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Lung alert

Influenza infection: the origin and role of IL10

With fears of an influenza pandemic looming, understanding the mechanism by which pathogenic strains of influenza cause lethal lung injury could not be more relevant. Interleukin 10 (IL10) is an anti-inflammatory cytokine recognised to prevent excessive injury in bacterial and parasitic infections. This series of elegant experiments focuses on the role of IL10 in influenza-infected mice, the proportion of IL10 produced by effector T cell (Teff) subtypes and the kinetics of this response.

The authors demonstrate that CD8+ Teff cells are the main contributor of IL10 in influenza-infected lungs, with IL10-producing CD8+ Teff cells outnumbering IL10-producing CD4+ Teff cells twofold. They propose that the time at which IL10-producing T cells are found (days 6–26 after infection) corresponds to our understanding of CD8+ migration kinetics, providing further evidence that CD8+ Teff cells are the main source of IL10. Similarly, using knock-out mice, the authors showed that T cell depletion correlates with a significant reduction in IL10 production. Bronchoalveolar lavage fluid from infected wild-type mice produced nearly 6000 pg/ml IL10 while production in infected T cell-deficient mice approached zero.

An interesting final experiment examined the effect of IL10 receptor (IL10R) blockage on survival rate. Twelve mice were treated with IL10R-specific monoclonal antibodies. A dramatically higher proportion of these mice died once infected with influenza and this was partially reversible with corticosteroid administration.

This paper provides novel evidence of the significance of IL10 in viral infections, specifically that Teff cells are crucial in regulating excess injury in influenza infection via production of IL10. The authors speculate that highly pathogenic influenza strains may selectively suppress IL10 production by Teff cells. This will be important in guiding future studies in humans.

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