Table 1—Percentage of T-lymphocyte Subsets in Bronchoalveolar Lavage Fluid

<table>
<thead>
<tr>
<th></th>
<th>Lymphocyte % of Total Cells</th>
<th>OKT*</th>
<th>OKT*</th>
<th>OKT*</th>
<th>OKT*/OKT* Ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>Controls (n = 6)</td>
<td>3.5 ± 1.4</td>
<td>71.8 ± 16.5</td>
<td>54.3 ± 9.8</td>
<td>36.1 ± 9.8</td>
<td>1.57 ± 0.52</td>
</tr>
<tr>
<td>Hypersensitivity Pneumonitis exposed (n = 9)</td>
<td>55.8 ± 20.4</td>
<td>91.5 ± 4.7</td>
<td>31.2 ± 11.2</td>
<td>66.3 ± 11.3</td>
<td>0.47 ± 0.22</td>
</tr>
<tr>
<td>Hypersensitivity Pneumonitis not exposed (n = 4)</td>
<td>45.2 ± 24.1</td>
<td>94.0 ± 2.1</td>
<td>33.7 ± 9.0</td>
<td>67.0 ± 8.3</td>
<td>0.47 ± 0.20</td>
</tr>
</tbody>
</table>

Significant difference between groups (t-test) A/B A/B A/B A/B A/B

To the Editor:

We used a smaller lavage volume than Delaval et al (a total of 100 ml saline solution instead of 250 ml). This is why the recovery of total numbers of cells was smaller in our study. If we correct the recovery of cells for the instilled volume, the values of both studies are comparable. In our study, the greater number of cells and lymphocytes in hypersensitivity pneumonitis (HP) did not reach statistical significance compared to sarcoidosis (SARC) due to small case numbers and large SD. In the meantime, having enlarged our study population to 9 patients with recent HP and to 17 patients with active SARC, the difference is significant (t-test): total cell count (×10⁶) in HP 42 ± 10, in SARC 25 ± 18, p < 0.01, lymphocytes (×10³/ml recovery) in HP 519 ± 264, in SARC 207 ± 167, p < 0.01.

We cannot fully explain why we did not see the difference in the amount of OKT* cells between HP and SARC, as Delaval et al did. We think this might be due to different study populations (different criteria for defining active SARC) than due to a more accurate estimation of T-cell subpopulations by the method of Delaval et al. For example, our patients with SARC had a higher percentage of lymphocytes and a higher T/T₈ ratio and thus more T₈* cells per ml BAL recovered than the patients of Delaval et al with SARC. The most obvious difference between both diseases, the 6- to 8-fold higher numbers of OKT₈* cells in BAL of HP compared to SARC was a joint finding in both studies.

Regarding methodology, we think there are clear cut advantages to our method, the immunoperoxidase slide assay, compared to immunofluorescence techniques. These are the use of a light microscope, the durability of the glass slide preparations, simultaneous information about cell morphology, higher sensitivity, and lower amounts of antiserum and especially cells needed for this technique. Thus, by our method, we were able to study multiple surface markers in case of low BAL cell numbers and to report recently that normal smokers (who have only 2.5% lymphocytes among BAL cells) had a markedly decreased helper-suppressor cell ratio in BAL, but not in blood.

Finally, regarding the fact that there are no standardized protocols for BAL technique so far, we are pleased that our findings were widely confirmed by another independent group using different lavage procedures, a different technique for surface marker analysis, and maybe differently selected patients.

In response to Drs. Pesci, Bertorelli and Marchioni, we wish to point out that we wrote in our article: "In patients with hypersensitivity pneumonitis (HP) not exposed to antigens for more than five days, the OKT₈/OKT₆ ratio may be increased." We are well aware that this limit of 5 days defined in our 8 patients is somewhat arbitrary, and that single cases may still have lowered OKT₈/OKT₆ ratios also after this limit. On the other hand, one of the purposes of our paper was to show that there are serial changes in lymphocyte subpopulations of bronchoalveolar lavage (BAL) fluid during the course of disease, and that these changes consist of an increase in BAL helper (OKT₈*) and a decrease in suppressor (OKT₆*) cells. That this concept may be true was recently confirmed by a fourth patient whom we were able to re-lavage after 2 years. His data are shown in Table 2. He actually followed the same pattern as the 3 patients who were re-lavaged and reported in our article under debate.

Another aspect is that we are often not completely sure whether patients with HP were really not exposed to antigens any longer. It may be that it also depends on the amount of antigens having entered the lungs as to how fast these changes in lymphocyte subsets will occur after avoidance of further antigen exposure. How long were the 4 patients studied by Pesci et al not exposed to antigens: for days, for months, or for years? Our patients were not exposed for 5 days (T/T₈ ratio 3.0), for 6 days (T/T₈ 1.3), for 14 days (T/T₈ 1.3), and for 12 months (T/T₈ 5.5), respectively.

Clearly, further studies are needed doing serial lavages in more patients with HP to answer these questions and to fully clarify the role of T cells subsets during the course of disease.

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REFERENCE

ACTH Therapy in Status Asthmaticus

To the Editor:

ACTH therapy was effectively used during the past 8 years in treating 15 patients with status asthmaticus who failed to respond to intensive therapy which included IV hydration, O₂, adequate doses of IV aminophylline, massive doses of Solucortef, sympathomimetic drugs, and respiratory physiotherapy.

In case of failure of the patient to respond to an adequate dosage of a corticosteroid drug for at least 48 hours in the absence of acidosis,