**Potential therapeutic target in a murine model of eosinophilic airway inflammation**

Guillaume Bendavid1, Natacha Rocks2, Philippe Lefèbvre1, Didier Cataldo3  
1ENT department, University Hospital of Liege, Belgium  
2Laboratory of Tumor and Development Biology, University of Liege, Belgium  
3Pneumology department, University Hospital of Liege, Belgium  
  
**Introduction and Aim:**  
  
Patients suffering from allergy may display an eosinophilic inflammation of the airway. A Disintegrin And Metalloproteinase (ADAM) might play an important role since they are able to cleave various modulators of inflammation and modulate their biological activity. ADAM28 is soluble and a multipotent membrane-bound or secreted proteinase expressed by epithelial cells in human normal tissues. ADAM28 influences lymphocytes adhesion and migration, and cleaves the low affinity IgE receptor (CD23).  
The aim of our research is to study the potential role of ADAM28 in eosinophilic airway inflammation.  
  
**Material-Methods:**  
  
Sputum from asthmatics and healthy subjects were collected and PCR analysis performed.   
C57 mice, ADAM28 knock-out (KO) mice and corresponding wild-type (WT) Mice were sensitized twice to ovalbumin (OVA) by intraperitoneal injection and exposed to OVA inhalation for 5 days. ADAM28 expression was measured in lung tissue by RTPCR. Airway resistances were measured by Flexivent® during a methacholine challenge. Collagen deposition was measured by using saffron staining, glandular hyperplasia was assessed by counting Alcian blue-stained goblet cells and smooth muscle thickness was evaluated by measuring alpha-SMA positive areas. Bronchial inflammation was assessed in broncho-alveolar lavage cells by manually counting 300 cells. Peribronchial eosinophils were quantified after Congo red staining. Airway Inflammation was also evaluated using a score related to the thickness of inflammatory cells around the bronchi.  
  
**Results:**  
  
ADAM28 expression was increased in mucosa from allergic patients as compared to controls. In mice, ADAM28 was overexpressed in lung parenchyma after OVA exposure.   
ADAM28 KO mice did develop eosinophilic inflammation in a similar extent as WT but less airway remodeling as compared to WT counterparts.   
  
**Conclusions:**  
  
ADAM28 is overexpressed in sputum cells form asthma patients and could play a key role in the remodeling of the airway following eosinophilic inflammation. Further investigations are necessary to confirm these results.

**Keywords:**Proteases, Adamalysins, ADAM, allergy