RAGE-TLR Crosstalk Sustains Chronic Inflammation in Neurodegeneration

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Chronic inflammatory reactions are consistenly present in neurodegeneration of Alzheimer type and are considered important factors that accelerate progression of the disease. Receptors of innate immunity participate in triggering and driving inflammatory reactions. For example, Toll-like receptors (TLRs) and receptor for advanced glycation end product (RAGE), major receptors of innate immunity, play a central role in perpetuation of inflammation. RAGE activation should be perceived as a primary mechanism which determines self-perpetuated chronic inflammation, and RAGE cooperation with TLRs amplifies inflammatory signaling. In this review, we highlight and discuss that RAGE-TLR crosstalk emerges as an important driving force of chronic inflammation in Alzheimer?s disease.

Chronic inflammation

Neurodegeneration

Rage

Self-perpetuated stimulation

TLR

advanced glycation end product receptor

free radical

toll like receptor

advanced glycation end product receptor

toll like receptor

Alzheimer disease

brain level

chronic inflammation

chronic stress

disease course

gene amplification

human

immunoreactivity

innate immunity

nerve degeneration

protein expression

protein function

protein protein interaction

protein structure

receptor cross-talk

Review

signal transduction

animal

degenerative disease

inflammation

metabolism

nerve degeneration

physiology

Animals

Humans

Inflammation

Nerve Degeneration

Neurodegenerative Diseases

Receptor for Advanced Glycation End Products

Signal Transduction

Toll-Like Receptors