Endothelial amine oxidase AOC3 transiently contributes to adaptive immune responses in the airways.

Abstract:
Amine oxidase, copper containing 3 (AOC3, also known as vascular adhesion protein-1 (VAP-1)) is an endothelial adhesion molecule that contributes to the extravasation of neutrophils, macrophages, and lymphocytes to sites of inflammation. However, the role of AOC3/VAP-1 in allergic responses remains unknown. Here, we studied eosinophil and CD4+ T-cell recruitment to the airways using AOC3/VAP-1-deficient mice. In an OVA-triggered asthma model, AOC3/VAP-1 slightly contributed to the accumulation of leukocytes in lungs in an age-dependent manner. We then established a new model to kinetically measure recruitment of OVA-specific CD4+ T cells to different airway immune compartments during the priming and effector phases of an adaptive immune response. The results showed that in the absence of AOC3/VAP-1, recruitment of antigen-specific CD4+ T cells to draining bronchial lymph nodes is reduced by 89% on day 3 after tracheal allergen exposure, but this difference was not observed on day 6. The dispersal of effector cells to lung and tracheal mucosa is AOC3/VAP-1 independent. Thus, in allergic airway reactions, AOC3/VAP-1 transiently contributes to the antigen-specific, CD4+ T-cell traffic to secondary lymphatic tissues, but not to airway mucosa or lung parenchyma. Our results suggest a largely redundant
function for AOC3/VAP-1 in allergic inflammatory responses of the airways.