Chapter 16
Astrocytes Reprogramming for Neurodegenerative Disease Management

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ABSTRACT

Neurodegenerative disorders (NDD) are chronic conditions that lead to nerve cells degeneration in the central nervous system. The most common NDD are Alzheimer's disease (AD), Parkinson's disease (PD), amyotrophic lateral sclerosis (ALS), multiple sclerosis (MS), and Huntington's disease (HD). Parkinson's disease (PD) is recognized as the second most common neurodegenerative disease after Alzheimer's

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disease (AD) and Huntington disease (HD). Astrocytes reprogramming has been recently proposed as one of the most important treatments to stop the progression of these neurological diseases. By targeting the microRNA, astrocytes in the animal brain could be reprogrammed into various functional neural systems. Activation of endogenous genes could be a revolutionary therapeutic approach for treating neurological diseases. Reprogrammed astrocytes could also be used to generate various types of neurons lost in case of Huntington's disease, Parkinson's disease, and Alzheimer's disease. The ability to reprogram astrocytes into functional neurons was classified as a new advancement for PD, AD, and HD treatments.

INTRODUCTION

Neurodegenerative diseases (NDD) are serious, chronic conditions that damage and destroy the nerve cells of the central nervous system, affecting millions of people worldwide. The most common neurodegenerative diseases are Parkinson's disease (PD), Alzheimer disease (AD) and Huntington's disease (HD). Parkinson's disease is a progressive neurodegenerative disorder characterized mainly by the motor features (Jankovic, 2008). Impairment of voluntary motor control leads to signs of bradykinesia, rigidity, postural instability and tremor. Additionally, the disease is characterised by the presence of other non-motor symptoms (El-Mansoury et al., 2023). Alzheimer's disease is a neurodegenerative disorder with an obvious degeneration of the neuronal cells. In order to clarify the mechanisms controlling AD progression, two hypotheses in general were proposed; the deposition of beta-amyloid (Aβ) protein between neurons (Hamd-Ghadareh et al., 2022; Hu et al., 2022), and the presence of neurofibrillary tangles (NFT) accumulation within the neurons (Kim and Park, 2020; Schneider et al., 2022). In addition, recent studies suggest that environmental factors including metal ions play an important role in disease pathogenesis (Lee et al., 2018; Tian et al., 2022; Z. Zhou et al., 2022). Huntington's disease (HD) was defined for the first time by Georges Huntingtonin 1872. The neuropathology of HD characterised by a dysfunction and death of specific neurons within the brain. The prevalence of Huntington's disease is about 5 cases per 100,000 individuals. The frequency of occurrence is the same for both men and women. A study revealed that motor, cognitive, and behavioral dysfunction disorders are all impacted by HD (Zuccato and Cattaneo, 2014).

At the moment, symptomatic treatment is in general; the only therapeutic approach for AD. This kind of treatment was based on using acetylcholinesterase (AChE) inhibitors to enhance ACh bioavailability at the synaptic area (Pasandideh and Arasteh, 2021; Richter et al., 2018). Food and Drug Administration (FAD) approved recently five molecules (donepezil, galantamine, rivastigmine, memantine and memantine combined with donepezil). A six drug aducanumab, under FDA review waiting for potential approval (Wiley, 2021). In addition various treatment were used for treatment of PD and HD, but all these current treatment approaches of NDD still limited then a new therapeutic approach based on reprogrammed astrocytes could be a potential treatment for these disorders.

Various studies revealed the application of reprogrammed astrocytes for treatment of NNDs such as PD (Dopamine), AD (Glutamate and Acetylcholine) and HD (GABA) (Guo et al., 2014) **(Figure 1)**. Astrocytes are mainly reprogrammed into glutamatergic neurons, whereas NG2 cells can be reprogrammed into both glutamatergic and GABAergic neurons. Such different cell fates after reprogramming by the same transcription factor may provide important clues regarding the lineage relationship between neu-

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