pin surge in the latter woman but, owing to problems in sample collection, I could not demonstrate this unequivocally. Intra-assay CVs (derived from 10 measurements) for FSH were 14.4% at a sample concentration of 3.1 int. units/L and 6.1% at a sample concentration of 9.8 int. units/L. For LH the corresponding figures were 11.5% (at 8.6 int. units/L) and 5.5% (at 33.7 int. units/L).

The results of this preliminary study suggest that this assay kit can reliably be used to measure concentrations of LH in urine. Measurements of FSH, however, are likely to be less reliable (as evinced by the wide variation in recovery of FSH standard from urine), given the low concentration of this hormone in urine.

The kits used in this study were kindly supplied by Dr. T. Appleton, Becton Dickinson U.K., Ltd., Oxford, U.K.

References

Adenosine Deaminase Activity and Acquired Immunodeficiency Syndrome (AIDS), Salvatore Delia, Claudio Maria Mastroianni, Anna Paola Massetti, Giuseppe Turbessi, Augusto Cirelli, Salvatore Catania, and Vincenzo Vullo (Dept. of Infectious Diseases, "La Sapienza" University, Policlinico Umberto I, 00161 Rome, Italy)

Adenosine deaminase (ADA; EC 3.5.4.4), a purine enzyme that specifically catalyzes the deamination of adenosine and other adenine nucleoside analogs to inosine, is found in most human tissues, but its physiological role appears particularly relevant in lymphoid cells.

In the absence of ADA, the increased concentrations of adenosine inhibit the in vitro growth of lymphoid cell lines (1). In addition, ADA activity in lymphocytes and erythrocytes, as well as in serum, is absent in about 20%–30% of children affected by a severe inherited T-cell immunodeficiency (2), whose clinical and laboratory findings are similar to those in patients with AIDS. Caused by a retrovirus, the human immunodeficiency virus (HIV), AIDS is characterized by severe impairment of T-cell functions and cellular immune response.

In view of the possible relationship between ADA activity and the immune response, we measured this enzymatic activity in nonhemolyzed sera from patients with HIV infection. The patients were classified according to a recent classification of the Centers for Disease Control (4): 24 patients from Group II (asymptomatic infection), 13 from Group III (persistent generalized lymphadenopathy), and 20 from Group IV (other HIV diseases, including opportunistic infections and malignancies). We also tested sera from 12 HIV-negative male homosexuals and from 15 healthy heterosexuals not at risk for HIV infection. We used the colorimetric method of Giusti (5).

From our results (Table 1), increased ADA activity seems to be correlated with the detection of anti-HIV antibodies, which express the presence of the virus; not only AIDS patients, but also asymptomatic anti-HIV-positive subjects, had high ADA activity. On the other hand, increased ADA activity in serum does not seem to be due to specific factors, such as opportunistic infections, malignancies, etc. Sera from patients with asymptomatic HIV infection and sera from patients of Group IV, who had no opportunistic infections or malignancies at the moment of sample collection, both had high ADA activity.

The enzyme activity seems therefore to be mainly correlated with the retrovirus infection. HIV encodes for a trans-activating factor, which activates the expression of genes linked to the HIV long-terminal repeat (6, 7). Perhaps this factor can also alter the expression of cellular genes (8), including the promoter region of the ADA gene, thus increasing the activity of ADA.

Table 1. Serum ADA Activity in Different Groups Studied

<table>
<thead>
<tr>
<th></th>
<th>ADA acy (37 °C), U/L</th>
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<tbody>
<tr>
<td></td>
<td>Mean ± SD</td>
</tr>
<tr>
<td>Group II</td>
<td>24</td>
</tr>
<tr>
<td>Group III</td>
<td>13</td>
</tr>
<tr>
<td>Group IV</td>
<td>20</td>
</tr>
<tr>
<td>Homosexuals,</td>
<td></td>
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<tr>
<td>anti-HIV-neg.</td>
<td>12</td>
</tr>
<tr>
<td>Normal controls</td>
<td>15</td>
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</tbody>
</table>

References

Quantification of Glutathione and Glutathione Disulfide in Human Plasma, James D. Adams, Jan N. Johannessen,1 and John P. Bacon1 (College of Pharmacy, Washington State Univ., Pullman, WA 99164; 1 Lab. of Clin. Sci., Natl. Inst. of Mental Health, Bethesda, MD 20892)

Until now, the determination of concentrations of glutathione and glutathione disulfide in human plasma has been

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