Interleukin-17 enhances the removal of respiratory syncytial virus in mice by promoting neutrophil migration and reducing interferon-gamma expression

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ABSTRACT. The aim of this study was to observe the effect of interleukin (IL)-17 on early immune response and inflammation in the lungs of respiratory syncytial virus (RSV)-infected mice. Specific pathogen-free BALB/c mice were randomly assigned to control, RSV-infected, RSV-infected with phosphate-buffered saline, and RSV-infected + IL-17 intervention groups. The RSV infection model was set up by nasal mucosa immunization. The intervention group was provided with restructured IL-17 (intranasal). The viral load and cytokine concentrations in the lung tissues and broncho-alveolar lavage fluid (BALF) were determined by real-time-polymerase chain reaction and enzyme-linked immunosorbent assay. RSV caused acute lung inflammation in mice with a significantly higher number of neutrophils and cytokines such as interferon-gamma (IFN-γ), IL-1β, IL-6, and G-CSF in the BALF than that in the control group. IL-17 intervention led to a significant increase in the number of neutrophilic granulocytes in the BALF. Alternately, IL-17 intervention led to a significant decrease in
the IFN-γ concentration and a significant increase in the IL-1β, IL-6 and G-CSF levels in the BALF. IL-17 induced a reduction in the viral load and an increase in the survival rate of mice on the third day of infection. IL-17 mucosal immunity enhances the removal of RSV and strengthens the immune defense by promoting neutrophil migration and reducing the IFN-γ levels in mouse lungs.

**Key words:** Respiratory syncytial virus; Interleukin-17; Interferon-gamma; Neutrophil