The Expression of Adhesion Molecules, including CD11a and CD11b, on the Human T Lymphocyte Surface Membrane in Patients with Bronchial Asthma

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Summary: The $\beta$-2 integrin consists of lymphocyte function-antigen-1 (LFA-1, CD11a/CD18) which binds to the major ligand intracellular adhesion molecules 1 and 2 (ICAM-1 and ICAM-2), Mac-1 (CD11b/CD18, ligand iC3b) and p150,95 (CD11c/CD18, ligand iC3b). These molecules function not only as adhesion molecules but as signal transduction agents. To investigate the expression of an integrin family in asthma, lymphocyte subsets were assessed using bronchoalveolar lavage fluid (BALF) cells obtained from asthma patients. The relationship between the integrins and lymphocyte subpopulations and their activation state in the BALF was analyzed using two-color flow cytometry in 15 subjects with asthma and in 13 control subjects with chronic bronchitis. The absolute number of CD11a+CD4+ lymphocytes was significantly greater in the asthmatics than in the non-smoking subjects with chronic bronchitis. In the asthmatics, the expression of the CD11a+CD4+ lymphocytes was significantly higher than the expression of CD11a+CD8+ lymphocytes. The CD2+ lymphocytes had a significant positive regression with the CD11a+CD4+ lymphocytes in the asthmatics. Analysis of BALF revealed the representation of integrins for the ongoing bronchial transfer of information, and may provide an approach to obtain evidence about the inflammatory processes on the surface of the bronchioles in asthma.

Key words: adhesion molecules — T lymphocyte — bronchial asthma — bronchoalveolar lavage — CD11a — CD11b

Introduction

Bronchial asthma (BA) has the characteristic feature of inflammation which leads to bronchial hyperreactivity (Chung, 1986; Kay, 1991). There is increasing evidence that T cells, as well as eosinophils, play a central role in the pathogenesis of BA. During an exacerbation of BA, the numbers of activated T cells increase in peripheral blood; and activated lymphocytes and eosinophils in both blood and bronchoalveolar lavage fluid (BALF) are fundamental to

The cells, cytokine producing and effector cells, interact with each other in two ways, by producing soluble cytokines such as interleukins, interferons, and tumor necrosis factor, and by expressing molecules on the surface of leucocytes (receptors) and potential target cells (ligands) (Editorials, 1990). These interactions between receptors and ligands are critical for both specificity and function of cell adhesion molecules.

The β-2 integrin, known as the leucocyte integrin receptor family, consists of three components as follows: LFA-1, which binds to the major ligand intracellular adhesion molecules 1 and 2 (ICAM-1 and ICAM-2); Mac-1 (ligand iC3b); and p150, 95 (ligand iC3b). These molecules belong to the CD11/CD18 heterodimer complex, and are predominantly involved in immune adherence (Editorials, 1990).

Some precedents indicate that the co-stimulatory role of LFA-1 is essential not only for adhesion, but also for signal transduction. CD11a moAb can enhance the T cell response to immobilized CD3 moAb and can induce activation of long term T-cell lines (van Noesel et al. 1988; van Seventer et al. 1990). Furthermore, expression of adhesion molecules is not a static process and can be modified by diverse extracellular and intracellular factors.

Alteration in expression of adhesion molecules may be quantitative or qualitative (Arnaout, 1990; Editorials, 1990; Shimizu et al. 1990). The existence of multiple adhesion pathways, of multiple ligands for a single receptor (such as LFA-1/ICAM-1 and LFA-1/ICAM-2), and of regulation of ligand expression (ICAM-1) provides opportunities for cooperativity, redundancy and diversity that the T-cell utilizes to exquisitely regulate its adhesive interactions (Shimizu et al. 1990).

In the present study, the relationship between integrins and T-lymphocyte subpopulations in BA and their activation state was investigated using BALF obtained from subjects with stable asthma. This was compared to results from control subjects with chronic obstructive pulmonary disease (chronic bronchitis).

**Methods**

**Subjects**

Alveolar lymphocytes were obtained from BALF from 15 patients with BA and 13 control subjects with chronic bronchitis (CB). All subjects with BA (8 females and 7 males, mean age 43±4 yr, range 27 to 65 yr) met the definition for BA of the American Thoracic Society (Dantzker et al. 1987). At the time of the study, the patients had stable asthma without administration of a steroid, were not in acute respiratory distress, and showed no evidence of a pulmonary infection. All participating asthmatics were non-smokers, and 5 of the 13 control subjects with CB were smokers. Informed consent was obtained orally. A summary of the clinical and laboratory data for the individual patients with asthma is presented in Table 1. The control subjects (10 males and 3 females, mean age 51±5 yr, range 21 to 74 yr) had CB and no
history of allergies or asthma.

