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Alveolar cells participate in CD8^{+}T cell recruitment

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Keywords

Infectious diseases, interstitial lung diseases, respiratory immunology

Context

$CD8^+$ T lymphocytes are important in the recognition and clearance of viral infections, and are also associated with the chronic lung inflammation found in chronic obstructive pulmonary disease (COPD) and interstitial lung diseases such as allergic alveolitis and silicosis. At present, the mechanisms by which these effector cells interact with cells found within the alveolus remain poorly characterized. The aim of this study was to explore the interaction of the alveolar epithelial cell and the activated $CD8^+$ T lymphocyte.

Significant findings

The authors have taken advantage of transgenic mice expressing influenza hemagglutinin (HA) under the control of the surfactant protein-C promoter. In these animals, alveolar epithelial cells express the HA protein constitutively. In previously published work (see Additional information), the authors found that adoptively transferred HA specific $CD8^+$ T cells recognized antigen expressed by alveolar epithelial cells, and caused progressive, fatal lung inflammation characterized by host-derived macrophages. The authors found in that study that this lung injury could be abrogated by blockade of tumor necrosis factor (TNF). In the current study, the authors explore the interaction of the antigen-specific $CD8^+$ T cell and the alveolar epithelial cell. They found that antigen-specific release of TNF by the $CD8^+$ T cells led not to apoptosis of the alveolar epithelial cells, but to the release by these cells of the potent macrophage chemokines monocyte chemoattractant protein (MCP)-1 and macrophage inflammatory protein (MIP)-2. Blockade of MCP-1 reduced the degree of inflammation following adoptive transfer of the $CD8^+$ T cells.

Comments

While the study does not prove that alveolar epithelial cells are directly responsible for the recruitment of monocytes, the study is important in that it provides a plausible mechanism by which chronic antigenic stimulation of $CD8^+$ T cells by alveolar epithelial cells could lead to lung injury. Additionally, the study might also suggest a therapeutic role for the blockade of either TNF-a or MCP-1 in diseases associated with a predominance of $CD8^+$ T cells.

Methods

Adoptive transfer, cytokine measurement, immunohistochemistry

Additional information

Liu AN, Mohammed AZ, Rice WR, Fiedeldey DT, Liebermann JS, Whitsett JA, Braciale TJ, Enelow RI: **Perforin-independent $CD8^+$ T cell mediated cytotoxicity of alveolar epithelial cells is preferentially mediated by tumor necrosis factor-a: relative insensitivity to Fas ligand.** *Am J Respir Cell Mol Biol* 1999, **20**:849-858

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