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Rosalki and Foo respond:

To the Editor:

As we reported (1), both liver and biliary alkaline phosphatase (ALP) could be eluted with N-acetylglucoasamine (NAG) from wheat-germ lectin precipitates of plasmas, which by conventional electrophoresis appeared to consist solely of these fractions. However, analytical recovery was low, and, for biliary ALP, was decreased further by sample pretreatment with Triton X-100. Precipitated liver and biliary enzyme in seven samples averaged 17% of total activity, diminishing to 12% after pretreatment.

Precipitated biliary ALP will be interpreted as bone ALP, but significant interference was uncommon. Correction for biliary ALP should bear in mind the fact that conventional cellulose acetate electrophoresis with quantification by reflectance densitometry may substantially overestimate the activity of this fraction, in our experience by an average of 60% (2). Though biliary ALP was present in the plasma of 41 of the 50 liver-disease patients we used for comparison studies with heat inactivation, correlation was good over a wide range of sample activities.

We examined several commercially available wheat-germ lectin batches from Sigma Chemical Co., and a single high-purity batch from Boehringer Mannheim. No significant differences were found. Nor did we observe any difference between plasma and serum specimens.

References

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More on the Dilution Dependence of the Coomassie Brilliant Blue Technique for Urinary Protein

To the Editor:

We read with keen interest the recent report (1) on the dilution dependence of the Coomassie Blue determination of several College of American Pathologists (CAP) urine survey specimens. Although we have reported both decreases and increases in the measured protein concentration upon dilution (2, 3), the shape of our response curve for protein dilution has not been erratic as Goren et al. suggest (1). In the cases where significant dilutional variation could be observed, calculated protein concentrations changed with the extent of dilution in a unidirectional manner (2, 3). The increase in calculated protein concentration observed by us (2, 3) and by others (1, 4) may be explained, at least partly, by disaggregation of the multimeric urinary protein-protein interactions, thus exposing more binding sites for the dye (3). The decrease in measured protein after dilution (2, 3), however, needs further investigation.

References

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A 10-Min Pre-incubation is Required for Measurement of Fructosamine in Plasma

To the Editor:

Measurement of fructosamine in plasma has been suggested as an alternative to measuring glycated hemoglobin for assessment of diabetic control (1). Protein-based standards were recently proposed as the standardization of choice for the automated determination of fructosamine in serum (2), which is essentially as we have suggested (3).

Several conditions proposed for use with various automated analyses involve a pre-incubation time of less than 10 min (2). However, we want to emphasize that the specificity of reduction of nitro blue tetrazolium by the cis-diol form of glycated protein depends on the pre-incubation time being not less than 10 min.

We measured fructosamine in plasma of patients and controls, using a centrifugal analyzer (3) and the Technicon RA 1000 analyzer (2). Pre-incubation times were 10 min and 7 min in the former, 7 min in the latter. Both instruments were standardized with a protein-based standard, calibrated against 1-deoxy-1-morpholinofructose containing human albumin (3). Human-source-based liquid control materials were obtained from Beckman Instruments, Brea, CA 92621.

The results (Table 1) show a statistically significant difference (p <0.01) for the mean concentration of fructoseamine in patients’ plasma samples pre-incubated <10 min. Values for fructosamine in normal and abnormal control materials were not influenced by the pre-incubation time.

Although assay of plasma fructosamine is automatable, these results...