in regard to standardization and interference by lipemia, however, it should not be viewed, however, as a replacement for a standard assay of apoB. Any specimen found to have increased apoB by the EZ-HDL assay should be confirmed with a standard apoB test.

**References**


**Latex-enhanced Immunoturbidimetry Allows D-Dimer Determination in Plasma and Serum Samples, Wolfgang Korte and Walter Riesen (Institute for Clinical Chemistry and Hematology, Kantonsspital, 9007 St. Gallen, Switzerland; *author for correspondence: fax 41-71-494-3900, e-mail Wolfgang.Korte@gd-ikch.sg.ch)**

Quantitative D-dimer determination has become routine practice in patients evaluated for the presence of deep venous thrombosis or pulmonary emboli (1–3). D-Dimer concentrations below a certain cutoff specifically defined for each assay [500 µg/L for ELISA and comparable assays (3)] are considered sufficient evidence to exclude a deep venous thrombosis or pulmonary emboli if the pretest probability is low (4). In addition, D-dimer has been shown to be a reliable indicator of coagulation activation in disseminated intravascular coagulation (5) and malignancy (6). More recently, the relevance of the determination of D-dimer in arterial disease was evaluated (7), and it was shown that D-dimer is a very good predictor of recurrent acute coronary syndromes after a first event (8). There is also some indication that the amount of D-dimer generated correlates to some extent with the degree of atherosclerosis (9).

Fully quantitative D-dimer assays and their automation are recent improvements (10), and short turnaround times allow the routine use of such assays. Routinely, plasma is used for the D-dimer assays. Serum is believed not suitable because of the possibility of continued fibrinolytic activity, which (theoretically) could lead to a (falsely) increased D-dimer concentration. Here, we report that latex-enhanced immunoturbidimetric measurement allows the use of serum as a matrix for the measurement of D-dimer concentrations.

Samples were from patients who had a D-dimer test (from citrated plasma) ordered as well as available serum obtained during the same blood collection. The samples were selected without conscious bias during a 5-week period. Routine blood samples were collected with the Vacutainer® system (Becton Dickinson). For citrated plasma, blood (3.6 mL) was collected into 0.125 mol/L sodium citrate (0.4 mL). For serum, blood was collected into 10-mL tubes containing polystyrene granules. When the samples arrived in the laboratory, platelet-poor plasma was prepared from the citrated samples by centrifugation (1600×g for 10 min at 22 °C); serum was obtained by centrifugation (1500×g for 6 min at 9 °C). D-Dimer concentrations were determined using a latex-enhanced immunoturbidimetric assay (Tinaquant D-dimer on a Hitachi 917 analyzer; Roche Diagnostics). All D-dimer determinations from plasma and serum were performed according to the same routine protocol. D-Dimer concentrations were determined from both materials with three different methods of sample processing: (a) immediately after centrifugation (n = 33); (b) after incubation of the (centrifuged) original tubes at 4 °C for 24 h (i.e., with cell sediment/clot in place; n = 13); and (c) after incubation of the supernatant (with clot and cell sediment removed) for 24 h at 4 °C.

**Table 1. Testing for significant differences between D-dimer concentrations measured in plasma and serum and with three different methods of processing.**

<table>
<thead>
<tr>
<th>Method of Processing</th>
<th>n</th>
<th>Immediate Processing</th>
<th>P for difference</th>
<th>24 h at 4 °C</th>
<th>P for difference</th>
<th>Supernatant, 24 h at 4 °C</th>
<th>P for difference</th>
<th>Immediate Processing</th>
</tr>
</thead>
<tbody>
<tr>
<td>Plasma</td>
<td>13</td>
<td>0.81</td>
<td>0.678</td>
<td>0.64</td>
<td>0.942</td>
<td>0.68</td>
<td>0.701</td>
<td>0.81</td>
</tr>
<tr>
<td>Serum</td>
<td>14</td>
<td>0.753</td>
<td>0.918</td>
<td>0.982</td>
<td>0.926</td>
<td>0.77</td>
<td>0.926</td>
<td>0.77</td>
</tr>
<tr>
<td>r² for plasma vs serum</td>
<td>0.989</td>
<td>0.993</td>
<td>0.974</td>
<td>0.989</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>r² for serum vs supernatant</td>
<td>0.989</td>
<td>0.974</td>
<td>0.989</td>
<td>0.989</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* indicating differences (a) immediately after centrifugation vs processing after incubation of the centrifuged sample in the original tube (with clot and cell sediment present) for 24 h at 4 °C.

* indicating differences between incubation of the centrifuged sample in the original tube (with clot and cell sediment present) for 24 h at 4 °C vs incubation of the original supernatant (with clot and cell sediment removed) for 24 h at 4 °C.

* indicating differences between incubation of the original supernatant (with clot and cell sediment removed) for 24 h at 4 °C vs processing immediately after centrifugation.
D-dimer concentrations is of interest. However, the use of serum samples are available but where measuring D- especially important for clinical studies in which only applicable to a routine setting. These results may be tion with the immunoturbidimetric method used here is phlebotomy, suggesting that serum D-dimer determina-

For all available kits for D-dimer measurements suggest using plasma as the standard matrix. Of specific interest is that all available kits for D-dimer measurements suggest using plasma instead of serum. Thus, enough to allow replacement of plasma by serum. Thus, these results were from samples obtained through routine phlebotomy, suggesting that serum D-dimer determination with the immunoturbidimetric method used here is applicable to a routine setting. These results may be especially important for clinical studies in which only serum samples are available but where measuring D-dimer concentrations is of interest. However, the use of serum for D-dimer determinations needs to be evaluated and validated in a clinical study setting before such results can be used for the exclusion of deep venous thrombosis or pulmonary embolism.

Prof. W. Riesen is a member of an advisory board to Roche Diagnostics.

References