Infection with the LAV/HTLV-III virus leading to the clinical diseases of lymphadenopathy syndrome (LAS) or acquired immune deficiency syndrome (AIDS) goes along with marked dysregulation of the immune system (1, 2). Apart from well-known cellular immune defects observed in vitro and in vivo (3), a number of abnormalities of the humoral immune response have been described: polyclonal B-cell activation leading to increased IgG and IgA production with rather weakened production of IgM and increased response to neoantigens together with increased amounts of circulating immune complexes (1, 3, 4). There is little information on the role of IgE in this condition. This may be due to the fact that most laboratories tend to heat infected sera at 56°C for 30 min thereby destroying IgE antibodies.

PATIENTS AND METHODS
We studied 69 patients of our AIDS outpatient clinic for the presence of atopic diseases in their personal and family history as well as for total serum IgE values (measured by paper-radio-immuno-sothent-test (PRIST), Pharmacia, Uppsala) and specific IgE-antibodies (radio-allergo-sorbent test (RAST) Pharmacia, Uppsala) to 19 common environmental allergens.

Three groups of patients were compared: group I: LAV/HTLV-III-negative healthy homosexuals (n=39); group II: LAV/HTLV-III-positive healthy homosexuals (n=10); group III: patients with LAS or AIDS (n=20), among them four cases with full-blown AIDS including Kaposi's sarcoma.

RESULTS
As Table I shows, the highest IgE serum values and the highest frequency of positive RAST results were found in group I (31% of the patients showing specific IgE antibodies...
to one or more of 19 allergens tested (6 x birch, 5 x grass, 5 x cat epithelium, 3 x dermatophagoides pteronyssinus, 2 x horse, 2 x mugwort, 2 x penicillium, 1 x dog, 1 x alternaria, 1 x ovalbumin, 1 x cockroach dander).

In groups II and III on the contrary serum IgE levels were considerably lower (130 kU/l or 160 KU/I respectively compared to 570 kU/l median in group I). In the qualitative analysis 25% of group I showed markedly elevated IgE values (>400 kU/l) compared to only 10% in group II and 15% in group III.

Interestingly the elevated serum IgE values in two patients with AIDS were accompanied by negative RAST results. Only two patients with LAS showed specific IgE antibodies against some of the 19 allergens tested (1 x birch, mugwort, cat and 1 x grass, alternaria).

While the differences in serum IgE concentrations did not show statistical significance in our groups, the decreased frequency of specific IgE antibodies in LAV/HTLV-III infected patients was significant compared with the healthy noninfected homosexuals using the chi\(^2\) test (p<0.01).

Similarly the incidence of atopic diseases in the personal history was twice as high in group I compared with group III, while none of group II showed evidence for atopic disease.

DISCUSSION
According to our experience and the literature (5, 6, 7) the frequency of elevated serum IgE levels and IgE antibodies against environmental allergens in group I corresponds to the frequency of "latent atopy" in the general population although exact epidemiological data from our country are missing.

It is concluded that IgE production and/or atopic diseases are not, or rather inversely related to LAV/HTLV-III infection. It is too early to speculate about the relevance of these data, since it is not clear whether LAV/HTLV-III infection may suppress IgE production or atopic individuals with high IgE production bear a reduced risk of LAV/HTLV-III-infection. Longitudinal studies in a larger group of patients with long-term follow-up over the different stages of the disease should be performed to further elucidate this question. The fact that elevated serum IgE levels were found in two patients with AIDS might indicate that in the end stage of the disease IgE production may be turned on again. It would be interesting to learn more about the specificity of this increased IgE production; we could not find positive IgE antibodies to the most common environmental aeroallergens or food allergens. Maybe in AIDS the elevated IgE is directed against parasitic antigens not investigated in this study.

For several reasons it seems interesting to study the disturbed isotypic regulation both in atopy (elevated IgE) and in AIDS (impaired IgM response). In atopy decreased function

<table>
<thead>
<tr>
<th>Group</th>
<th>Serum IgE (median) (kU/l)</th>
<th>RAST-positive (%)</th>
<th>Atopic disease (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I (n=39)</td>
<td>570</td>
<td>31</td>
<td>10</td>
</tr>
<tr>
<td>II (n=10)</td>
<td>130</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>III (n=20)</td>
<td>160</td>
<td>10</td>
<td>5</td>
</tr>
</tbody>
</table>
of the T-8 suppressor lymphocyte subpopulation has been described together with other disturbances in cellular immunity (5, 8, 9, 10), while in AIDS the defect of T4 cells is obvious.

Apart from theoretical considerations the question of IgE production, atopic disease and AIDS could gain practical importance with regard to the possible risk of allergic reactions to vaccines or xenogeneic proteins (e.g. monoclonal antibodies) used for therapy.

REFERENCES


Serum Testosterone and Sex Hormone Binding Globulin Levels in Women with Androgenetic Alopecia

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Serum levels of testosterone and sex hormone binding globulin (SHBG) were assessed in eight women suffering from male pattern alopecia. Eight healthy women of similar age served as controls. A statistically non-significant increase was observed in serum testosterone levels as compared with those of the controls (p>0.1). On the other hand, a statistically significant fall was observed in serum SHBG levels as compared with those of the controls (p<0.001). (Received February 19, 1986.)

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Serum testosterone and sex hormone binding globulin levels in women with androgenetic alopecia. Acta Derm Venereol (Stockh) 1986; 66: 532-534.

Though the increased availability of potent androgens at the hair follicles is traditionally regarded as an essential prerequisite for the development of male-pattern alopecia (1), the etiology of this condition and the exact role of androgens in its pathogenesis still remain obscure.