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 emphasise that their studies concern parenchymal leukocytes. When egression is inhibited, leukocytes 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 transepithelial neutrophil egression. We are not equally convinced. For example, n-formyl-methyl-leucyl-phenylalanine (fMLP) may not be used as a specific inducer of neutrophil egression, as quoted by Porter. nFNLP-like peptides are multipotent agents and avidly induce neutrophil toxicity as well.

Nearly 130 years have passed since Julius Cohnheim held classic lectures on inflammation. He discussed the resolution of inflammatory infiltrates in mucosally lined organs, specifically noting the advantageous outward transport available to bronchi and lung alveoli. Cohnheim’s contemporary and perpetual authority, Henry Hyde Salt, observed cell-rich sputum production at resolution of severe asthma. Salt intriguingly analysed how the most peripheral airways could be cleared of cellular exudates since coughing would have little impact here. It seems overdue to find in the large gaps in our knowledge concerning clearance of cells from the human bronchoalveolar lumen.

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

We read with interest the recent paper by Faccò 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. 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-polysaccharide-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 Faccò et al was referenced, via the online supplement, to an original paper that used a 200 ml standard lung function tests.

We thank Dr Brodlie and coworkers for their letter 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. 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

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Authors’ response

We thank Dr Brodlie and coworkers for their letter 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. 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

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