**Bronchoalveolar lavage (BAL)**

BAL was performed according to the technical recommendations and guidelines of the Task Group on BAL of the European Society of Pneumology (Klech and Hutter, 1989). A solution of NaCl (3±50 ml of 0.9%) was instilled into the middle lobe using a bronchofiberscope BF1T-20 (Olympus, Tokyo), immediately aspirated into plastic syringes, and kept at 4 °C.

**Analysis of leukocytes and lymphocytes**

BALF samples were filtered through a one-layer gauze. The total number of nucleated cells in the recovered BALF was counted using a Bürker-Türk counter board (NITIRIN, Tokyo). Cells were sedimented by centrifugation at 500 g for 10 min. Cytological examination of the BAL fluid was performed after the cytocentrifugation using Cytospin II (SHANDON, England) and May-Grünwald Giemsa stain, followed by differential counting of 100 cells. An aliquot of the BAL cells (5±10⁶ cells) was incubated in the presence of saturating concentrations of fluorescein- or phycoerythrin-conjugated moAb in the dark at 4 °C for 30 min. The erythrocytes were lysed by adding 10 ml of lysing solution (8.29 gm ammonium chloride, 1 gm potassium bicarbonate, and 37 mg ethylenediaminetetraacetic acid in 1 L of distilled water at pH 7.3) for 5 min. The leukocytes were washed twice with phosphate-buffered saline containing 0.1% sodium azide at pH 7.4. Two-color flow cytometry for cytofluorometric analysis was performed using a FACScan (Becton Dickinson, Sunnyvale, CA) with Lysis software (Becton Dickinson). An electronic gate was set on the lymphocytes on the forward and side scatter plot. A total of 25,000 cells were counted for surface antigen analysis.

**Monoclonal antibodies**

The monoclonal antibodies, anti-human leukocyte function-associated antigen 1, α-chain (LFA-1) (CD11a) and anti-human C3bi receptor (CD11b) for fluorescein isothiocyanate (FITC), were purchased from Immunotech (Marseille, France). CD4 and CD8 for Phycoerythrin (PE) were obtained from Becton and Dickinson (Mountain View, CA), CD2 for PE from DAKO (Glostrup, Denmark) and anti-HLA-DR, CD20 for FITC from Coulter (Hialeah, FL).

**Statistics**

Statistical analysis was performed by the Mann-Whitney U test. Differences associated with a p<.05 were considered to be significant. Coefficients and their statistical significance were determined by Pearson’s linear regression analysis. All values are expressed as mean ± standard error of the mean (SEM).

**Results**

**Evaluation of ventilatory function**

The results of the pulmonary function tests are summarized in Table 1. These tests were performed during attack-free periods in the patients with BA, and during pulmonary distress-free and infection-free periods in the patients with CB. There was no statistically significant difference in the estimated values for the ventilatory functions or in
TABLE 1.
Patient characteristics*

<table>
<thead>
<tr>
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<th>Control (Chronic Bronchitis)**</th>
<th>Asthmatics</th>
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<tbody>
<tr>
<td>Number of patients</td>
<td>6</td>
<td>7</td>
</tr>
<tr>
<td>Mean age (yr)</td>
<td>44±7</td>
<td>57±7</td>
</tr>
<tr>
<td>Sex, F/M</td>
<td>0/6</td>
<td>3/4</td>
</tr>
<tr>
<td>Duration (month)</td>
<td>17.3±6.8</td>
<td>5.1±1.5</td>
</tr>
<tr>
<td>FEV1 (L)</td>
<td>3.2±.2</td>
<td>2.6±.2</td>
</tr>
<tr>
<td>FEV1 (%predicted)</td>
<td>810±4.7</td>
<td>69.3±3.2</td>
</tr>
<tr>
<td>FVC (L)</td>
<td>4.1±.2</td>
<td>3.2±.3</td>
</tr>
<tr>
<td>%VC (%)</td>
<td>110.4±1.8</td>
<td>100.4±6.7</td>
</tr>
<tr>
<td>PaO2 (torr)</td>
<td>88.5±4.7</td>
<td>91.2±1.9</td>
</tr>
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</table>

