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Absorption of silicon in healthy subjects

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1. INTRODUCTION
Silicon (Si) is next to oxygen the most abundant element in the earth’s crust. The distribution of Si in vertebral tissue and physiological changes in bone caused by dietary Si deficiency indicate that Si influences bone formation by affecting cartilage composition and ultimately cartilage calcification [1]. The dietary intake of Si was estimated for US citizens to be between 20-50 mg with the lower intake for animal-based diets and the higher intake for plant-based diets [2]. However, studies on the minimum Si requirement and supplementation experiments, comparing the bioavailability of different Si compounds, are lacking. Orthosilicic acid was suggested to have an important function in Si metabolism [1, 6] and is found in both fresh water and sea water. This monomeric form of silicic acid is stable in dilute concentrations of about 10^{-4} M, but condenses into silica gels at higher concentrations and low pH.

The absorption and urinary excretion of Si from (a) stabilized orthosilicic acid (OSA), (b) standardized herbal silica extract from the Si-accumulator plant Equisetum arvense, and (c) colloidal silicic acid were compared in a double-blind study with healthy subjects.

2. MATERIALS AND METHODS
Fourteen healthy subjects (8 females, 6 males, aged 22-34 y) were included after informed, written consent. None had taken Si supplements within 3 months before the start of the study. Each fasting subject received in a cross-over protocol an equimolar dose of Si p.o. (20 mg Si) in the form of stabilized orthosilicic acid (OSA, 1 ml of BioSil containing 20 g Si/l as stabilized monomeric silicic acid, Bio Minerals NV, Belgium [3]), herbal silica (533 mg of a dry Equisetum arvense extract containing 8 % (w/w) SiO_2), colloidal silicic acid (2 ml of 28 g H_2SiO_3/l), or a placebo (10 ml mineral water) with 1 week wash-out period between each supplement or the placebo. Blood samples were collected at Si free polypropylene tubes prior to supplementation and after 1, 2, 4, 6, and 8 hours post partem. Urine was collected between 0 and 24 hours post partem in polypropylene containers. Identical meals were consumed during the experiment after 2
and 6 hours supplementation. The Si concentration in serum and urine was determined for each subject in one batch with AAS [4]. A Zeeman/3030 Atomic Absorption Spectrometer equipped with a HGA-600 graphite furnace was used in combination with an AS-60 autosampler (Perkin-Elmer Corp., Norwalk CT). Pyrolytic coated graphite tubes were used. The hollow cathode lamp settings were respectively 30 mA lamp current, 251.6 nm spectral line and 0.7 nm band width. The injected sample volume was 10 μl and signals were measured in the peak-area mode. The furnace program is shown in Table I. Serum and urine samples were diluted 1/10 in a matrix-modifier solution containing 0.125 g/l La(NO₃)₃·6H₂O; 0.111 g/l CaCl₂; 1.50 g/l NH₄H₂PO₄; 0.50 g/l Na₂EDTA·4H₂O and 0.1% (v/v) HNO₃. Samples were measured against a calibration curve prepared in the matrix-modifier solution, using as standards 0 μg/l, 62.5 μg/l, 125 μg/l and 250 μg/l Si, respectively. The sensitivity determined as the amount of silicon yielding a 0.0044 Abs.s signal was 82 μg. The precision determined as the inter-assay c.v. was 13.3%, 6.2%, and 3.1% for Si concentrations of 240 μg/l (n=6), 627 μg/l (n=6), and 1823 μg/l (n=6), respectively.

The area under the time concentration curve (A.U.C.) was calculated using the linear trapezoidal rule. Statistical significance was investigated with the Wilcoxon matched-pairs signed-rank test. The relation between two parameters was investigated with the Pearson correlation procedure.

3. RESULTS

The mean baseline silicon concentration in serum was lower compared to reported values for serum of normal subjects (Table II).

A significant increase in serum Si concentration (Fig. 1) from the baseline value was observed after respectively 1 h for OSA (mean ± S.E.: 54 ± 19 μg/l, p<0.005), 4 h for the placebo (23 ± 11 μg/l, p<0.025) and herbal silica (37 ± 14 μg/l, p<0.025), and 8 h for colloidal Si (36 ± 12 μg/l, p = 0.01). The mean A.U.C. (Table III) was significantly higher after OSA supplementation (p<0.005), but was not significantly different for respectively herbal silica and colloidal Si compared to the placebo.

A significant correlation was found between the individual A.U.C. and the individual urinary Si concentration (r = 0.28, p<0.05, n = 56). The urinary silicon excretion (Table 2) was significantly higher after OSA supplementation (p<0.005) but was not significantly different for respectively herbal silica and colloidal Si compared to the placebo.

