Evidence that selenium affects CHD risk is equivocal, despite a good biological rationale for a beneficial effect of optimal selenoprotein activity and concentration. However, concern has surfaced recently about possible associations of high selenium exposure with cardio-metabolic risk. A number of sizeable cross-sectional analyses have shown an association between high selenium status in adults and elevated serum or plasma concentrations of total, LDL and non-HDL cholesterol\(^1\)\(^-\)\(^3\), but there is virtually no data from randomized clinical trials on the effect of selenium supplementation on plasma lipids.

The UK PRECISE Pilot study, in which 501 elderly volunteers were randomly assigned to a 6-month treatment with 100, 200 or 300 µg selenium/d as high-selenium yeast or placebo yeast, provided a unique opportunity to investigate the impact of selenium supplementation on plasma lipids. The total and HDL cholesterol were measured on stored plasma samples by routine colorimetric assays on an ILab 650 auto-analyser in 454 participants at baseline and 394 participants at 6 months. Plasma selenium was measured by hydride-generation inductively coupled-plasma mass spectrometry. Data were analysed using mixed models for longitudinal data, adjusted for the treatment centre.

Mean (SD) plasma selenium was 88.7 (19.3) ng/g at baseline, but rose significantly in the 100, 200 and 300 µg/d treatment groups at 6 months (see Table 1). Supplementation with selenium at 100 and 200 µg/d lowered total serum cholesterol and non-HDL cholesterol. The 300 µg/d dose had no significant effect on total or non-HDL cholesterol, but raised HDL cholesterol significantly.

Table 1. Effect of supplementation on mean plasma selenium and mean lipid concentrations after 6 months

<table>
<thead>
<tr>
<th>Selenium dose (µg/d)</th>
<th>Change (SD) in plasma Se (ng/g)</th>
<th>Total cholesterol (mmol/l)</th>
<th>HDL cholesterol (mmol/l)</th>
<th>Non-HDL cholesterol (mmol/l)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Change (95% CI)</td>
<td>P-value</td>
<td>Change (95% CI)</td>
</tr>
<tr>
<td>0 (placebo)</td>
<td>0.05 (18.7)</td>
<td>Reference</td>
<td>Reference</td>
<td>Reference</td>
</tr>
<tr>
<td>100</td>
<td>57.7 (26.0)</td>
<td>-0.23 (-0.42, -0.03)</td>
<td>0.02</td>
<td>-0.02 (-0.08, 0.03)</td>
</tr>
<tr>
<td>200</td>
<td>100.2 (37.6)</td>
<td>-0.25 (-0.44, -0.07)</td>
<td>0.008</td>
<td>0.01 (-0.04, 0.06)</td>
</tr>
<tr>
<td>300</td>
<td>135.0 (50.9)</td>
<td>-0.07 (-0.26, 0.12)</td>
<td>0.46</td>
<td>0.06 (0.00, 0.11)</td>
</tr>
<tr>
<td><em>P</em>-value*</td>
<td>&lt;0.001</td>
<td></td>
<td>0.02</td>
<td></td>
</tr>
</tbody>
</table>

*Overall *P*-value for comparing the three treatment groups v. placebo.

In view of the potential public-health implications of hyperlipidaemia and the widespread use of selenium-containing supplements, the results are reassuring. However, because of the relatively low baseline Se status of the population, the short duration of the intervention and the limited range of the age group involved (60–74 years), additional trials are needed to establish whether these results are more widely applicable. The full range of cardio-metabolic effects of selenium and its potential role in the prevention of CVD need to be further explored.