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*From the Authors*:

We thank Dr. Spagnolo and colleagues for their comments on our article (1), which showed that lymphocytosis found on bronchoalveolar lavage (BAL) changed diagnostic perception in 6 of 74 patients with a clinical diagnosis of idiopathic pulmonary fibrosis (IPF) based on high resolution computed tomography (HRCT) (1). Spagnolo and coworkers were surprised about the "typical" BAL profile of patients with IPF in our study. We chose a lymphocyte count below 30% as a cutoff level because analysis by receiver operating characteristics (ROC) demonstrated the highest accuracy with this cutoff. A mild lymphocytosis in the range between 15 and 30% was present in only 3 of 68 patients with IPF, and only 5 of 68 did not show a BAL granulocytosis. In this regard, our BAL findings are not different from the BAL data reported by Spagnolo and coworkers in their letter.

In their comments, Spagnolo and coworkers also emphasized the importance of a surgical lung biopsy for diagnosing IPF in patients with inconclusive HRCT findings, and stated that BAL profiles are unlikely to lead to a "confident" diagnosis of IPF. We agree with this. However, as described in our study, none of our patients had inconclusive HRCT findings. Only patients who fulfilled the criteria for a confident HRCT diagnosis of IPF (subpleural bibasal reticulation and honeycombing) were included. Our selection algorithm started with the identification of patients with such HRCT features, and these patients were labeled as "suspicious for IPF". Patients were then excluded based on the clinical ATS/ERS criteria (2), leaving 74 of 101 patients with a clinical/HRCT diagnosis of IPF. Only with the addition of the BAL analysis were 6 of these 74 patients finally diagnosed as non-IPF. This clearly shows that although BAL profiles are not able to lead to a "confident" diagnosis of IPF, they are able to reveal findings that are not consistent with IPF.

As for the diagnostic procedure for extrinsic allergic alveolitis (EAA), a careful environmental history, as required in the initial evaluation of patients with fibrotic lung disease, was taken in all patients. It is, however, sometimes difficult to get the complete history of antigen exposure, because the range of exposures in EAA is vast. In this context, it is noteworthy that in a series of 85 consecutive patients with EAA, the inciting antigen was not identifiable in 25% of patients, all of whom showed histologic evidence of EAA (3). It has also emerged from recent publications that not only histopathology but also HRCT in some patients with chronic EAA may show a usual interstitial pneumonia pattern (4–6). It is in exactly these patients that a BAL is of additional value because a BAL lymphocytosis may be seen.

Another point to consider is that IPF patients, currently included in clinical trials without surgical biopsy, based on a confident or probable HRCT diagnosis and appropriate clinical criteria, are enrolled without mandatory BAL performance. Based on the results of our study, some of these patients may have unrecognized EAA, and this would certainly lead to recruitment bias in such trials.

***Conflict of Interest Statement***: None of the authors has a financial relationship with a commercial entity that has an interest in the subject of this manuscript.

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## Erratum: Incorrect Cover Caption

*From the Editor*:

Because of a production error, the wrong cover caption was printed for the July 15, 2009 issue of *AJRCCM*. The cover image is based on Figure 4, Panel I from the article by Konishi and colleagues (1), beginning on page 167 of the issue. The correct description of the image should have been worded:

> Cover is based on a photomicrograph showing the coexpression of CCNA2 (*green*) and Ki-67 (*red*), a proliferation marker, in lung tissue obtained from patients with acute exacerbation of idiopathic pulmonary fibrosis.

The figure legend that appears in the article itself is correct. We apologize for any confusion this error may have caused.

EDWARD ABRAHAM
Editor

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