INTRODUCTION

Lung patholgy demonstrates that SB 9200 Reduces RSV Disease in Mice

SB 9200 indicates IFR-3 activation along with IFN-β and NF-κB production

Reduction of IFN-3 expression by SB 9200 is mediated by RIG-I and NO22

SB 9200 shows both prophylactic and therapeutic antiviral activity against RSV in a mouse model

SB 9200 treatment resulted in significant reduction of RSV load in lungs of a mouse model

SB9200 treatment also resulted in reduced RSV nucleocapsid expression in lungs

Reduced RIGI and NO22 expression was noted in SB 9200-treated airway. This scenario could be due to reduced RSV infection of SB 9200-treated lungs. Lower viral burden will lead to “switching-off” (i.e., reduced expression) of pro-inflammatory mediators including cytosolic sensors RIG-I and NO22.

Enhanced IFN-β production was observed in the lungs of SB 9200-treated mice infected with RSV. Since IFN-β plays an important role in antiviral response, increased IFN-β production in the respiratory tract of SB9200-administered mice indicated a potential therapeutic role of SB 92 in anti-RSV therapy.

Diminished levels of TNF were observed in the lungs of SB 9200-treated mice infected with RSV. Since, TNF plays a significant role in lung inflammation and RSV disease, diminished TNF production indicated a potential therapeutic role of SB 9200 in reducing RSV-associated lung disease.

Reduced inflammation and lesser severity of immunomodulatory disease was noted in lungs of SB 9200-treated mice compared to untreated mice.

Thus, SB 9200 diminishes disease severity associated with RSV infection and shows both prophylactic and therapeutic activity.

RESULTS

These results demonstrate modulation of the innate immune response that mediates the anti-RSV activity of SB 9200.

SB 9200 induces IFR-3 activation along with IFN-β and NF-κB production

Reduction of IFN-3 expression by SB 9200 is mediated by RIG-I and NO22

SB 9200 shows both prophylactic and therapeutic antiviral activity against RSV in a mouse model

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