* Clinical data and results of lung function tests from the patients enrolled in this study (mean±SEM).
** Patients with chronic bronchitis were controls.
FEV1 (L): forced expiratory volume in 1 sec (L); FEV1 (%predicted): forced expiratory volume in 1 sec as percent of FVC; FVC (L): forced vital capacity (L); %VC: vital capacity as percent of predicted VC; PaO2 (torr): partial pressure of arterial O2 (torr)

TABLE 2.
Differential cell counts in BALF*

<table>
<thead>
<tr>
<th></th>
<th>Control (Chronic Bronchitis)**</th>
<th>Asthmatics</th>
</tr>
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<tbody>
<tr>
<td>Fluid recovery(%)</td>
<td>74.5±9.5</td>
<td>47.1±7.7</td>
</tr>
<tr>
<td>Cells/ml BAL,×10⁶</td>
<td>1.8±.6</td>
<td>2.1±.5</td>
</tr>
<tr>
<td>%Alveolar macrophages</td>
<td>77.8±8.7</td>
<td>79.1±7.2</td>
</tr>
<tr>
<td>%Lymphocytes</td>
<td>19.8±9.1</td>
<td>19.6±7.2</td>
</tr>
<tr>
<td>%PMNs</td>
<td>2.2±1.1</td>
<td>.7±.4</td>
</tr>
<tr>
<td>%Eosinophils</td>
<td>0.0±.0</td>
<td>0.0±.0</td>
</tr>
</tbody>
</table>

* Results from analysis of bronchoalveolar lavage fluid (mean±SEM).
** Patients with chronic bronchitis were controls.
† Values significantly different from non-smoker control group (p<.05).
†† Values significantly different from both control groups (p<.05).

the arterial oxygen tensions among the three patient groups, the asthmatics and two control groups of patients with CB divided into smokers (6) and non-smokers (7). Thus all the patients enrolled in this study had neither an obstructive nor a restrictive ventilatory disturbance and no remarkable hypoxemia at the time of the BAL.

Distribution of leukocyte and lymphocyte subpopulations in BALF

As shown in Table 2, when the recoveries of BALFs were compared among the three groups, the mean recovery percentage was significantly greater
(p<.05) in the asthmatics than in the non-smoker control group. The mean total cell count/ml of BALF appeared to vary slightly among the three groups from 1.5 to $2.1 \times 10^6$, but the differences were not significant. The differential cell counts also revealed no significant differences in the percentages of alveolar macrophages, lymphocytes or PMNs. However, eosinophils were only found in the BALF from the asthmatics with a mean percentage of 3.1.

**Integrin expression**

The integrin expression on the surface of the lymphocytes was studied using MoAbs that recognize CD11a and CD11b, and the results are shown in Table 3. CD11a-antigen was detected on the surface of more than 80% of the lymphocytes in the BALF from all three groups and the CD11b antigen on less than 12.7%. The absolute number of CD11a expressing lymphocytes appeared to be greater in the asthmatics than in the control groups, although the difference was not significant.

Two surface antigens, CD4 and CD8, on T-lymphocytes in BALF were examined by two-color flow cytometry. As illustrated in Fig.1A and shown in Table 3, the absolute number of CD11a+CD4+ lymphocytes in the asthmatics was significantly (p<.05) greater than in the non-smoker control CB group, while there were no differences between the subsets of the CD11a+CD8+ antigen. As shown in Table 3, in the asthmatics and in both control groups the percentage of CD11a+ lymphocytes was 7 to 12 times greater than the percentage of CD11b+ lymphocytes. The absolute num-

<table>
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<th>TABLE 3.</th>
<th>Lymphocyte subpopulations in BALF*</th>
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<tr>
<td>Epitope</td>
<td>Control (Chronic Bronchitis)</td>
</tr>
<tr>
<td></td>
<td>smokers (%)</td>
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<tr>
<td></td>
<td>n=6 ($\times 10^6$)</td>
</tr>
<tr>
<td>CD2</td>
<td>84.7±12.5 (14.2±5.2)</td>
</tr>
<tr>
<td>CD4</td>
<td>48.0±2.0 (8.9±3.0)</td>
</tr>
<tr>
<td>CD8</td>
<td>37.2±6.8 (5.2±2.3)</td>
</tr>
<tr>
<td>CD11a</td>
<td>86.9±6.2 (14.5±5.3)</td>
</tr>
<tr>
<td>CD11b</td>
<td>7.1±2.3 (.9±.4)</td>
</tr>
<tr>
<td>CD4+CD11a+</td>
<td>47.5±6.7 (8.9±3.0)</td>
</tr>
<tr>
<td>CD8+CD11a+</td>
<td>36.7±6.7 (5.1±2.3)</td>
</tr>
<tr>
<td>CD4+CD11b+</td>
<td>5.5±2.6 (.6±.3)</td>
</tr>
<tr>
<td>CD8+CD11b+</td>
<td>2.0±.4 (.3±.2)</td>
</tr>
<tr>
<td>HLA-DR+</td>
<td>32.7±12.8 (8.9±3.0)</td>
</tr>
<tr>
<td>CD2+DR+</td>
<td>30.2±12.5 (7.5±4.7)</td>
</tr>
<tr>
<td>CD4/CD8 ratio</td>
<td>2.5±1.0</td>
</tr>
</tbody>
</table>

