alveolar cell traffic, that Porter highlights, may not be readily made in mice. In sharp contrast to humans, mice lack a bronchial circulation. The dominance of the pulmonary circulation is reflected in the work carried out by Corry et al. These authors repeatedly emphasised that their studies concern parenchymal leucocytes. When egession is inhibited, leucocytes accumulate in pulmonary parenchymal tissues ‘causing’ severely impeded gas exchange. Oxygen is an effective remedy in these lethally affected mice. Corry et al particularly underscore that ‘differences in smooth muscle or other contractile cell function cannot explain the increased mortality observed.’ Hence, Porter’s statement that death in this model is by ‘bronchoconstriction’ is puzzling. Then Porter discusses data suggesting that mouse lung injury evoked by intratracheal bleomycin is caused by neutrophils. Data suggesting that mouse lung injury restriction’ death in this model is by gap in our knowledge concerning clearance of neutrophils cannot explain the increased mortality observed. Cohnheim held classic lectures on in pulmonary parenchymal tissues accumulating in pulmonary parenchymal tissues like peptides are multipotent agents and role in the pathogenesis of sarcoidosis. The authors conclude that Th17 cells are involved in the pathogenesis of sarcoidosis as a multisystem disorder. Interestingly, the paper mentions expression of interleukin (IL)-17 protein by macrophages. Currently, a strong emphasis exists in the literature on the role of Th17 lymphocytes in the production of IL-17 in the lungs. However, Th17 cells are not the only source of IL-17 identified. IL-17 is also known to be produced by γδ and natural killer T cells. It has also been suggested that in human alcoholic liver disease, atherosclerosis and rodent models of lipo-poly saccharide-induced airway inflammation IL-17 can be localised to neutrophils. Furthermore, we have recently demonstrated that IL-17 protein expression is raised in the lower airway of people with advanced cystic fibrosis lung disease. This IL-17 protein expression was immunolocalised to both neutrophils and mononuclear cells. It is known that granulocytes may be part of the inflammatory process in sarcoidosis. The BAL method used by Facco et al which showed that Th17 cells are present in blood, bronchoalveolar lavage (BAL) and lung tissue from people with sarcoidosis. The authors conclude that Th17 cells are involved in the pathogenesis of sarcoidosis as a multisystem disorder. We read with interest the recent paper by Facco et al which showed that Th17 cells are present in blood, bronchoalveolar lavage (BAL) and lung tissue from people with sarcoidosis. The authors conclude that Th17 cells are involved in the pathogenesis of sarcoidosis as a multisystem disorder.

Can cells other than Th17 lymphocytes be important sources of IL-17 in the lungs?

We read with interest the recent paper by Facco et al which showed that Th17 cells are present in blood, bronchoalveolar lavage (BAL) and lung tissue from people with sarcoidosis. The authors conclude that Th17 cells are involved in the pathogenesis of sarcoidosis as a multisystem disorder.

Cohnheim held classic lectures on inflammation. He discussed the resolution of inflammatory infiltrates in mucusally lined organs, specifically noting the advantageous outward transport available to bronchi and lung alveoli. Cohnheim’s contemporary and perpetual authority, Henry Hyde Salter, observed cell-rich sputum production at resolution of severe asthma. Salter intriguingly analysed how the most peripheral airways could be cleared of cellular exudates since coughing would have little impact here. It seems overdue to fill in the large gaps in our knowledge concerning clearance of cells from the human bronchiolar–alveolar lumen.

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REFERENCES


Authors’ response

We thank Dr Brolidie and coworkers for their letter1 and fully agree on the necessity to evaluate whether cells other than lymphocytes and macrophages are involved in IL-17 release in sarcoid lungs. The main manifestation of sarcoidosis is an accumulation of mononuclear inflammatory cells, mostly CD4+ T cells and monocytes/macrophages in involved organs, including the lungs.2 As specified in our ‘Materials and methods’ section, we evaluated cells obtained by filtering bronchoalveolar lavage (BAL) fluid through gauze. A standard morphological and immunological analysis of BAL cellular components was performed. The analysis included cell recovery and differential count of macrophages, lymphocytes, neutrophils