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论文

JAK/STAT途径介导Aβ寡聚体诱导小胶质细胞TNF-α的释放

杨丽玲，王璐，任晓燕，韩晓娟，杜怡峰

山东大学附属省立医院神经内科，济南 250021

摘要：

目的 研究β淀粉样蛋白（Aβ）寡聚体对晚期糖基化蛋白相关受体（RAGE）介导小胶质细胞产生炎症反应的影响，分析Janus 激酶/ 信号转导和转录激活子（JAK/STAT）途径与Aβ寡聚体诱导肿瘤坏死因子α（TNF-α）释放的关系。方法 取经过刺激及阻断处理原代培养的小胶质细胞，经酶联免疫吸附法（ELISA）检测细胞上清液TNF-α的水平。结果 经Aβ寡聚体处理24h后TNF-α表达增加，分别经抗RAGE IgG和AG490预处理后再经Aβ寡聚体处理小胶质细胞，TNF-α释放明显被抑制。 结论 RAGE是Aβ寡聚体诱导小胶质细胞炎症反应的受体，JAK/STAT途径可能参与Aβ寡聚体诱导小胶质细胞TNF-α的释放。

关键词： 淀粉样β蛋白；小神经胶质细胞；糖基化终产物，高级；信号传导；肿瘤坏死因子

JAK/STAT signaling pathway mediates β-amyloid protein-induced TNF-α action of microglia in vitro

YANG Li-ling， WANG Lu， REN Xiao-yan， HAN Xiao-juan， DU Yi-feng

Department of Neurology, Provincial Hospital Affiliated to Shandong University, Jinan 250021, China

Abstract:

Objective To explore the inflammatory response of microglia induced by β-amyloid (Aβ) oligomers through the receptor for advanced glycation end product (RAGE) ,and to further analyze the relationship between Janus kinase/signal transducer and activator of transcription (JAK/STAT) signaling pathway and tumor necrosis factor-α (TNF-α) of microglia induced by Aβoligomers. Methods After the primary rat microglial cells were stimulated and obstructed, the level of TNF-α extracted from supernatant liquid of microglia was measured by enzyme-linked immunosorbent assay (ELISA). Results TNF-αwas increased after primary rat microglia cells were treated by Aβoligomers for 24h. However, anti-RAGE IgG and AG490 reduced the activation of microglia cells and obviously inhibited Aβ oligomer-induced release of TNF-α. Conclusions RAGE is the receptor of microglia in inflammatory response induced by Aβ oligomer. JAK/STAT pathway may participate in the release of TNF-α induced by Aβ oligomer.

Keywords: Amyloid beta-protein; Microglia; Glycosylation end products, advanced; Signal transduction; Tumor necrosis factor

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通讯作者: 杜怡峰（1962- ），男，教授，博士生导师，主要从事老年性痴呆、神经变性病以及脑血管病发病机制及防治的研究。 Email:duyifengpmd2000@yahoo.com.cn

作者简介: 杨丽玲（1982- ），女，硕士研究生，主要从事老年性痴呆发病机制的研究。 Email:yangliling.123@163.com

作者Email:

参考文献：

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