Eotaxins may contribute to both accumulation and elimination of eosinophils in asthma

I read with interest the study by Coleman *et al*[1] on lumen eotaxins and eosinophils, and epithelial brushing eotaxins in chronic asthma. Authors conclude that eotaxin-2 and -3 may contribute to luminal migration of eosinophils.[1] However, potential roles of such transepithelial cell traffic are not discussed. In a recent editorial, Rosenberg highlights the possibility that luminal migration of eosinophils importantly eliminates these cells from diseased bronchial tissues.[2] (Coleman *et al*[1] cite an earlier review by Rosenberg *et al*, dealing with the complex regulation of eosinophil trafficking). A resolving role of luminal migration would complicate interpretation of eosinophil numbers recorded in sputum and broncho-alveoar lavage (BAL) fluid samples. For instance, a negative correlation between lumen and tissue eosinophils occurs at inflammation resolution.[3]

Involvement of eotaxin in luminal migration was implicated in the first experimental in vivo studies in guinea pigs, demonstrating efficient and non-injurious elimination of mucosal tissue eosinophils across the epithelial lining into the airway lumen (ref 3 and references cited therein). In allergen-challenged allergic mice, peak eotaxin-2 and associated eosinophilia, occurred initially in lung tissue and later in BAL fluid.[4] These data agree with a role of eotaxin-2 first in early accumulation of bronchial tissue eosinophils and then in elimination of these eosinophils by luminal migration.

Coleman *et al* state that their data are consistent with a previous report by Ravensberg *et al*[5] of 'increased eotaxin-2 and -3 in the epithelium of patients with asthma following allergen challenge'.[1] Ravensberg *et al* actually demonstrated pronounced immunostaining of eotaxin-2 and -3, particularly in lamina propria, and sustained eosinophilia in that location. A strong positive correlation between eotaxins (-2 and -3) and both the subepithelial eosinophilia and the magnitude of late-phase reaction was also demonstrated[5] agreeing with roles of eotaxin-2 and -3 in retaining disease-driving eosinophils in the tissue.

Three studies have recorded time course of bronchial lumen and tissue eosinophilia in allergen-challenged patients with asthma: consistently, resolution of the allergen exposure-induced asthma is associated with reduced tissue eosinophilia and increased lumen eosinophilia (references cited in ref. 3). Since apoptosis/phagocytosis of bronchial tissue eosinophils has not been compellingly demonstrated,[2 3] these data strongly support the view that luminal migration is a major mode of elimination of eosinophils from diseased asthma tissues.[3] Indeed, the luminal migration mechanism may swiftly eliminate several types of cells (eosinophils, neutrophils, mast cells, lymphocytes, dendritic cells) from diseased mucosal tissues.[3]

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