

FLU-B-GONE INDUCES IL-6 RESPONSES IN MACROPHAGES

FLU-B-GONE IMMUNOLOGICAL RESPONSE UPDATE

Antia E Veal

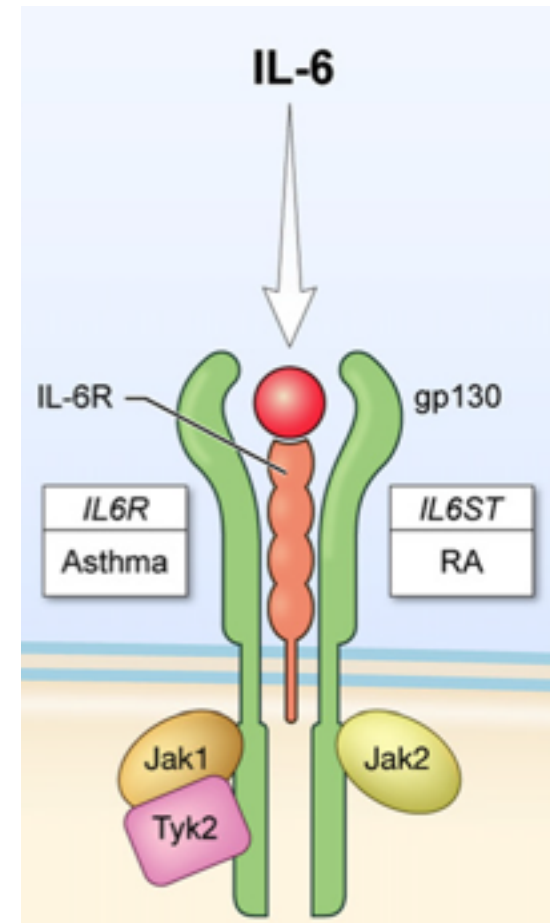
Introduction

The immune response is differentially regulated by a diversity of **cytokines**

Cytokines are chemical messengers that recruits other immune cells to function and response to cellular processes, inflammation, and homeostasis.

IL-6

- Pro-inflammatory cytokine and an anti-inflammatory cytokine
- Produced by a variety of cells including **macrophages**, dendritic cells, mast cells, B cells, and various non-leukocytic cells



Previous Literature

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Mucosal Immunol. 2012 May;5(3):258-66. doi: 10.1038/imi.2012.2. Epub 2012 Feb 1.

Essential role of IL-6 in protection against H1N1 influenza virus by promoting neutrophil survival in the lung.

Dienz O¹, Rud JG, Eaton SM, Lanthier PA, Burg E, Drew A, Bunn J, Suratt BT, Haynes L, Rincon M.

Author information

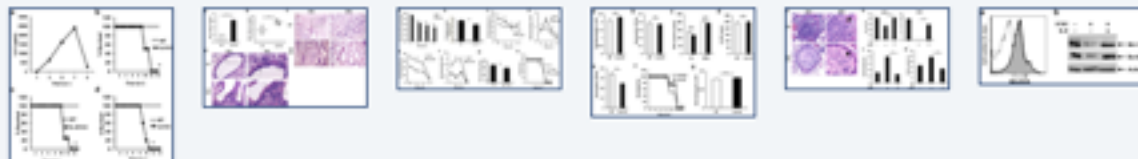
Abstract

Influenza virus infection is considered a major worldwide public health problem. Seasonal infections with the most common influenza virus strains (e.g., H1N1) can usually be resolved, but they still cause a high rate of mortality. The factors that influence the outcome of the infection remain unclear. Here, we show that deficiency of interleukin (IL)-6 or IL-6 receptor is sufficient for normally sublethal doses of H1N1 influenza A virus to cause death in mice. IL-6 is necessary for resolution of influenza infection by protecting neutrophils from virus-induced death in the lung and by promoting neutrophil-mediated viral clearance. Loss of IL-6 results in persistence of the influenza virus in the lung leading to pronounced lung damage and, ultimately, death. Thus, we demonstrate that IL-6 is a vital innate immune cytokine in providing protection against influenza A infection. Genetic or environmental factors that impair IL-6 production or signaling could increase mortality to influenza virus infection.

