Silicic acid: its gastrointestinal uptake and urinary excretion in man and effects on aluminium excretion

David M. Reffitt *, Ravin Jugdaohsingh, Richard P.H. Thompson, Jonathan J. Powell

Gastrointestinal Laboratory, The Rayne Institute, St Thomas’ Hospital, London SE1 7EH, UK

Abstract

Silicon (Si), as silicic acid, is suggested to be the natural antidote to aluminium (Al) toxicity, and was recently shown to promote the urinary excretion of Al from body stores. The metabolism of Si in man, however, remains poorly investigated. Here we report on the pharmacokinetics and metabolism of Si in healthy volunteers following ingestion of orthosilicic acid (27–55 mg/l Si) in water. We also investigated whether orthosilicic acid promotes the urinary excretion of endogenous Al. Minimum, median uptake of Si from the ingested dose was 50.3% (range: 21.9–74.7%, n = 8) based on urinary analysis following dosing. Significant correlations were observed between creatinine clearance and Si levels in serum or urine (r = 0.95 and 0.99, respectively). Renal clearance of Si was 82–96 ml/min suggesting high renal filterability. These results suggest that orthosilicic acid is readily absorbed from the gastrointestinal tract of man and then readily excreted in urine. There was no significant increase in Al excretion, over 32 h, following ingestion of the orthosilicic acid dose (P = 0.5; n = 5). © 1999 Elsevier Science Inc. All rights reserved.

Keywords: Silicon; Silicic acid; Silicon metabolism; Silica ingestion; Aluminium

1. Introduction

Silicon is the second most abundant element in the Earth’s crust and, after carbon, shows the most diverse chemistry [1]. It is metabolised from lower life forms up to man, and at least for rats and chicks, it is suggested to be essential, since its absence from the diet is associated with growth defects, primarily with bone and cartilage formation [2–4].

In man, Si is present in blood at levels similar to other physiologically important elements (iron, copper and zinc) [5] and is excreted in urine in similar orders of magnitude to calcium. The main route of entry is from the gastrointestinal tract, and the primary source in the diet is in the form of silica (–O–Si–O–), providing 10–50 mg Si/day [6,7]. Plant derived foods (cereals, rice and other types of grains) are the main source of biogenic/phytolythic Si in the diet [7], but since these are largely insoluble forms of Si, they are suggested to be relatively unavailable [6–9]. Absorption would require their breakdown to much smaller soluble species such as orthosilicic acid. Orthosilicic acid (Si(OH)₄) is the major Si species present in drinking water and other fluids, including beer, and is the most readily available source of Si to man [6], although data on its metabolism are limited [6,8–10].

Whether Si has a biological role in man is not known, but it seems likely that such an abundant element in the body has a function, other than just being a universal contaminant. Indeed, as noted above, results from animal studies and in vitro studies with bone cells (osteoblast) suggest that Si may be a requirement of bone and collagen synthesis [4,11,12]. One study in man has demonstrated an increase in bone mass following ingestion of a Si supplement [13]. Mechanisms are unclear, but it has been suggested that Si may be the natural ‘antidote’ to the ubiquitous Al³⁺ ion so that, in the absence of Si, Al is toxic [14]. Indeed, oral Al toxicity is not easily observed in mammals with normal renal function [15], and perhaps this is partly due to the protective effects of Si. In support of this hypothesis, silicic acid reduces Al bioavailability in man [16] and ameliorates toxicity in a number of diverse biological systems [17–19]. Recently, it has been suggested that orthosilicic acid (in beer) increased the urinary output of Al perhaps by interacting with filterable Al in renal tubules, forming hydroxyaluminosilicates, and preventing re-absorption of Al [20]. There is, however, some argument as to whether orthosilicic acid will compete for Al in the presence of endogenous chelators to form hydroxylaluminosilicates [21–28]. Thus, it is unclear whether orthosilicic acid or other components of beer increase the ultrafilterability and renal clearance of Al.
Here, we investigated, in healthy volunteers, the gastrointestinal uptake and elimination of Si from an orthosilicic acid dose solution and its effect on the excretion of Al from body stores. Total Si in serum and urine samples was measured by inductively coupled plasma optical emission spectrometry (ICPOES), as this is well suited to the analysis of elements such as Si which form thermally refractory oxides and are otherwise difficult to measure.

2. Materials and methods

During a wider study [29] to look at the effect of silicic acid on Al absorption, we investigated the gastrointestinal uptake and elimination of Si from a solution of orthosilicic acid in healthy volunteers. Two distinct studies were performed (study 1 and study 2) using nearly identical methods but 55 mg/l orthosilicic acid was ingested in study 1 and 27 mg/l orthosilicic acid in study 2. Data from the two studies have been collated to give information on kinetics of Si absorption and excretion. In study 2, low levels of Al (including radio-labelled Al, $^{26}$Al) were also added as presented elsewhere [29]. Here, the additional low level of Al appeared to have no significant effect on Si pharmacokinetics but total Al in the dosing solutions is indicated in the text for completeness. The effect of the ingested orthosilicic acid dose on the excretion of Al from body stores was also investigated in study 1 and the results are reported here.

