



Editorial

Neurodegenerative Diseases: Molecular Mechanisms and Therapies

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Neurodegenerative diseases are characterized by the progressive degeneration or death of neurons in the central or peripheral nervous system. Genetic, environmental, and lifestyle factors all contribute to neurodegenerative diseases [1,2]. Although some neurodegenerative diseases can be managed with treatments, and the progression of the disease may be slow, many remain incurable [3]. Common underlying processes contribute to the degeneration of neurons, but the molecular mechanisms of neurodegenerative diseases are complex and varied, and they can differ depending on the specific conditions [4]. Neurodegenerative processes and neuronal disorders also occur in the spinal cord [5], retina [6], and enteric nervous system [7], eventually reflecting and/or affecting what occurs in the brain. The consequences of these conditions are often a gradual decline and abnormality in perceptual, cognitive, motor, behavioral, and social abilities. Neurodegenerative diseases pose a significant public health threat. These diseases commonly occur in elderly individuals and their prevalence increases with age. The increasing lifespan and decreasing fertility rate lead to an increase in the median age prevalence of these disorders and aggravated health and economic burdens to society.

Long-lived people have a higher concentration of the longevity-associated variant (LAV) homozygous genotype in member 4 of the bactericidal/permeability-increasing fold-containing family B (BPIFB4) gene [8]. It was previously shown that LAV-BPIFB4 prevented Huntington's disease (HD) progression in mice [9]. The production of mutant Huntingtin protein, which exhibits pathological lengthy polyglutamine repeats and toxic consequences, results in HD. In this context, there is a substantial clinical need yet unmet, as there are currently no clinically validated treatment medicines or therapies that can slow or stop neurodegeneration and disease progression in HD. In this Special Issue, Cattaneo et al. demonstrate that LAV-BPIFB4 generates neuroprotection in the striatum-derived STHdh cell line, an in vitro model of HD [10].

The use of mutant cell lines enables the study of the underlying pathophysiological processes causing neurodegeneration, such as oxidative stress. Besides the NADPH oxidase 2 (NOX2/gp91) gene [11,12], other NADPH oxidase family members (NOX 1, 3–5, DUOX1, and DUOX2) have been implicated in the production of reactive oxygen species (ROS) [13]. Interestingly, the NADPH oxidase organizer 1 (NOXO1) is essential for the organization and formation of the NOX1-dependent NADPH oxidase complex. Benssouina et al. demonstrate that, in a mutated NOXO1 colorectal cancer cell line with mutated D-box, there is an increase in ROS production and cytotoxicity through NOX1 activity, affecting the mitochondrial organization. These effects were observed with the translocation of the mutated protein from the soluble membrane fraction to a cytoskeletal insoluble fraction [14]. The D-box of NOXO1 protein seems to maintain the balanced distribution of Noxo1 between



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Molecular Mechanisms Of Neurodegenerative Diseases

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