Phagocytic Elimination of Influenza Virus-Infected Cells by Phagocytes

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Infection with a variety of viruses induces apoptosis in host cells. This phenomenon may be considered to be a self-defense mechanism to avoid viral propagation. However, the growth of influenza virus completes before host cells become dysfunctional by apoptosis. To know the physiological consequences of influenza virus-induced apoptosis, we have examined the fate of influenza virus-infected cells in vitro as well as in vivo.

Influenza virus-infected cells were engulfed by macrophages in vitro, and virus propagation was almost completely inhibited. This phagocytosis was dependent on the specific recognition of the membrane phospholipid phosphatidylserine exposed on the surface of virus-infected apoptotic cells by macrophages. In addition, the activity of viral neuraminidase expressed at the surface of virus-infected cells was necessary for the maximal level of phagocytosis. When mice infected with influenza virus were administered with phagocytosis inhibitors, the level of lethality and inflammation in the lung were augmented. These results show that apoptosis makes influenza virus-infected cells susceptible to phagocytosis by macrophages, and that this leads to a reduction in the extent of influenza pathology.