allied academies Joint Event on
International Conference on
NEUROSCIENCE AND NEUROLOGICAL DISORDERS
&
International Conference on
PSYCHIATRY AND PSYCHOLOGICAL DISORDERS
June 28-29, 2018 | Dublin, Ireland
J Neurol Neurorehabil Res 2018, Volume 3

MECHANISMS OF SELF-MAINTENANCE OF CHRONIC INFLAMMATION IN ALZHEIMER'S DISEASE

Jerzy Leszek
Wroclaw Medical University, Poland

Low grade inflammatory reactions are considered important factors that accelerate the progression of Alzheimer's disease. Major receptors of innate immunity, Toll-like receptors (TLRs) and receptor for advanced glycation end-product (RAGE), play a central role in triggering and driving these chronic inflammatory reactions. Signal transduction pathways from TLR and RAGE receptors lead to activation of transcription factors, mainly NF-kB and AP-1, which enhance the synthesis of proinflammatory cytokines. Activation of NF-kB enhances the transcription and expression of additional amounts of RAGE receptor protein in immune cell membranes, which results in increased further activation of this receptor. Many of the RAGE signal transduction pathway proteins can activate proteins from TLR pathways and vice versa. Such RAGE-TLR cross-activation emerges as an important driving force for maintenance of chronic inflammation in Alzheimer's disease, which finally increases the amount of proinflammatory cytokines released. Intractable, self-sustaining inflammatory reactions in the brain tissue, accompanied by an increased level of released proinflammatory cytokines, create a microenvironment for the development of an autoimmune component in neurodegeneration. Lowering the level of RAGE activation should weaken the RAGE-TLR cooperation and could bring about a significant slowdown in the disease progression.

jerzy.leszek@umed.wroc.pl