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NK cells aggravate acute lung injury via up-regulation of NKG2D during early stage of H1N1 influenza infection

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Acute lung injury was considered as the major pathological contribution of 2009 pandemic H1N1 influenza virus infection. NK cells were the first line to defend against virus infection, but their roles in the lung pathogenesis and virus elimination were not fully elucidated. The influenza infection model was established with Balb/c mice. The flow cytometry was used to detect the expression of extracellular and intracellular molecular. H&E straining was done to evaluate the pathological lession of lung. Severe Balb/c mice infection model was intranasally inoculated with influenza A virus strain A/California/07/2009. Following virus challenge, the body weight was lost and survival rate decreased. The infected lung showed severe lung injury, including pulmonary edema and capillary leak, and a large number of infiltrating lymphocytes were recruited to perivascular and parenchyma areas in mice model and patients. Total lymphocytes in lung and bronchoalveolar lavage fluid (BALF) were increased as the infection progressed, and the ratio and number of NK cells was significantly increased. But the ratio of T cells in lung had no change. Furthermore, NK cells were rapidly activated, and secreted a large amount of IFN-γ and increased high level of perforin and granzyme B. H1N1 infection induced significant high expression of NKG2D, but not NKG2A, on NK cells. NKp46, which can recognize virus HA, was also improved. Meanwhile, H1N1 infection induced significantly high expression of NKG2D ligands (RAE-1) and low expression of NKG2A ligands (Qa-1α). Depletion of NK cells with AsGM1 show lighter lung damage and weight loss, but higher virus titer compared with PBS control for the first three days after infection, accompanying with reduced secretion of IFN-γ. Our data demonstrated NK cells played dual roles in lung injury and virus elimination during the early stage of H1N1 virus infection.

Biography

Xulong Zhang received his MD degree from Shandong University and completed his Post-doctoral Training at USTC. Now, he is focusing on the innate immune cells or molecular medicated protection or damage after influenza virus infection.

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