



# Characterisation of grass pollen derived substances modulating the barrier functions of bronchial epithelial cells [Abstract]

Cornelia Blume, Emily J. Swindle, Heidrun Behrendt, Claudia Traidl-Hoffmann, Stephen T. Holgate, Donna E. Davies

### Angaben zur Veröffentlichung / Publication details:

Blume, Cornelia, Emily J. Swindle, Heidrun Behrendt, Claudia Traidl-Hoffmann, Stephen T. Holgate, and Donna E. Davies. 2011. "Characterisation of grass pollen derived substances modulating the barrier functions of bronchial epithelial cells [Abstract]." *American Journal of Respiratory and Critical Care Medicine* 183: A1420.

https://doi.org/10.1164/ajrccm-conference.2011.183.1\_meetingabstracts.a1420.



## Characterisation Of Grass Pollen Derived Substances Modulating The Barrier Functions Of Bronchial Epithelial Cells

### C. Blume 1, E. J. Swindle 1, H. Behrendt 2, C. Traidl-Hoffmann 2, S. T. Holgate 3, D. E. Davies 1

 $^{1}$ School of Medicine, University of Southampton, Southampton, United Kingdom,  $^{2}$ Center of Allergy and Environment, Munich, Germany,  $^{3}$ 

#### Corresponding author's email: C.Blume@soton.ac.uk

**RATIONALE:** During the pollen season, the airways are exposed to grass pollen causing allergic immune reactions in sensitised patients. Besides allergens, pollen also releases proteases and immunostimulatory substances after contact with aqueous surfaces. The aim of this study was to analyse the effects of grass pollen derived substances on the barrier properties of bronchial epithelial cells (BECs) and to characterise the active component.

**METHODS:** Polarised 16HBE14o-BECs in transwell inserts were exposed to aqueous pollen extract (APE). Proteins were separated from low molecular weight substances by ultrafiltration of APE. Further separation of APE was obtained by solid phase extraction. Transepithelial electrical resistance (TER) was measured over time and cytokine release was analysed by ELISA. Involvement of mitogen-activated protein kinase (MAPK) pathways was assayed using specific inhibitors.

**RESULTS:** APE increased TER in BECs dose-dependently with a maximum after 3h. This activity was caused by hydrophilic low molecular weight substances in APE, rather than high molecular weight substances which did not affect TER. p38 and JNK MAPK pathways were found to contribute to these responses. Hydrophilic low molecular weight substances in APE also induced a polarised release of IL-8 and GM-CSF. IL-8 was released in higher concentrations basolaterally, whereas GM-CSF release was predominantly apical. The JNK MAPK pathway was not involved in APE induced mediator release. IL-8 release was reduced after blocking the p38 MAPK pathway and the release of GM-CSF was mediated by both p38 and ERK1/2 pathways. The results obtained in this study indicate an independent regulation of the physical and immunological barrier functions of BECs.

**CONCLUSION:** The rapid decrease in permeability of the bronchial epithelium suggests that tightening the barrier may be an early defence mechanism triggered by exposure to grass pollen. Later induction of mediator release by grass pollen substances suggests alterations in the immunological barrier function of the bronchial epithelium may further contribute to defence against environmental insults.

This abstract is funded by: Bayerische Forschungsstiftung, Germany University of Southampton, England

Am J Respir Crit Care Med 183;2011:A1420 Internet address: www.atsjournals.org

**Online Abstracts Issue**