Airway cells infected by HRV-16 may pass infection to pulmonary microvascular endothelium in vitro- possible mechanism in asthma exacerbations.

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Introduction

Rhinovirus is the most common cause of asthma viral exacerbations. It is unknown if HRV-16 may pass from infected airway epithelium to uninfected adjacent vascular lung endothelium.

Aim

To assess if HRV-16 may pass from infected normal human bronchial epithelium (NHBE) and human microvascular lung endothelial cells (HMVEC-L) to uninfected HMVEC-L in an *in vitro* model.

Material and methods

Donor NHBEC and HMVEC-L were grown in serum-free growth medium and incubated with HRV-16 for 3h, washed and cultured for subsequent 5, 24 or 96h. Additionally, a half of infected HMVEC-L were washed out in 72h to further collect supernatants in 96h. Next, HMVEC-L were cultured for 24h with supernatants from donor cultures of NHBECs or HMVEC-L infected with HRV-16. HRV-16 copy numer and IFN-β and RANTES mRNA expression were assessed in real time PCR.

Results

In donor NHBEC incubated with HRV-16, virus copies were detected (662±116 copies/µl). In HMVEC-L cultured with supernatants derived from HRV-16-infected HNBEC, 6 and 471 copies/µl in 5 and 24-hour were detected. It was accompanied by an increase of RANTES and IFN-β expression. In HMVEC-L stimulated by HRV16-infected HMVEC-L-derived supernatants collected in 96h (not washed), 511 copies/µl of HRV16 were detected, which was

accompanied by an 85-fold increase of RANTES mRNA expression. In HMVEC-L stimulated by supernatants collected in 96h and washed in 72h no HRV16 particles and RANTES mRNA expression increase were found.

Conclusions

HRV-16 infection may pass from infected airway epithelial and endothelial cells to uninfected lung vascular endothelium in an in vitro model. Endothelial infection may also induce proinflamatory response leading to exacerbations in asthmatics subjects.

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The authors declare no conflict of interest.