automated ELISA plate reader and related the antigen concentration of the unknown plasma samples to the standard curve. Standard curves were constructed in triplicate on each plate to compensate for any plate-to-plate variation. The unknown plasma samples were run in triplicate at two dilutions, five- and 20-fold.

The lower limit of assay detection for EDP was 10 ng/L. CV for 10 determinations of a plasma sample at an EDP concentration of 0.4 μg/L was 5.2% interbatch, 4.3% intra-batch; for 10 determinations at 20 ng/L, 1.4% interbatch, 1.2% intra-batch.

Measurement of sialic acid therefore may be useful in diagnosis and in assessment of therapy for this disease.

References

Concentrations of Sialic Acid in Serum in Behçet Disease, Taner Onat, Nurşen Eğilmez, and Arif Çimrin (Dept. of Medicine, Ege University, Bornova, Izmir, Turkey)

Sialic acid (N-acetylneuraminic acid) is the main part of the carbohydrates in glycoproteins and glycolipids in cell membranes (1) and may have an important immunological role (2). Recent immunopathological studies of Behçet disease, a multi-system disease, indicate a genetic involvement, but details of its origin are not yet known (3).

Samples were collected from 20 healthy volunteers (14 men, six women, ages 17–49 y) and 27 patients with Behçet disease (19 men, eight women, ages 17–45 y).

Sialic acid concentrations in serum were determined manually with a kit from Boehringer Mannheim. The rate of formation of a colored complex with 4-amino-antipyryne and N-ethyl-N-2-hydroxyethyl-3-toluidine is proportional to the sialic acid concentration in serum.

The mean concentration of sialic acid was 628.7 mg/L (SD 34.8 mg/L) in the control group (range 550 to 739 mg/L), with no correlation with age and sex. The mean value for sialic acid in the acute Behçet group (n = 17) was about 1141 mg/L (SD 54 mg/L, range 904 to 1395 mg/L), again with no correlation with age and sex, but signifi- cantly (P < 0.01) higher than for the remission group (n = 20), 863 mg/L (range 678 to 1082 mg/L).

In 80 healthy subjects, Boehm (4) determined the normal sialic acid concentration to be 635.9 mg/L (SD 96.9 mg/L). The normal value for serum sialic acid concentration was 600 mg/L (SD 88) according to Sugahara et al. (5) for 24 specimens.

Our result for serum sialic acid, about 628 mg/L (SD 35 mg/L) for 20 normal volunteers, was quite similar to those reported by others. We thus conclude that our clearly different results for patients with Behçet disease reflect changes of cell metabolism and antigenity.

Serum Lipoprotein Profile and Concentration of Dehydroepiandrosterone Sulfate (DHEAS) in Glucose-6-Phosphate Dehydrogenase-Deficient Subjects, D. S. Sheriff and M. El Fakhri (Dept. of Biochem., Al Arab Med Univ., Post Box 7025, Benghazi, Libya)

DHEAS, the major circulating adrenal steroid, reportedly exerts a protective effect on possible accumulation of cholesterol in atherosclerosis by acting as a noncompetitive inhibitor of glucose-6-phosphate dehydrogenase (G6PD, EC 1.1.1.49) activity. G6PD, the key enzyme of the hexose monophosphate shunt, helps generate the NADPH required for reductive biosynthesis of steroids, including cholesterol. Connor et al. (1) have postulated that DHEAS by its inhibitory effect on G6PD activity may influence cholesterol metabolism.

In the present study we attempted to determine the concentration of serum total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), and DHEAS in age-matched controls and G6PD-deficient subjects to find out whether the concentration of DHEAS and G6PD activity affected the concentrations of serum lipoproteins. G6PD activity was determined by the WHO-recommended procedure (2) in erythrocytes. Blood samples (collected during 0830 and 1030 hours on three consecutive days) were subjected to the following analyses: DHEAS was estimated by a standard radioimmunoassay (3) (sensitivity, 0.02 mg/L; intra- and interassay CVs, 5.5% and 10%, respectively). Serum TC and triglycerides were estimated by specific enzymic assays (Boehringer Knoll, Mannheim, F.R.G.). HDL-C was measured after precipitation with heparin and manganese.

<table>
<thead>
<tr>
<th>Analyte</th>
<th>Control subjects (n = 50)</th>
<th>G6PD-deficient subjects (n = 10)</th>
</tr>
</thead>
<tbody>
<tr>
<td>G6PD acty, U/mL</td>
<td>0.82 ± 0.10</td>
<td>0.08 ± 0.05</td>
</tr>
<tr>
<td>erythrocytes per minute</td>
<td></td>
<td></td>
</tr>
<tr>
<td>DHEAS, mg/L</td>
<td>2.90 ± 1.45</td>
<td>3.85 ± 0.45*</td>
</tr>
<tr>
<td>TC, mmol/L</td>
<td>4.15 ± 0.15</td>
<td>4.05 ± 0.55</td>
</tr>
<tr>
<td>LDL-C, mmol/L</td>
<td>2.70 ± 0.65</td>
<td>2.50 ± 0.50</td>
</tr>
<tr>
<td>HDL-C, mmol/L</td>
<td>1.25 ± 0.35</td>
<td>1.50 ± 0.45</td>
</tr>
</tbody>
</table>

* Significantly different (P < 0.01) from control subjects.

Table 1. DHEAS Concentration and Serum Lipid Profile in Normal and G6PD-Deficient Subjects
chloride (4). Serum LDL-C was calculated with the formula (4) LDL-C = TC - (HDL-C + triglycerides/5). Statistical significance between the variables studied were calculated by using Student's t-test.

DHEAS concentration was found to be increased in G6PD-deficient subjects, with no significant changes observed in the other analytes studied (Table 1). However, the greater concentration of DHEAS observed in G6PD-deficient subjects may be an independent factor not related to G6PD activity, and G6PD activity may have no bearing on the metabolism of serum lipoproteins.

References