* Results from analysis of bronchoalveolar lavage fluid (mean±SEM). Each value represents the percentage of lymphocytes positive for each antigen. The absolute numbers of lymphocytes are shown in parentheses.
† Values significantly different from control (p<.05).
Number of lymphocytes expressing both CD4 and CD11a was 1.9 times more than the number expressing both CD8 and CD11a in asthmatics (p<.05), while the absolute number of CD11b+ lymphocytes expressing CD4+ or CD8+ antigen was almost equal in asthmatics (Fig. 1A).

**T-lymphocyte activation antigens**

As shown in Table 3 and in Fig.1B, the absolute number of CD2+ lymphocytes was slightly greater in the asthmatic patients than in the control groups, and the number of HLA-DR+CD2+ lymphocytes was also greater in the asthmatic.

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_Fig. 1._ Distribution of lymphocytes obtained from BALF of 15 asthmatics and 7 non-smoking subjects with CB in A, B and C. The p values are shown within the figures as calculated from the Mann-Whitney U test for comparing given markers respectively. The mean values for the absolute number of positive cells are indicated as open circles and squares, and the standard errors are indicated by the vertical lines. A: Distribution for LeuCAM expression on CD4+ lymphocytes and CD8+ lymphocytes in BALF. The absolute numbers of CD11a+CD4+, CD11a+CD8+, CD11b+CD4+ and CD11b+CD8+ antigens were compared in the BA and non-smoker CB groups. B: The distribution of T lymphocyte-activation antigens in BALF. The absolute numbers of CD2+ antigens, HLA-DR+ antigens and CD2+DR+ antigens were compared in non-smoking subjects with CB and asthmatics. C: Distribution of CD4+ lymphocytes and CD8+ lymphocytes and the CD4/CD8 ratio in BALF. The absolute number of CD4+ antigens and CD8+ antigens and the CD4/CD8 ratio are described in the figure.
patients than in the non-smoker control group, but smaller than in the smoker control group.

**CD4+, CD8+ antigen**

As illustrated in Fig.1C and shown in Table 3, there was no significant difference in the percentages of CD4+ lymphocytes of the asthmatics and control groups. The absolute numbers of both CD4+ lymphocytes and CD8+ lymphocytes were greater in the asthmatics than in the control groups, but the differences were not significant.

**CD4/CD8 ratio**

The CD4+/CD8+ ratio was largest in the smokers, followed by the asthmatics and non-smokers (Table 3).

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**Fig. 2.** Correlation between CD2+ lymphocytes and leuCAM antigen lymphocytes in BALF from individuals with asthma. A: Correlation between the percentages of CD2+ lymphocytes (x-axis) and CD4+ lymphocytes (y-axis). B: Correlation between the percentages of CD2+ lymphocytes (x-axis) and CD4+CD11a+ lymphocytes (y-axis). C: Correlation between the percentages of CD2+ lymphocytes (x-axis) and CD4+CD11b+ lymphocytes (y-axis). D: Correlation between the percentages of CD2+ lymphocytes (x-axis) and CD11b+ lymphocytes (y-axis). Regression lines, correlation coefficients (r), and statistical significance (p) were calculated using Pearson's linear regression analysis.
Regression

A significant correlation was found between the percentages of CD2+ lymphocytes and CD4+ lymphocytes with \( r = 0.667, \ p = 0.006 \) (Fig. 2A). Furthermore, the percentage of CD2+ lymphocytes showed a positive regression with the percentage of CD4+CD11a+ lymphocytes with \( r = 0.673, \ p = 0.006 \) (Fig. 2B).

