

Higher Susceptibility of Allergic Rhinitis Nasal Mucosa to Influenza a Viral Infection: *In vitro* and *In vivo* Study

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Rationale: We studied whether the nasal mucosa in allergic rhinitis (AR) would be more susceptible to influenza A virus (IAV) infection due to lower induction of interferon (IFN)-related immune responses.

Objectives: To determine whether IFN induction would be impaired in allergic nasal mucosa and to identify which IFN was correlated with higher viral loads in IAV-infected allergic nasal mucosa.

Methods: IAV mRNA, viral titers and IFN expression were compared in IAV-infected normal human nasal epithelial (NHNE, N=10) and allergic rhinitis nasal epithelial (ARNE, N=10) cells. We used *in vivo* model of AR (BALB/C mouse, N=10) and human nasal mucosa from healthy volunteers (N=72) and AR patients (N=29) to assess the induction of IFNs after IAV infection.

Results: IAV mRNA levels and viral titers were significantly higher in ARNE compared with NHNE cells. IFN- β and - λ s were induced in NHNE and ARNE cells up to 3 days after IAV infection. Interestingly, induction of IFN- λ s mRNA levels and the amount of secreted proteins were considerably lower in ARNE cells. The mean IFN- λ s mRNA level was also significantly lower in the nasal mucosa of AR patients. We found that recombinant IFN- λ treatment attenuated IAV mRNA levels and viral titers in IAV-infected ARNE cells and completely controlled IAV infection in an *in vivo* AR model.

Conclusion: Higher susceptibility of the allergic nasal mucosa to IAV may depend on impairment of type III IFN induction, and type III IFN is a key mechanistic link between higher viral loads and control of IAV infection in allergic nasal mucosa.

Keywords: Influenza A virus; type III interferon; allergic rhinitis; nasal mucosa.

Biography:

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