vivo T cell depletion with ATG and graft manipulation. Three patients with remission of psoriasis (without arthropathy) after PBSCT due to a haematological disease relapsed within 6–14 months as described in a previously published report. However, skin involvement was not of major concern in our patient.

We conclude that it may be worth exploring PBSCT further in young patients with mutilating psoriatic arthropathy who are resistant to disease modifying antirheumatic drugs and for whom tumour necrosis factor α antagonist treatment has failed.

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Peripheral blood lymphocyte phenotypes in patients with spondyloarthritis
A Plonquet, A Gleizes, K Briot, P Maisin, X Chevalier, J-P Farce, P Claudepierre

A prevalent hypothesis in the pathogenesis of the spondyloarthropathies (SpA) is that gut lymphocytes primed by antigens in the gut lumen may enter the bloodstream and home in on target tissues—for example, the entheses and synovial membranes, where they may initiate a local immune process responsible for further local inflammation. Selective homing of lymphocytes is a well known process that is made possible, in part, by the lymphocyte phenotype acquired as a response to antigenic stimulation and characterised by a subset of receptors (such as integrins) associated with targeted trafficking. Among integrins, \( \alpha_4 \beta_7 \) is expressed by T lymphocytes in or adjacent to mucosal epithelia, and exhibits features that indicate a possible role in this pathogenic process. To our knowledge, there are no data on the markers for activation and adhesion of circulating lymphocytes in the SpA. The main objective of this study was to look for differences in \( \alpha_4 \beta_7 \) (CD103) blood lymphocyte counts between patients with active and those with inactive SpA, using flow cytometry. We also studied markers for other adhesion molecules, lymphocyte subsets, and lymphocyte activation.

PATIENTS, METHODS, AND RESULTS
Twenty patients meeting European Spondylarthropathy Study Group criteria or Amor’s criteria for SpA were enrolled in the study, together with control patients followed up for chronic degenerative low back pain. They did not have any other underlying disease and only had a non-steroidal anti-inflammatory drug (NSAID) or acetaminophen as current treatment. All patients and controls gave their written informed consent to participation in the study, which was approved by the local ethics committee. Table 1 shows the main characteristics of the patients. The 20 control patients had a mean (SD) age of 39.8 (9.6) years (p>0.05 v the SpA group) and six of them were men. Flow cytometry was performed using a Coulter EPICS(r) XL within 24 hours after staining. List mode parameters were analysed and stored on System II software (Beckman-Coulter).

The studied markers (absolute count and/or percentage of positive cells) were compared between the patients with SpA and the controls, and between the patients with SpA with active disease and those with quiescent or controlled disease. Active disease was defined a priori as a Bath Ankylosing Spondylitis Disease Activity Index (BASDAI) score >30 with a C reactive protein (CRP) level >15 mg/l (Mann-Whitney test). The mean (SD) percentage of CD103 lymphocytes did not differ between the patients with SpA (0.9 (0.4%) and controls (1 (0.6%)], but was significantly lower in the patients with active SpA (0.66 (0.26%)) than in those with inactive SpA (1.2 (0.7%), p<0.05). Except for a small but significant decrease in the percentage of CD49d positive cells in the patients with SpA as compared with the controls, no significant differences between the groups with active and inactive SpA were seen for any of the three other integrin markers (CD11a, CD29, and CD49d). The frequencies of the main CD4 and CD8 T cell subsets and NK cells were not significantly different between the patients with SpA and controls or between the groups with active and inactive SpA. However, there was a trend toward an increase in the CD3+ lymphocyte count in the group with active SpA, which was

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Patients with active spondyloarthritis (SpA) may reflect different immunological accumulations of these cells in tissue and add to earlier observations seen in patients with active SpA, which may suggest an involvement of CD8+ T cells expressing L-selectin observed in the same subgroup of patients with active SpA, lymphocytes, although the difference was not statistically significant. None of the between-group differences for these molecules (CD25, CD38, DR, CD45RA-RO), which can be considered markers of lymphocyte activation, were statistically significant.

**DISCUSSION**

This study provides the first data on blood lymphocyte phenotypes according to disease activity in patients with SpA. It found no differences in peripheral blood lymphocyte phenotypes between patients with SpA and controls. However, a decrease of lymphocytes expressing αβ integrin was seen in patients with active SpA, which may suggest an accumulation of these cells in tissue and add to earlier evidence that T cells expressing αββ may be involved in the pathogenesis of SpA. The increase in absolute counts of CD8+ T cells expressing L-selectin observed in the same subgroup of patients with active SpA may reflect different immunological processes and also deserves further study.

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**REFERENCES**


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**Table 1** Demographic data and main disease characteristics in the 20 patients with spondyloarthritis (SpA)

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>All patients with SpA (n = 20)</th>
<th>Active disease (n = 7)</th>
<th>Inactive disease (n = 13)</th>
<th>p* (active v inactive)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>35.4 (9.1)</td>
<td>36.4 (7.5)</td>
<td>34.8 (10.1)</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>Sex ratio, M/F</td>
<td>13/7</td>
<td>6/1</td>
<td>7/6</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>Disease duration (years)</td>
<td>8.2 (5.6)</td>
<td>10.1 (3.1)</td>
<td>7.1 (6.5)</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>Sacroiliitis</td>
<td>14</td>
<td>6</td>
<td>8</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>Psoriasis</td>
<td>4</td>
<td>2</td>
<td>2</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>Presence of E27</td>
<td>16†</td>
<td>5</td>
<td>11</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>BASDAI score</td>
<td>50.7 (22.7)</td>
<td>59.4 (13.5)</td>
<td>45.7 (25.9)</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>BASFI score</td>
<td>40 (26.6)</td>
<td>64.2 (13)</td>
<td>23.9 (20)</td>
<td>0.02</td>
</tr>
<tr>
<td>ESR (mm/1st h)</td>
<td>22.4 (20)</td>
<td>39.5 (26.4)</td>
<td>14.5 (10.3)</td>
<td>0.05</td>
</tr>
<tr>
<td>CRP (mg/l)</td>
<td>18.2 (22)</td>
<td>44.8 (17.8)</td>
<td>5.8 (8.6)</td>
<td>0.002</td>
</tr>
</tbody>
</table>

Results for quantitative parameters are given as means (SD).
*Comparisons were performed using the χ2 test or Fisher’s exact test for qualitative parameters and the Mann-Whitney test for quantitative parameters; †two not done.

BASDAI, Bath Ankylosing Spondylitis Disease Activity Index; BASFI, Bath Ankylosing Spondylitis Functional Index; ESR, erythrocyte sedimentation rate; CRP, C reactive protein.

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