Conversely, there were significant negative regressions between the percentages of CD2+ lymphocytes and CD4+CD11b+ lymphocytes with \( r = -0.737, \ p = 0.002 \) (Fig. 2C), and between the percentages of CD2+ lymphocytes and CD11b+ lymphocytes with \( r = -0.904, \ p = 0.001 \) (Fig. 2D).

The above observations clearly demonstrate that the increase in the number of CD4+CD11a+ lymphocytes was accompanied by an increase of CD2+ lymphocytes. Therefore it is plausible in asthmatic patients, in whom the absolute number of CD4+CD11a+ lymphocytes in BALF increases in parallel to the CD2+ lymphocytes, that the former subset may play an important role in the pathogenesis of the bronchial inflammation.

Discussion

In the present study of lung function, surface markers on alveolar lymphocytes in BALF were obtained from patients with BA or CB. The patients, who were at a stable state of asthma, had less obstructive dysfunction in terms of FEV\(_1\) and FEV\(_1\)/%predicted than reported in previous studies (Walker et al. 1991; Gratziou et al. 1992), and was almost equal to that from normal volunteers in previous studies; mainly because our patients with asthma were less severe than those in previous studies, as indicated by the lung function tests. On the other hand, it is known that the airways of patients with CB are easily collapsed by aspiration. Therefore, the low recovery efficiency of BALF in CB could have resulted from the easily collapsed peripheral airways. The total number of cells in the BALF of BA was less than that of controls, although the fluid recovery was greater. This was due to a reduction in the number of alveolar macrophages in BA (Walker et al. 1991; Gratziou et al. 1992). As in previous studies, the differential cell counts showed no significant differences between the two patient groups, except for eosinophils which were absent in the BALF from the controls.

In inflammatory responses, the importance of adhesion molecules has been demonstrated, especially for congenital diseases. There is an autosomal recessively inherited defect in the \( \beta_2 \) subunit of leukocyte integrins called leukocyte adhesion deficiency (Anderson and Springer, 1987). This is manifested clinically as recurrent severe bacterial infections, and demonstrates the critical role of \( \beta_2 \) integrins especially in the firm adhesion phase of neutrophilic inflammatory responses. Expression of adhesion molecules is not a static process.

An alteration in the expression of adhesion molecules may be quantitative or qualitative (Arnaout, 1990). Acute activation of the T cell induces a rapid but transient integrin binding function, and a higher expression of each integrin

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on memory T cells compared to naive T cells results in greater binding of memory cells to each ligand (Shimizu et al. 1990).

Integrin molecules could change in structure spontaneously from a non-activated type to an activated type, which could cause increased binding to ligands. In due course, they could attach to each other, and conduct information (Springer, 1990; Hynes, 1992).

On the other hand, an increase in integrins could conduct information by attaching to the cells. This might occur quantitatively and/or qualitatively on the surface of the lymphocytes in BA. Few reports have described this possibility.

Van Noesel et al. (1988) reported that the binding of the ligand to LFA-1(CD11a/CD18 heterodimer) resulted in the transduction of regulatory signals across the plasma membrane with signal-modifying properties involved in the adhesion of T cells to target cells.

In the present study, the absolute number of CD11a+ CD4+ lymphocytes in BA was significantly greater than in the non-smoker CB subgroup, as determined by two-color flow cytometry. The absolute number of CD11b+ lymphocytes tended to increase, but not significantly. In contrast, the absolute number of CD11a+ CD4+ lymphocytes was significantly (p<.05) greater than the number of CD11a+ CD8+ lymphocytes in BA. The CD11a+ lymphocytes were located mainly on the surface of the CD4+ lymphocytes in BALF of BA. Therefore, CD11a+CD4+ antigens, an integrin family, could play meaningful roles on the surface of the lymphocytes in BA. Consequently, the change in function of the transduction of information through integrin molecules in BA seems to be more quantitative than qualitative. Sensitized T lymphocytes, when exposed to specific allergens, respond by expressing a range of cell adhesion molecules including LFA-1, which could interact with intercellular adhesion molecule-1 (ICAM-1) known to be expressed by bronchial epithelial cells (Jeffery et al. 1989). CD11a+CD4+ lymphocytes might be a useful marker for grading inflammation severity in BA. Because the study included only a few cases of severe BA, no significant correlation was found between this surface marker and the indicators of lung function.

