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Editorial OMICS International

# Oxidative Stress and Alzheimer's Disease

**Sandeep Kumar Singh***

Indian Scientific Education and Technology Foundation, Lucknow, UP, India

***Corresponding author**: Sandeep Kumar Singh, Indian Scientific Education and Technology Foundation, Lucknow, UP, India, E-mail: sks.1247@gmail.com

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## Editorial

Alzheimer's disease (AD) is a major human neurodegenerative disorder in aged (65 onwards) people. There are basically two hallmark of the AD; one is extracellular deposition of amyloid beta (Aβ) and other is intracellular formation of neurofibrillary tangles (NFT) [1]. Amyloid beta produced due to abnormal processing of amyloid beta precursor protein (APP) by the action of two important serine proteases; β and γ- secretases. Extracellular Aβ deposition results into senile plaque formation. AD is basically of two types, sporadic and familial. Most common among them is sporadic and have greater risk factor. Also environmental factors play a very important role in occurrence and progression of AD. Current estimates suggest that till now, there are about 5.2 million people diagnosed with AD in the USA. This number would be approximate 13-14 million, means going to be triple by the end of 2050, and approx. associated cost will be around 2 trillion US dollars [2]. Till now there is no available treatment for AD and scientific community is continuously searching for the same in best possible way by finding suitable pathway responsible for AD. Several hypotheses have been reported to explain various causes of AD, but the exact mechanisms remain unclear. Amyloid cascade hypothesis is most accepted hypothesis for AD but still faced lot of challenges in past decades due to unavailability of any drug based on this hypothesis [3,4]. Another most attractive and attentive area to explain the disease mechanism in AD is related to mitochondrial dysfunction and elevated production of reactive oxygen species (ROS). Generally, in most of the diseases, oxidative stress plays an important role. Several studies clearly mentioned that mitochondrial dysfunction has been observed in AD [5-7]. In mitochondrial cascade hypothesis, it has been clearly demonstrated that in case of sporadic AD, late onset AD, mitochondrial dysfunction associated with the expression and APP processing and Aβ accumulation [8].

Increased production of reactive oxygen species (ROS) associated with age- and disease-dependent mitochondrial dysfunction, altered metal homeostasis, and reduced antioxidant defense mechanism by affecting neuronal activity leads to cognitive dysfunction. In addition to this there are several other targets affected by ROS which includes lipids, proteins, nuclear and mitochondrial DNA, cellular architecture, calcium homeostasis, mitochondrial dynamics and function, receptor trafficking and energy homeostasis. Directly or indirectly production of amyloid beta (Aβ) and hyperphosphorylated Tau protein exacerbate ROS production and mitochondrial dysfunction, thereby contributing to the abnormal cellular mechanism. In search of treatment of AD, several clinical trials have been performed targeting various therapeutic approaches including antioxidant therapeutic approaches but no consistent and satisfied results are being observed. So, we need to explore further on this topic, that how and what way the oxidative stress influencing the AD pathology and what are the possible therapeutic approaches we can go for targeting oxidative cascade pathway. The main aim of this editorial is to draw the attention of the scientific community to explore more and more research activity focus on the oxidative stress in context to neurodegeneration; specially towards Alzheimer's disease so that possible therapeutic drug can be discovered.

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