz et al. elaborates on physical training that provides protection for dopaminergic neurons in rodent Parkinsonism models produced by neurotoxins.

We should also mention innovative therapeutic methods to improve neuroplasticity such as deep brain stimulation (DBS). DBS is a recognized therapy for Parkinson’s disease. There is evidence that DBS exerts dopaminergic neuroprotection in animal models with disease-modifying effects. Rodent studies have shown an increase in cholinergic neurotransmitters, hippocampal neurogenesis, synaptic plasticity, and reduction of β-amyloid plaques with DBS. The effectiveness of DBS is currently being evaluated in patients with mild Alzheimer’s disease 65 years of age and older.

Finally, with better understanding of brain plasticity mechanisms in neurodegeneration, more effective therapies and improvement in the quality of life for neurological patients can be achieved.

Conflicts of Interest

The editors declare that they have no conflicts of interest regarding the publication of this special issue.

Authors’ Contributions

The authors of the Editorial are guest coeditors of this special issue.

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