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The role of galectin-3 in influenza virus-induced host immune responses

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H⁵N¹ is a highly pathogenic avian influenza virus which causes pneumonia and acute respiratory distress syndrome in human. Virus-induced cytokine dysregulation and excessive inflammatory response contribute to the pathogenesis of human H⁵N¹ disease. Galectin-3 is a β -galactoside binding animal lectin widely distributed in immune and epithelial cells. Galectin-3 has been reported to regulate various immune functions and shows pro-inflammatory activity. In the present study, we investigated the roles of endogenous galectin-3 in H⁵N¹ virus infection-induced host immune responses. We observed that galectin-3 was up-regulated in the lungs after H⁵N¹ infection in mouse model, and the increased galectin-3 was mainly expressed by the infiltrating cells. Our preliminary data also showed that galectin-3 knockout (Gal-3KO) mice were less susceptible to H⁵N¹ virus infection compared to wild-type (WT) mice, whereas the viral loads in lungs were comparable. Moreover, we found that H⁵N¹-infected Gal-3KO mice exhibited lower degree of lung inflammation, and lower level of neutrophil and macrophage recruitment to lung. In addition, the level of IL-1 β in both bronchoalveolar lavage and lung were significantly reduced in H⁵N¹-infected Gal-3KO mice. In the in vitro study, we also showed that bone marrow-derived macrophages from Gal-3KO mice produced reduced level of proinflammatory cytokines IL-1 β and chemokine MCP-1 compared to those from WT mice in response to H⁵N¹ infection. Combined, our results suggest that galectin-3 may enhance the pathological effects of H⁵N¹ virus infection by promoting inflammatory response and that galectin-3 may be targeted for H⁵N¹ virus-induced severe lung inflammation.

Biography

Huan-Yuan Chen is associate research scientist in Academia Sinica, Institute of Biomedical Sciences. He completed his Ph.D. from National Tsing Hua University. His laboratory focuses on the study of the roles of galectin-3 in cells involved in skin inflammation.

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