4. DISCUSSION

Most likely, the fasting condition of the subjects in the present study explains the lower Si concentration in serum compared to values reported in the literature. In fact, the serum Si concentration of subjects receiving a placebo increased 2 hours after consumption of a meal, indicating a significant variation of the serum level during the day.
Table I
Temperature programming of furnace for the analysis of Si in serum and urine.

<table>
<thead>
<tr>
<th>Temperature (°C)</th>
<th>Ramp time (s)</th>
<th>Hold time (s)</th>
<th>Gasflow* (ml/min.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>100</td>
<td>15</td>
<td>15</td>
<td>300</td>
</tr>
<tr>
<td>120</td>
<td>10</td>
<td>10</td>
<td>300</td>
</tr>
<tr>
<td>700</td>
<td>5</td>
<td>5</td>
<td>300</td>
</tr>
<tr>
<td>1400</td>
<td>60</td>
<td>10</td>
<td>300</td>
</tr>
<tr>
<td>2500</td>
<td>0</td>
<td>4</td>
<td>0</td>
</tr>
<tr>
<td>2700</td>
<td>1</td>
<td>3</td>
<td>300</td>
</tr>
</tbody>
</table>

*Argon was used as purge gas.

Table II
Silicon concentration in serum of fasting, healthy subjects compared to serum concentrations reported in the literature for normal adults.

<table>
<thead>
<tr>
<th>Author, (year of publication)</th>
<th>Number (female, male)</th>
<th>Method</th>
<th>Si conc. (µg/l)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Berlyne et al. (1986), [6]</td>
<td>23 (2, 21)</td>
<td>AAS</td>
<td>265 ± 17c</td>
</tr>
<tr>
<td>Hosokawa et al. (1990), [7]</td>
<td></td>
<td>AAS</td>
<td>200 ± 60b</td>
</tr>
<tr>
<td>Teuber et al. (1995), [9]</td>
<td>55 (55, 0)</td>
<td>ICP-AES</td>
<td>130 ± 70b</td>
</tr>
<tr>
<td>Calomme et al., (1998), [present study]</td>
<td>14 (8, 6)</td>
<td>AAS</td>
<td>112 ± 42b</td>
</tr>
</tbody>
</table>

*Not specified; bmean ± S.D.; cmean ± S.E.; drange.

Table III
Silicon absorption and excretion from different silicon sources in healthy subjects.

<table>
<thead>
<tr>
<th>Group</th>
<th>Number</th>
<th>Si absorption 0 - 8 hours p.p.</th>
<th>Urinary Si excretion (µg/g creatinine)*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>A.U.C. (µg/l)*</td>
<td></td>
</tr>
<tr>
<td>Placebo</td>
<td>14</td>
<td>202 ± 37</td>
<td>13.3 ± 1.5</td>
</tr>
<tr>
<td>Stabilized orthosilicic acid (OSA)</td>
<td>14</td>
<td>659 ± 95*</td>
<td>17.5 ± 0.9*</td>
</tr>
<tr>
<td>Herbal silica</td>
<td>14</td>
<td>248 ± 53*</td>
<td>12.0 ± 1.1*</td>
</tr>
<tr>
<td>Colloidal silicic acid</td>
<td>14</td>
<td>164 ± 47*</td>
<td>13.8 ± 2.1*</td>
</tr>
</tbody>
</table>

*Mean ± S.E.; b<.002 versus placebo; cnot significantly different versus placebo.
Fig. 1
Increase in serum Si concentration from the baseline value in healthy subjects.
An equimolar dose of Si (20 mg) was given in the form of stabilized orthosilicic acid (OSA), herbal silica, and colloidal silicic acid. Mean values ± S.E. are shown (n = 14)

The present study demonstrates that the bioavailability of silicon is largely dependent on its chemical form. The absorption of Si is faster, higher and less subject dependent for stabilized orthosilicic acid compared to herbal silica and colloidal silicon which are polymerized forms of orthosilicic acid. Consequently, the chemical form of Si in the diet will influence the minimal Si requirement for humans.
The difference in absorption between Si supplements is confirmed by a higher urinary Si excretion for OSA and is in agreement with the dose-dependent Si absorption from stabilized orthosilicic acid which was demonstrated in an animal supplementation study [3]. The significant correlation between the absorption measured by the A.U.C. and the urinary excretion confirms earlier reports suggesting renal clearance as a major route for silicon excretion.
5. REFERENCES