iMedPub Journals www.imedpub.com

Molecular Enzymology and Drug Targets

ISSN 2572-5475

2021 Vol.7 No.3:e009

Potential Drug Targets for Neurodegenerative Diseases

Leo Tolstoy*

Bio Discovery Institute, School of Chemistry, University of Nottingham, University Park, Nottingham, UK Corresponding author: Leo Tolstoy, Bio Discovery Institute, School of Chemistry, University of Nottingham, University Park, Nottingham, UK Received date: December 7, 2021; Accepted date: December 21, 2021; Published date:December 28, 2021 Citation: Tolstoy L (2021) Potential Drug Targets for Neurodegenerative Diseases. Mol Enzy Drug Targ Vol. 7 No.3:e009

Description

Alzheimer's disease (AD) and Parkinson's disease (PD) are two NDs with similar pathologies that could be treated by controlling PREP levels. The NEUROPRO project brought together eight academic institutes and three small and medium-sized companies (SMEs) to provide the skills required to transform research into commercial technologies for clinical use.

Significant progress was made during the course of the fouryear project. PREP function and interaction with other proteins and cytoskeletal components were studied using a variety of approaches including immunofluorescence double labelling, confocal laser scanning microscopy, and peptidomics.

Researchers discovered that adjusting PREP levels to avoid learning and memory loss will not work since neurodegeneration and neuroinflammation disrupt multiple important processes. PREPs have been connected to intracrine and endocrine activities, as well as intracellular and extracellular processes like signalling and secretion and brain plasticity. PREP inhibition with Z-Pro-Prolinal (ZPP) or KYP-2047 provided innovative medications for ND therapy based on structural analysis, PREP inhibition, and peptidomics.

PREP is required for the processing of amyloid precursor protein, according to cellular models (APP). AD may be caused by abnormal changes in PREP interaction with tau proteins, as well as defective APP metabolism or clearance.

PREPs changed the aggregation patterns of alpha-synuclein, which promote plaque formation and neurodegeneration. This was clearly proven in animal and cellular models of Parkinson's disease, with PREP inhibitors significantly reducing alphasynuclein plaque density. PREP and alpha-synuclein colocalization in genuine PD brains supported key findings.

Alzheimer's Disease

Alzheimer's disease (AD) is a chronic neurodegenerative illness that causes the loss of neurons and synapses in the cerebral cortex and some subcortical structures, resulting in temporal lobe, parietal lobe, frontal cortex, and cingulate gyrus atrophy. It is the most frequent kind of neurodegeneration. Despite billions of dollars spent on research, no effective cures for Alzheimer's disease have been discovered. Clinical trials, on the other hand, have generated specific molecules that could transform the future of Alzheimer's disease therapy.

Parkinson's Disease

The second most common neurodegenerative ailment is Parkinson's disease (PD). Bradykinesia, stiffness, resting tremor, and posture instability are common symptoms. The prevalence rate of Parkinson's disease has been reported to range from 15 per 100,000 to 12,500 per 100,000, with an incidence rate of 15 per 100,000 to 328 per 100,000, with Asian countries having a lower prevalence rate.

The death of dopaminergic neurons in the substantia nigra, an area of the midbrain, is the hallmark of Parkinson's disease. The cause of this cell death that is selective is unknown. Within afflicted neurons, alpha-synuclein-ubiquitin complexes and aggregates have been found to accumulate in Lewy bodies. Defects in protein transport machinery and regulation, such as RAB1, are thought to be involved in the disease pathogenesis. Alpha-synucle in buildup in Lewy bodies may be caused by impaired axonal transport.

Huntington's Disease

Huntington's disease (HD) is a neurological condition caused by abnormalities in the huntingtin gene (HTT). Loss of medium spiny neurons and astrogliosis are hallmarks of HD. The striatum is the first brain region to be significantly impacted, followed by degeneration of the frontal and temporal cortices. Subthalamic nuclei in the striatum give control impulses to the globus pallidus, which begins and controls motion.

As a result of the decreased impulses from the subthalamic nuclei, movement initiation and modulation are diminished, resulting in the disorder's signature movements, particularly chorea. Huntington's disease appears later in life, despite the fact that the proteins that cause the disease work towards manifestation in persons afflicted by the proteins from an early stage. HD has been linked to neurodevelopmental issues in addition to being a neurodegenerative condition.