

Editorial

Role of NLRP3 Inflammasome in Development and Progression of Alzheimer's Disease with Emphasis of Chemokines/Cytokines with the Approach to Develop Therapeutic Agent

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Inflammasomes are multiprotein complexes consist of having nucleotide binding domain and leucine rich repeat with the pyrin and HIN domain family. The NLRP3 inflammasome one of these members of family it is form upon sensing microbes or danger associated molecular pattern. NLRP3 inflammasome activation leads to activation of caspase 1 which is in turn activate proinflammatory cytokines/chemokines [1].

The role of NLRP3 inflammasome in Alzheimer's disease has recently being identified. NLRP3 inflammasome activation is necessary for maturation of these chemokines. Prolong activation of NLRP3 inflammasome and release of cytokines/chemokines, and neuronal cell death serve as danger signal to NLRP3 activation, in this way neuronal cell death provide feedback loop and deteriorate the pathological condition [2].

Deficiency or inhibition of NLRP3 inflammasome can be beneficial to reduce the deleterious effect of neuroinflammation in pathophysiology of AD [3]. It has been described that NLRP3 inflammasome activity is under additional transcriptional regulation Identification of the myeloid-specific microRNA miR-223 using animal modal/Immortalized human/animal cell line. MiR223 is a critical regulator of NLRP3 inflammasome activity miR-223 suppresses NLRP3 expression through a conserved binding site within the 3' untranslated region of NLRP3, translating to reduced

NLRP3 inflammasome activity [4]. It is interested to note that miR-223 itself is not regulated by proinflammatory signals; its expression varies among different myeloid cell types. Therefore, given the tight transcriptional control of NLRP3 message itself, miR-223 functions as an important rheostat controlling NLRP3 inflammasome activity. Therefore, induction of therapeutic treatment target to NLRP3 inflammasome may be beneficial to AD patients [5,6].

References

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