ed pH and then after adjustment to pH 7. All samples were centrifuged (1000 × g, 10 min) before analysis.

Albumin concentrations ranged from 1.3 to 41.8 mg/L in 33 samples, the remaining six exceeding the upper limit of the method (80 mg/L). We compared the results of different sample treatments by paired t-test and found a significant decrease only for samples stored at −20 °C without pH adjustment (P < 0.05, n = 33, degrees of freedom = 32). This difference is illustrated by eight samples showing decreases of 23% to 91% from their original values, samples not otherwise distinguished by extremes of pH, osmolarity, or glucose concentration.

We conclude that adjusting the pH of urine to neutral, either before or after deep-freeze storage, protects against precipitation of albumin.

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References

Increased IgA as a Predictor of Development of AIDS in HIV-Infected Subjects, Jean-Jacques Lefrère, Jean-Marius Fine, Patrick Lambin, Denise Salmon, and Charles Salmon (Institut National de Transfusion Sanguine, 6 rue Alexandre-Cabanel, 75015 Paris, France)

Aberrations in immunoglobulin (Ig) concentrations are well recognized in AIDS (1–3). After considering the frequent increase of IgA concentration in AIDS, we initiated a prospective study in asymptomatic HIV-infected subjects in an attempt to see whether an increased IgA concentration could constitute a predictor of evolution towards AIDS.

Eighty seropositive subjects were followed up over a one-year period, being tested at 0, 6, and 12 months. None had AIDS symptoms at the start of the year. IgA concentration, measured by laser nephelometry, was considered increased when >4 g/L. Ten subjects had increased IgA at all three tests; two had increased IgA at the second and third testing but not at the first one; and five had increased IgA at the third testing only. Thus, after one year of follow-up, 25 subjects (31.2%) had an increased IgA. The existence of this anomaly was not correlated with a particular risk factor. The average number of helper (CD4) lymphocytes of subjects with increased IgA during the study period was 410 per microliter at the first testing and 170 per microliter at the third; the average CD4 lymphocytes count of subjects with normal IgA concentration at all three tests was 590 per microliter at the first examination and 420 per microliter one year later, significantly different from the first group of subjects (P <0.01). The subjects with IgA between 4 and 5 g/L at the first testing and >6 g/L at the third testing had a mean of 112 CD4 per microliter at the last testing; the subjects with an IgA concentration between 4 and 5 g/L at the first testing and <6 g/L at the third testing had a mean of 416 CD4 per microliter at the last testing, again significantly different (P <0.01). Eleven of the 80 subjects developed AIDS at the end of the follow-up. All had an increased IgA concentration (mean: 7.11 g/L) at the onset of the disease (third testing), while the HIV antigenemia was positive in 10/11. One year before AIDS (start of the testing), the IgA concentration was increased in five and the HIV antigenemia was positive in six, one or both of these two anomalies being observed in nine patients. The weak correlation between these two anomalies is of interest for the prognosis of the evolution of HIV infection. The correlation is also weak between the increase of IgA concentration and that of other predictors of the disease such as neopterin and β₂-microglobulin (unpublished data). The increase of IgA concentration can be important: in those subjects of our series with an increased IgA, the concentration of IgA was regularly increased and could attain very high values (>8 g/L) when full-blown AIDS developed.

We conclude that an increased IgA concentration in asymptomatic HIV-infected subjects constitutes a predictor of evolution towards AIDS.

References

The “Index of Fiduciality” Proposed for Use in Evaluation and Comparison of Methods, Callum G. Fraser and Margaret C. K. Browning (Dept. Biochem. Med., Ninewells Hospital and Medical School, Dundee DD1 9SY, Scotland)

In spite of the findings (1, 2) that most published evaluations and method comparison studies in both clinical chemistry and hematology are flawed, empirical criteria to judge the acceptability of analytical performance characteristics continue to be widely used. This is surprising in view of the considerable published work (3) on the setting and use of analytical goals.

It may be that the availability of a single number index that would show clearly whether or not the analytical performance of a method was satisfactory would encourage more objectivity in evaluations. A “fiducial” term is one that is used as a fixed basis for comparison. We therefore propose that the index of fiduciality of a method be defined as the performance achieved in practice divided by an objective analytical goal.

The widely accepted view is that the goal for precision is equal to or less than half the average intra-individual biological variation, and that the goal for accuracy (or bias) is that there should be no bias. In reality, however, many methods do exhibit bias. Total analytical error is made up of precision (CVp) and bias (CVb), and can be calculated in a number of ways, including (4) as (CVf + CVp)². It is therefore proposed that the goal for total analytical error should be equal to or less than one-half of the average intra-individual biological variation.