PMID: 22294047 PMCID: PMC3328598 DOI: 10.1038/imi.2012.2

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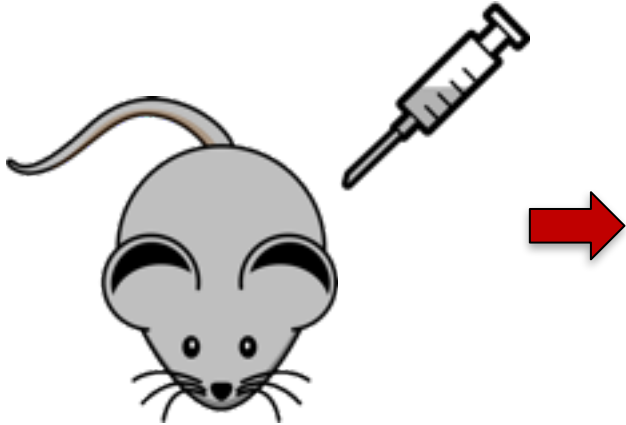
[Kinetics of pulmonary immune cells, antibody responses and their correlations with viral load in mice infected with influenza A virus \[Virol J. 2014\]](#)[Activation of A1-adenosine receptors promotes leukocyte recruitment to the lung in mice \[J Virol. 2014\]](#)[The role of IL-27 in susceptibility to post-influenza Staphylococcus aureus infection \[Respir Res. 2015\]](#)[Leptin mediates the pathogenesis of severe 2009 pandemic influenza A\(H1N1\) infection \[J Infect Dis. 2013\]](#)[Review Selective proteolytic cleavage of the ligand-binding chains of influenza A virus hemagglutinin \[Adv Exp Med Biol. 1997\]](#)[See reviews...](#)[See all...](#)

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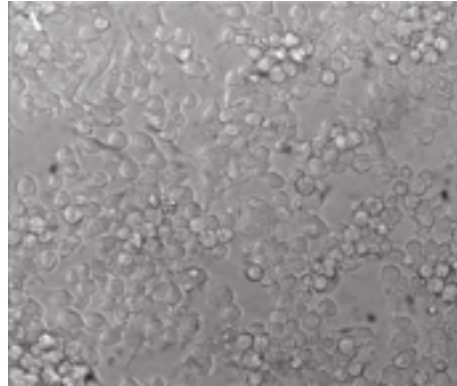
[Zika virus preferentially replicates in the female reproductive tract after vaginal infection \[PLoS Pathog. 2017\]](#)[Review Bacterial ghosts as adjuvants: mechanisms and potential. \[Vet Res. 2017\]](#)[Pulmonary immune cells and inflammatory cytokine dysregulation are associated with influenza A virus infection \[Zool Res. 2017\]](#)[See all...](#)

Experimental Design

Experimental Design



Balb/c mice macrophages were extracted and fused with cancerous cells creating an immortal mouse cell line called RAW 264.7 macrophages



RAW 264.7 macrophages were plated for 24hours for adherence

Treatments/Conditions

Cells

Lipopolysaccharide (LPS)

LPS + FBG

RAW 264.7 macrophages cells were treated with various stimuli with and without the presence of FLU-B-GONE (FBG) to detect IL-6 reponses produced by macrophages

Results

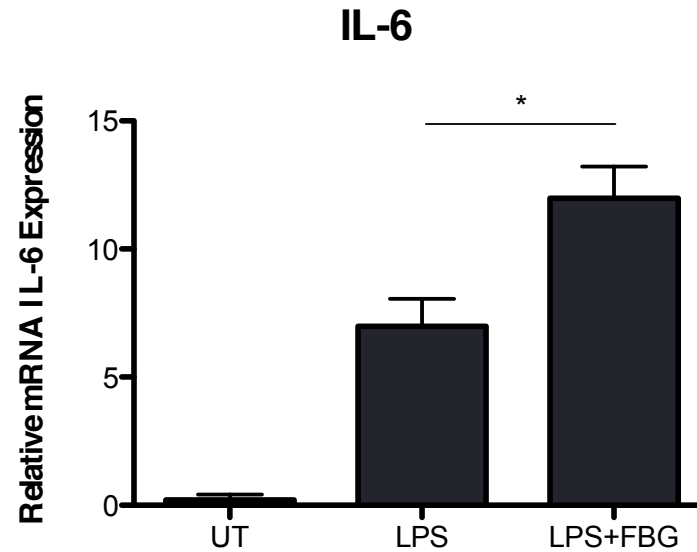


Figure: IL-6 is significantly increased with the influence of LPS + Flu-B-Gone. Cells were cultured with the presence and absence of FBG for IL-6 detection through quantitative PCR mRNA expression. Experiments were conducted five times (n=5). P value= 0.0183 (*)