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## Poster Presentation

### Effects of Dimethyl Sulfoxide on NLRP3 Inflammasome and Alzheimer's Disease

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#### **Abstract**

Alzheimer's disease (AD), the most ordinary form of dementia and extracellular accumulation of Amyloid- $\beta$  ( $A\beta$ ) in senile plaques, is an important and a main event in the pathogenesis of AD. Deposition of  $A\beta$  Peptide initiates a spectrum of cellular responses that are interposed by the resident neuroimmune cells of the brain, the microglia. Recently, a novel inflammasome signaling pathway has been uncovered and  $A\beta$  can activate the NLRP3 inflammasome in microglia, which is fundamental for the secretion of pro-inflammatory cytokines and subsequent inflammatory events. More importantly, the activation of NLRP3 inflammasome has demonstrated a serious role in AD pathogenesis by interposing a harmful chronic inflammatory response, while inhibition of NLRP3 mainly protected from loss of spatial memory and decreased  $A\beta$  deposition in an AD mouse model. Dimethyl Sulfoxide (DMSO) is an amphipathic molecule that is widely used as a solvent for biological compounds. In addition, DMSO has been studied as a medicine for the treatment of inflammation, cystitis, and arthritis. Based on the anti-inflammatory characteristics of DMSO, the effects of DMSO on activation of inflammasomes has elucidated, which are cytoplasmic multi-protein complexes that interpose the maturation of interleukin (IL)-1 $\beta$  by activating caspase-1 (casp1). The aim is discussing about effects of DMSO on NLRP3 inflammasome and AD. It has proved that DMSO attenuates IL-1 $\beta$  maturation, casp1 activity, and ASC pyroptosome formation by NLRP3 inflammasome activators. DMSO is a selective inhibitor of the NLRP3 inflammasomes. The anti-inflammatory effect of DMSO was further proved in animal studies, LPS-endotoxin sepsis, and inflammatory bowel disease models. DMSO shows anti-inflammatory characteristics, attenuates NLRP3 inflammasome activation. According to studies, it is hypothesized that DMSO inhibits activation of inflammasomes, NLRP3, CASP1 in Alzheimer's disease that are pathogenesis by mediating a harmful chronic inflammatory response.

**Keywords:** Alzheimer's disease, NLRP3, Casp1, Inflammasome, DMSO

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