







THE ROLE OF THE NLRP3 INFLAMMASOME FOR THE PROGRESSION OF ALZHEIMER DISEASE PATHOLOGY

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Abstract

Innate immunity has been identified as an important component of Alzheimer disease pathogenesis. Activation of the NLRP3 inflammasome represents a key immune signaling event and has been shown in microglial cells in the AD brain. Downstream effects include impaired microglial clearance of beta-amyloid deposits, seeding and spreading of disease by ASC speck release and IL-1beta-mediated hyperphosphorylation of tau, ultimately resulting in the neurofibrillary tangle formation. The presentation will review know and new data in order to discuss whether NLRP3 inflammasome inhibitors may serve as future treatment for AD.

Biosketch

Michael Heneka studied medicine at the University of Tübingen. He received clinical training in Neurology at the University of Tübingen, University of Münster and the University of Bonn. He directed the Department of Neurodegenerative Diseases and Gerontopsychiatry-Neurology at the University Hospital Bonn and led the Neurodegenerative Disease unit at the Deutsches Zentrum für Neurodegenerative Erkrankungen (DZNE). He is further adjunct professor at the Department of Infectious Diseases and Immunology at the University of Massachusetts Chan Medical School. In mid 2021, he became the director of the LCSB - Luxembourg Centre for Systems Biomedicine at the University of Luxembourg. He also holds an adjunct professorship at the University of Bonn and heads the Department of Neuroinflammation at the Institut of Innate Immunity at the University Hospital Bonn.

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February 13th Tuesday at 12:00h



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