

Signaling Through the Prostaglandin I₂ Receptor IP Protects Against Respiratory Syncytial Virus-Induced Illness

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Abstract

The role of prostanoids in modulating respiratory syncytial virus (RSV) infection is unknown. We found that RSV infection in mice increases production of prostaglandin I₂ (PGI₂). Mice that overexpress PGI₂ synthase selectively in bronchial epithelium are protected against RSV-induced weight loss and have decreased peak viral replication and gamma interferon levels in the lung compared to nontransgenic littermates. In contrast, mice deficient in the PGI₂ receptor IP have exacerbated RSV-induced weight loss with delayed viral clearance and increased levels of gamma interferon in the lung compared to wild-type mice. These results suggest that signaling through IP has antiviral effects while protecting against RSV-induced illness and that PGI₂ is a potential therapeutic target in the treatment of RSV.

Figures

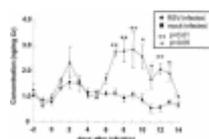


FIG. 1.

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Concentrations of the PGI₂...

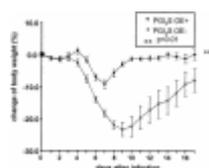


FIG. 2.

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Daily weight curves of RSV-infected...

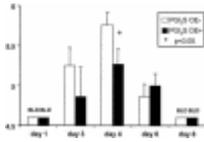


FIG. 3.

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Viral titers in RSV-infected transgenic...

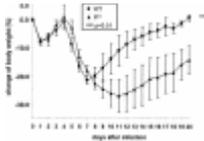


FIG. 4.

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Daily weight curves of RSV-infected...

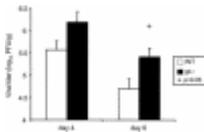


FIG. 5.

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Viral titers in RSV-infected transgenic...

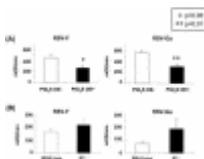


FIG. 6.

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Measurement of IgG1 anti-RSV antibody...

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