

Chemical Engineering Master's Defense

Toxicity study in Alzheimer's disease cell model

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abstract

Alzheimer's disease (AD) is the most common type of dementia, affecting one in nine people age 65 and older. One of the most important neuropathological characteristics of Alzheimer's disease is the aggregation and deposition of the protein beta-amyloid. Beta-amyloid is produced by proteolytic processing of the Amyloid Precursor Protein (APP). Production of beta-amyloid from APP is increased when cells are subject to stress since both APP and beta-secretase are upregulated by stress. An increased beta-amyloid level promotes aggregation of beta-amyloid into toxic species which cause an increase in reactive oxygen species (ROS) and a decrease in cell viability. Therefore reducing beta-amyloid generation is a promising method to control cell damage following stress.

The goal of this project was to test the effect of inhibiting beta-amyloid production inside stressed AD cell model. Increase in reactive oxygen species and decrease in cell viability were observed after addition of hydrogen peroxide to AD cell model. The increase in stress induced toxicity caused by addition of hydrogen peroxide was dramatically decreased by simultaneously treating the cells with iBSec1 or DIA10D to block the increase in beta-amyloid levels resulting from the upregulation of APP and beta-secretase.



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