
# The effect of human rhinovirus HRV16 on enzyme expression of the arachidonic acid metabolic pathway in human pulmonary vascular endothelium.

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## Introduction

Rhinoviral infections lead to exacerbation of asthma. Eicosanoid synthesis is regulated by: phospholipase A2 (PLPA2), cyclooxygenases (COX1 and COX2), lipoxygenases (5LO, 12LO, 15LO) and leukotriene synthases (LTA4S, LTB4S and LTC4S). The involvement of eicosanoids in asthma immunopathology is well understood in epithelial cells. However, little is known about the engagement of arachidonic acid (AA) metabolic pathway enzymes and the production of eicosanoids by endothelial cells during the rhinoviral infection.

## Aim

To assess the effect of rhinovirus HRV16 on AA metabolic pathway enzyme expression in the human pulmonary microvascular endothelial cells (HMVEC-L).

## Material and methods

HMVEC-L were incubated with HRV16 (MOI 3) for 3 h. After virus removal, cells were further cultured for 5, 24 and 72 h to analyze COX2, 5LO, LTC4S, PLA2, and COX1 expression in real time PCR. RANTES mRNA expression was assessed as an indicato of efficient infection of HMVEC-L

## Results

HRV16 caused 10.6-fold and 3,9-fold increase of the mRNA expression of COX2 at 24 and 72 h in HMVEC-L ($p < 0.05$). Similarly, the expression of 5LO at 24 h was up-regulated (13.5-fold; $p < 0.05$). Expression of PLPA2 at 24 h was enhanced 2.7-fold ($p < 0.05$). COX1 showed a 2.3-fold increase of mRNA expression at 24 h of the culture. In contrast, HRV16 caused 0.5 and 0.3 down-regulation of LTC4S mRNA expression at 24 and 72 h

($p < 0.05$). The effectiveness of infection with HRV was confirmed by the 6000-fold (24 h) and 5300-fold (72 h) increase of RANTES mRNA expression.

## Conclusions

The regulation of AA metabolic pathway enzymes by HRV16 in the lung vascular endothelium suggests a possible involvement of AA metabolites in the immunopathology of rhinoviral asthma exacerbations.

The authors declare no conflict of interest.