

SALMETEROL ATTENUATES CHEMOTAXIS IN RHINOVIRUS-INDUCED EXACERBATION OF ASTHMA VIA MODULATION OF PP2A

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Aim: To investigate the effectiveness of Salmeterol as an anti-inflammatory drug in allergic airways disease and rhinovirus (RV) exacerbation.

Methods: Allergic House dust mite (HDM)-challenged mice were infected with RV1B. Prior to exacerbation, mice were administered with either dexamethasone, salmeterol, vehicle or the protein phosphatase 2A (PP2A) activating drug AAL(s). Airway hyperreactivity (AHR), inflammation, cytokine/chemokine expression and viral titres/antiviral responses were determined.

Results: Salmeterol and dexamethasone treatment suppressed AHR to an equal extent. Salmeterol and AAL(s) ameliorated eosinophil recruitment by suppressing eotaxin-1 (CCL11) and increased PP2A activity within the lung. This correlated with suppressed levels of phosphorylated-JNK and -ERK1.

Conclusions: This study identifies a novel anti-inflammatory role of Salmeterol *in-vivo*, by potentially modulating PP2A-mediated dephosphorylation of p-JNK and p-ERK1.

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