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Study of patterns of cellular traffic indicative of molecular switches operative in inflammation

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Inflammation and degeneration are the two edged swords that impale a pulmonary system with the maladies like asthma and idiopathic pulmonary fibrosis. To explore critical role players that orchestrate the etiology and pathogenesis of these diseases, we used various lung disease models in mice in specific genetic knockout templates. Acute and chronic allergic asthma and idiopathic pulmonary fibrosis model in mouse was developed in various genetic knockout templates namely $\alpha4^{\Delta/\Delta}$ (α4β1-/-), β2-/-, α4-/-β2-/-, VCAM-1 -/-, gp91phox-/-, MMP-12-/-, gp91phox-/-MMP-12-/-, and ELP-/- mice, and the following parameters were measured to assess development of composite asthma phenotype- (i) airway hyperresponsiveness to methacholine by measuring lung resistance and compliance by invasive and Penh by non-invasive plethysmography as well as lung resistance and compliance using invasive plethysmography, (ii) in situ inflammation status in lung parenchyma and lung interstitium and also resultant airway remodeling measured by histochemical staining namely Masson's Trichrome staining and Hematoxylin & Eosin staining, (iii) formation of metaplastic goblet cells around lung airways by Alcian blue dye, (iv) measurement of Th1 and Th2 cytokines in serum and bronchoalveolar lavage fluid (BALf), (v) serum allergen-specific IgE. We noticed a pattern of cellular traffic between bone marrow (BM)→ peripheral blood (PB) → lung parenchyma (LP) → (BALf) in terms of cellular recruitment of key cell sub-types critical for onset and development of the diseases which is different for maintenance and exacerbations in chronic cyclically occurring asthma that leads to airway remodeling. While inflammation is the central theme of this particular disease, degeneration and shift in cellular profile, subtly modifying the clinical nature of the disease were also noted. In addition we recorded the pattern of cell movement between the secondary lymphoid organs namely, the cervical, axillary, ingunal, and mesenteric lymph nodes vis-à-vis spleen and their sites of poiesis BM, PB and lung tissue. While mechanistic role is the chief domain of the integrins (α4 i.e. VLA-4 or α4β1, VCAM-1; β2 i.e. CD-18 or ICAM-1) and selectins (i.e. E-, L- and P- selectin), oxidative signaling and resulting tissue damage seems to be the forte of the NADPH oxidase and MMP-12 interactions. The following paper thoroughly compares and formulates the pattern of cellular traffic and ancillary signals in the above models and reports some interesting findings with respect to adult lung stem cell niches and its resident progenitors and their role in pathogenesis and disease amelioration.

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