If CD4+ helper T lymphocytes induce or potentiate inflammation and airway hyperreactivity in BA, CD8+ suppressor T cells may serve to suppress or prevent asthmatic responses in some circumstances (Gonzalez et al. 1987). The fact that CD4+ lymphocytes decrease in numbers after local segmental allergen challenge strongly supports the participation of T-lymphocytes in the development of the early allergen-induced inflammatory response in BA (Gratziou et al. 1992).

It was shown in the present study that the number of CD4+ lymphocytes did not increase significantly, but the patients at the time of the BAL were supposed to have had a low opportunity for allergen-induced inflammation. As a result, no distinct correlation could be found between the inflammation of the airways and the hyperreactivity in the patients with BA. The numbers and types of T lymphocytes in BALF may differ with the timing of the BAL, the type of asthma, and/or the severity of the disease. The absolute number of CD8+ lymphocytes increased in the BALF
from BA as compared to CB. Previous studies found increased numbers of CD8+ T lymphocytes in the BALF of asthmatics (Gonzalez et al. 1987). A similar result was found in this study. This is mainly because asthmatics who develop a late phase reaction (LPR) in response to allergens may have a relatively higher ability to recruit CD8+ T lymphocytes than those subjects who respond with only an early reaction to allergens.

The CD4/CD8+ ratio in the smoker CB subgroup was slightly greater than that in the BA and non-smoker CB groups, but the differences were not significant. The present study and previous studies (Walker et al. 1991; Gratziou et al. 1992) have demonstrated that some stimulants can increase of the ratio. An increase in the number of CD8+ lymphocytes in the BALF from the BA group reduced the CD4/CD8 ratio below that of the CB group (Gratziou et al. 1992).

The CD11b antigen is one component of the Mac-1 molecule in the integrin family, which is usually expressed on monocytes, neutrophils and some lymphocytes including NK cells and it recognizes iC3b receptors in the immunocomplex. The CD11b is not only a receptor for phagocytosis of opsonized substances but functions as a co-factor in the adhesion to other cells (Yokoyama, 1990). The results of this study show that the expression of CD11b was significantly lower than that of CD11a in the BA group. In addition, as surface markers of lymphocytes in BA, both CD11b+CD4+ antigen and CD11b+CD8+ antigen were almost equally expressed. Therefore, the CD11b+ antigen could not function sufficiently and quantitatively in BA. A qualitative assessment in BA was difficult in this study.

The CD2 antigen is one component of the LFA-2 immunoglobulin supergene family that has a counterreceptor of LFA-3, and is usually expressed on all T-lymphocytes. This antigen is known not only as an activation molecule for T-lymphocytes but as an adhesion molecule. The absolute number of CD2+ antigens was greater in the BA group than in the CB group. This means that CD2+ lymphocytes might have a higher capability as adhesion molecules.

Previous studies have evaluated the expression of three surface proteins, IL-2R, very late activation antigen-1 (VLA-1) and HLA-DR, associated with T lymphocyte activation in status asthmatic patients as compared to patients with chronic obstructive airway disease and/or mild asthma (Corrigan et al. 1988). Furthermore, T lymphocyte activation was evidenced by an increased density of HLA-DR (Kay, 1992) in endothelial cells in allergen-challenged biopsied specimens. The present study showed that the number of HLA-DR+ lymphocytes in BA was greater than in the non-smoker controls, but smaller than in the smoker controls. Therefore, the subjects might have had a slightly activated asthma.

Almost no previous studies have described the changes of CD2, CD4, CD11a and CD11b antigens on lymphocytes obtained from BALF. The present data showed a significant positive correlation between the CD2+ lymphocytes and the CD4+ lymphocytes, and between the CD2+ lymphocytes and the CD4+ CD11a+ lymphocytes. This evidence indicates that the number of CD4+ CD11a+
lymphocytes increased in parallel to the total number of T lymphocytes in BA. Therefore, the CD4+CD11a+ antigen functions as an integrin family and a leading transferring agent of information in BA.

BALF consists of washings from alveolar and bronchiole surfaces and therefore does not necessarily reflect the events within the respiratory tissues. However, the present data indicated a close correlation between the CD2+ lymphocytes and the CD4+CD11a+ lymphocytes. The results are probably representative of the ongoing bronchial inflammatory processes in BA. Analysis of BALF provided additional evidence for an important role for adhesion molecules, such as integrin on T lymphocytes, in the pathogenesis of BA. A relationship of adhesion molecules on the surface of lymphocytes is fundamental to the conduction of information, the pathogenesis and the clinical manifestations of asthma.

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References


