OPEN ACCESS Freely available online

Translational Medicine

ISSN: 2161-1025

Commentary

A Brief Note on Regulation of Oxidative Stress and Cell Damage Caused by Aβ142

Mark William*

Department of Neurology, IU Health Neuroscience Center, Indianapolis, USA

DESCRIPTION

Alzheimer's Disease (AD) is a degenerative condition that commonly affects elderly people and is marked by dementia and deteriorating memory. The treatment of AD has become crucial for public healthcare systems due to increased longevity worldwide. Although some AD medications have been authorized for sale, AD still has an enigmatic pathogenesis and no effective treatments that oxidative stress may be a key factor in the pathogenesis, where microRNA-668-3p acts downstream of OXR1 to affect p53-p21 signaling and mediate the oxidative stress induced by Amyloid Beta-peptide (Aβ) Aβ1-42, thereby alleviating AD progression.

Hematoxylin-eosin (HE) staining, immunohistochemistry, and Immunofluorescence (IF) were used to analyze of the characteristics the hippocampus, neuron cell separation, and related protein expression. Quantitative Reverse Transcription Polymerase Chain Reaction (RT-qPCR) and western blot analysis were used to examine the changes in microRNA and mRNA levels before and after amyloid (A) 1-42 induction, and Gene Expression Omnibus (GEO) data analysis was used to monitor microRNAs and mRNAs that affect AD progression. In addition, luciferase analysis is used to examine microRNA and mRNA binding and the effect of microRNA/mRNA interaction on neuronal cell proliferation. Using the Cell Counting Kit-8 analysis and Flow Cytometry (FCM) techniques, the levels of apoptosis and Reactive Oxygen Species (ROS) were evaluated. Through the interaction of microRNA and mRNA, oxidative stress-related protein levels secreted by neuronal cells were examined using the enzyme-linked immunosorbent assay. The AD mouse model significantly increases the oxidative stress levels and 67 dysregulated miRNAs were discovered identified by the GEO data analysis and microRNA-668-3p was discovered as a potential AD therapeutic target. The AD and Aβ1-42-induced models showed an increase in microRNA-668-3p and a decrease in oxidation resistance 1 (OXR1) expression. The luciferase analysis results revealed that microRNA-668-3p plays a role in AD development by targeting OXR1 and promoting intracellular oxidative stress by activating p53-p21 signaling. A1-42-induction decreases cell proliferation, increases apoptosis, which leads to increased cell cycle arrest, and promotes oxidative stress. Although the effect of the microRNA-668-3p inhibitor has been diminished, it still partially mitigated the effect of A1-42 and Tenovin-1 (TEN). Where microRNA-668-3p as a therapeutic target for AD. Therefore, microRNA-668-3p as a potential therapeutic target for AD and explored its role in AD pathogenesis. Subsequently the suppression of microRNA-668-3p expression had a neuroprotective effect on cell damage induced by AD or Aβ1-42. Furthermore, this neuroprotective effect was related to the inhibition of oxidative stress and neuronal apoptosis as well as the regulation of the miR-668-3p/OXR1/p53-p21 axis. Excessive amounts of ROS, MDA, and NO, and inhibition of the secretion of SOD, GSH, and other antioxidant enzymes, resulting in damage to the hippocampal neurons in the brain, which is consistent. These large amounts of indelible ROS can induce damage to cell membranes and mitochondria, leading to NSC apoptosis. Additionally, that microRNA-668-3p was up regulated in the GEO data analysis, AD mouse models, and *in vitro* Aβ1-42-induced Neural Stem Cells (NSCs). This result was consistent, and *in vitro* Aβ1-42 induction decreased NSC proliferation and promoted apoptosis, cell cycle arrest, and oxidative stress. The negative impacts of A1-42 induction on cells could be reversed by inhibiting microRNA-668-3p expression. As a result, microRNA-668-3p expression may be essential for the development of AD, though its exact mechanism is still unknown.

Correspondence to: Mark William, Department of Neurology, IU Health Neuroscience Center, Indianapolis, USA, E-mail: markwilliam@IU.edu

Received: 27-Feb-2023, Manuscript No. TMCR-23-21537; **Editor assigned:** 01-Mar-2023, Pre QC No. TMCR-23-21537 (PQ); **Reviewed:** 15-Mar-2023, QC No. TMCR-23-21537; **Revised:** 22-Mar-2023, Manuscript No. TMCR-23-21537 (R); **Published:** 30-March-2023, DOI: 10.35248/ 2161-1025.23.13. 280

Citation: William M (2023) A Brief Note on Regulation of Oxidative Stress and Cell Damage Caused by Aβ1-42. Trans Med.13:280.