To assess interferences of carboxylester hydrolase (EC 3.1.1.1), which can cleave some pancreatic lipase substrates (4), we added 12 200 U of porcine liver carboxylesterase (Sigma Chemical Co., St. Louis, MO; cat. no. E 3128) per liter to the reaction mixture and found no increase in lipase activity.

Thirty samples with absorbance increases, resulting in negative values with the triolein assay, were also determined with the UV kinetic assay. In 28 samples, a value <5 U/L was observed, confirming genuine low lipase activities. In the other two samples, however, UV kinetic results were within the reference limits. Rheumatoid factor can cause interferences in turbidimetric lipase assays because of aggregation of sample components (5), so we investigated 40 patients with active rheumatoid arthritis but without pancreatic disease. Rheumatoid factor determined with a rate-nephelometric latex method (Behringwerke, Marburg, F.R.G.; cat. no. OOUA) yielded values between 64 and 3650 kilo-int. units/L (median: 339 kilo-int. units/L, reference values <5 kilo-int. units/L). With the triolein assay, three patients' values were outside the reference limits (30–190 U/L): one patient had a value of 8 U/L and two patients' values exceeded 190 U/L. With the UV kinetic assay, however, all patients' values were within the reference limits.

Evidently, the Wako UV kinetic lipase assay performs well and is a valuable alternative for the triolein assay, particularly in circumstances when the latter fails owing to nonlinear reaction conditions during the usual measurement interval in automated analysis.

References

Effect of Oral Citrate on Calcium Absorption after an Oral Load of Calcium Phosphate, E. A. de Leacy,1 D. M. Cowley,2 J. M. Brown,2 B. C. McWhinney,2 and A. H. Chalmers3 (Depts. of 1 Pathol. and 2 Med., Mater Misericordiae Public Hospitals, South Brisbane, Queensland 4101, Australia. 3 Current address: Dept. of Haematol., Flinders Med. Center, Bedford Park, South Australia 5042

Orally administered calcium carbonate is poorly absorbed. Citrate given orally at the same time substantially increases calcium absorption and urine calcium excretion, even in small amounts (1 mol of citrate per 8 mol of calcium) (1). Calcium phosphate, the predominant salt of calcium in milk, is also poorly soluble at physiological pH. We investigated whether the effect on the phosphate salt would be similar to that for the carbonate salt in such oral loading experiments.

Oral loading studies were done as described previously (1), on the same three normal subjects. They ate a low-calcium diet the day before both loads and fasted overnight. Water was permitted. Calcium hydrogen phosphate dihydrate (4.3 g) with and without dipotassium hydrogen citrate (6 g) and citric acid (1.2 g) loads were taken orally with 300 mL of water on different days. Urine specimens were collected before this at (−2 to 0) and 0 to 2, 2 to 4, and 4 to 6 h later. We measured urine volume and creatinine, calcium, phosphate, and citrate, all as described previously (1, 2). Data were analyzed by use of the Wilcoxon nonparametric statistic.

The results are tabulated below:

<table>
<thead>
<tr>
<th>Load</th>
<th>0 to 2 h</th>
<th>2 to 4 h</th>
<th>4 to 6 h</th>
</tr>
</thead>
<tbody>
<tr>
<td>Calcium</td>
<td>156 (13)</td>
<td>188 (14)</td>
<td>228 (25)</td>
</tr>
<tr>
<td>+ CaHPO4 · 2H2O</td>
<td>128 (51)</td>
<td>118 (52)</td>
<td>104 (45)</td>
</tr>
<tr>
<td>Citrate</td>
<td>127 (52)</td>
<td>251 (98)</td>
<td>208 (74)</td>
</tr>
<tr>
<td>+ CaHPO4 · 2H2O</td>
<td>1817 (67)</td>
<td>1957 (122)</td>
<td>1951 (263)</td>
</tr>
</tbody>
</table>

* Significant increase from basal, P <0.05. n = 3 each.

Mean (and SEM) urinary creatinine excretions at the respective intervals, with (and without) citrate, were 0.68 (0.03), 0.75 (0.03), 0.69 (0.09), 0.70 (0.08) and 0.75 (0.04), 0.76 (0.05), 0.66 (0.12), 0.71 (0.03) mmol/h, respectively. There were no significant differences between collections.

Urineary calcium excretion did not increase when calcium phosphate was given alone. When an equimolar amount of citrate (25 mmol) was added, urinary calcium excretion increased significantly (P <0.05) above baseline 2–4 and 4–6 h later. There were no significant changes in phosphate excretion. Although citrate excretion doubled when oral citrate was given, the time course of citrate excretion differed from that of calcium excretion, the peak citrate excretion occurring at 0–2 h after ingestion. Citrate excretion also was not significantly changed. Citrate given alone has no effect on calcium excretion (1). These findings are consistent with the suggestion that citrate acts by solubilizing insoluble salts in the gut lumen rather than by mobilizing calcium from bone or promoting its renal excretion (1).

In summary, the effect of equimolar citrate in enhancing calcium absorption and excretion was also seen with calcium phosphate, but was less marked than for calcium carbonate.

References