regression data for several sera (undiluted sera on abscissa, diluted sera on originate):

<table>
<thead>
<tr>
<th>Dilution</th>
<th>Dilution with NaCl with BSA</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of sera</td>
<td>16</td>
</tr>
<tr>
<td>Slope</td>
<td>0.778</td>
</tr>
<tr>
<td>Intercept, mmol/L</td>
<td>0.118</td>
</tr>
<tr>
<td>r</td>
<td>0.946</td>
</tr>
</tbody>
</table>

In the case of six other undiluted sera the precipitation was incomplete, and the results after dilution with the albumin solution were 6 to 61% higher than after dilution with NaCl.

If desired, the amount of supernatant fluid to be used for the enzymatic reaction can be increased, to create higher absorbances.

References

J. C. Koedam
H. J. van Dreumel
J. B. A. Terlingen
Lab. of Clin. Chem.
Nat. Instut. of Public Health
and Environ. Hygiene
P.O. Box 1, 3720 BA Bilthoven
The Netherlands

Creatine Kinase Isoenzyme CK-MB Mass/Total CK Activity Ratio In Differentiating Muscle Damage from Myocardial Injury

To the Editor:
El Allaf et al. (1) recently reported the potential use of the creatine kinase (CK; EC 2.7.3.2) isoenzyme MB mass/total CK activity ratio in differentiating patients with multiple trauma and burns but no myocardial involvement from those having suffered acute myocardial infarction (AMI) (1). In the last sentence of their article they conclude "... use of the cutoff value of 80 ng/U will completely differentiate AMI patients from those with trauma or burns, thus allowing detection of myocardial necrosis even in the presence of pre-existing muscular damage." Considering that none of their trauma or burn patients older than one year demonstrated a CK-MB mass/total CK activity ratio >40 ng/U at any time during the first 48 h following theacci-

dent, a ratio ≥80 ng/U would be a very strong indication of myocardial involvement. However, use of a low CK-MB mass/total CK activity ratio alone as a diagnostic parameter, without the CK-MB mass value, cannot rule out myocardial ischemia in burn and trauma patients. Extensive damage to the skeletal muscle with its massive CK reservoir and relatively low CK-MB fraction can very well mask the effect of any myocardial involvement, thus making it impossible to detect myocardial necrosis in the presence of pre-existing muscle damage (2).

References

Shahram Shahangian
Div. of Lab. Med.
The Univ. of Texas System Cancer Center
M.D. Anderson Hosp. and Tumor Inst.
Houston, TX 77030

Two authors of the report in question respond:

To the Editor:
The main goal of our report (1) was to assess the value of the serum CK-MB mass/total CK activity ratio in the differential diagnosis of myocardial and muscular injuries, when the origin of the CK-MB isoenzyme released in the blood is in doubt. Obviously, however, in many cases the absolute CK-MB concentration itself gives definite information on the presence or the absence of a myocardial lesion. According to the reference values recorded for the "Tandem" technique in presumably healthy subjects (2), serum CK-MB concentrations <6 µg/L are normal and rule out myocardial infarction (MI). The serial CK-MB determinations performed in MI patients during our study demonstrated at least one CK-MB value ≥ 86 µg/L between 8 and 32 h after the onset of chest pain. In contrast, all patients with muscular trauma or burns demonstrated CK-MB concentrations below this value (maximum 67 µg/L in the patients older than one year); consequently, whatever the total CK activities in serum may be, all CK-MB concentrations above the cutoff value of 85 µg/L are highly suggestive of MI.

When the CK-MB concentration is between 6 and 85 µg/L, one may consider that the isoenzyme originates either from myocardium or from skeletal muscles. Therefore in those patients with moderately increased CK-MB, myocardial involvement can only be assessed by a CK-MB/total CK ratio ≥ 80 ng/U. Admittedly, small infarctions resulting in low masses of CK-MB or determinations performed more than 32 h after the onset of MI (when the CK-MB values have already decreased) may lead to false-negative results. Only serial measurements performed during the 24-h window surrounding the serum CK-MB peak can reduce the risk of falsely excluding MI in the presence—or even in the absence—of pre-existing muscular damage.

References

Magdelaine El Allaf
Jean-Paul Chapelle
Dept. of Clin. Chem.
University of Liège
1, rue des Bonnes-Villes
B-4020 Liège, Belgium

Screening for Human Antibodies That Interfere In Monoclonal Antibody-Based Assays

To the Editor:
Thompson et al. (1) recently reported a 9% incidence of false-positive results in a two-site immunoassay for creatine kinase (EC 2.7.3.2) MB isoenzyme, ascribed to the presence of antibodies in human sera that cross react with the mouse monoclonal antibodies used in the assay. Furthermore, they suggest that their paper is the first report to describe this interference in an assay in which monoclonal antibodies are used both as the labeled tracer and as the solid-phase antibody.

We, too, have observed this serum interference during the adaptation of a urine-based assay for adenosine deaminase (EC 3.5.4.4) binding protein (ABP) (2) to a serum-based assay (3) for monitoring patients with renal car-

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