2.1. Materials

This work utilised a class J clean air room, within which there was a class C laminar air flow workstation, to reduce contamination with Al and Si during collection and preparation of blood samples. The inductively coupled plasma optical emission spectrometer was also housed in the clean air room, thus enabling contaminant-free analysis. Water was ultrapure, 18 MΩ/cm, from an Elga (High Wycombe, UK) water purifier. Silicic acid was prepared from a stock basic (194.8 g/l NaOH) concentrated (196 g/l Si by ICPOES) sodium silicate solution (Aldrich Chemical Co., Gillingham, UK). ICPOES analysis showed the presence of contaminant Al (1.59 μg/l per mg/l Si) in this stock solution. Polypropylene plastic ware (Merck Ltd, Lutterworth, UK; Aldrich Chemical Co.; and Elkay Laboratory Products Ltd, Basingstoke, UK), acid washed (10% v/v nitric acid) for 24 h, rinsed with ultrapure water and air dried in the clean air room, was used throughout. Syringes (Terumo; Terumo Europe N.V., Leuven, Belgium), used for blood collection, were also acid cleaned as above. Analytical grade nitric acid (AnalaR, 69% v/v; Merck Ltd) was used for acid washing, while ultrahigh purity nitric acid (70% v/v; Spa, Romil; Romil Pure Chemistry, Cambridge, UK) was used for acid dilution of samples for ICPOES analysis.

2.2. Subjects

Healthy volunteers, aged 24–31 years with normal renal function as measured by creatinine clearance, were recruited in both studies from the Gastrointestinal Laboratory (St Thomas’). Throughout the studies subjects ingested a normal diet, except for the avoidance of foods high in Si and Al. Tea (a diuretic and also contains Al chelators), coffee and alcohol (diuretics), and high citric acid (Al chelator) containing foods (e.g. soft drinks) were also avoided as these may markedly interfere with Al uptake and excretion. Mineral water was made available and subjects consumed 2–2.5 l of fluid daily. Subjects fasted overnight from 10 p.m. and remained fasted the next day until 4–5 h after ingestion of the dose solution, and then ate normally, but avoiding the foodstuffs above.

2.3. Preparation and ingestion of orthosilicic acid dose solutions

Orthosilicic acid dose solutions were prepared in ultrapure water at 27 or 55 mg/l Si by dilution of the stock sodium silicate solution and pH neutralisation to 7.2 with HCl. Solutions were then incubated at room temperature for more than 7 days prior to ingestion.

2.3.1. Study 1

The study was conducted in five different volunteers (3 males and 2 females, subjects M1–M3 and F1 and F2, respectively) and each ingested 600 ml water containing 54.9 mg/l Si (87.3 μg/l contaminant Al was also present). Day 1 of the study was used as a run-in period. Two pre-dose urine collections were performed on day 2, each collection period being a total of 8 h (0–8 h and 16–24 h). Following ingestion of the dose solutions (9 a.m.) on day 3, all urine was collected for 32 h. The post-dose urine collections were again made over 8 h periods (i.e. 4 × 8 h collections). Each 8 h urine collection was analysed for its Si and Al contents. Blood samples were also collected in two male volunteers (M1 and M3) who gave consent, from an intravenous all-plastic cannula inserted in a forearm vein. A 5 ml pre-dose sample was collected prior to ingestion and further samples (5 ml) were collected at varying intervals up to 24 h post-dose. Urine samples and aliquots of the dose solutions were analysed for total Si and Al, and the serum samples were analysed for Si, by ICPOES. Orthosilicic acid in the dose solutions was determined at the time of ingestion by molybdc acid assay [30]. Ultrafilterable Si, as a further measure of low molecular weight silicic acid (orthosilicic acid) in the dose solutions, was determined in aliquots using 5000 nominal molecular weight filter units (Biomax Ultrafree-15; Millipore UK Ltd, Watford, UK). The dose solutions were ingested from water-rinsed polystyrene beakers, and following ingestion the empty beakers were rinsed with 5 ml 1% (v/v) HNO$_3$, and the rinses analysed for residual Si adherent to the beakers.
2.3.2. Study 2

The study was conducted in three different male volunteers (subjects M4–M6) and each ingested two different dose solutions 3 weeks apart. Subjects first ingested 621 ml ultrapure water containing 27 mg/l Si (220.6 µg/l Al was also present, contaminant added; see above), then 3 weeks later 621 ml ultrapure water was ingested, again containing 220.6 µg/l total Al. On each occasion, all pre-dose urine was collected for 24 h on day 1 (1 × 24 h collection). Then, after fasting overnight, dose solutions were ingested on day 2 (9:30 a.m.) and all post-dose urine was collected for 72 h (in 3 × 4 h, then 1 × 12 h and 2 × 24 h collections). Blood samples (10 ml) were also collected in all three subjects, as before, from an intravenous all-plastic cannula. Pre-dose and post-dose samples were collected, the latter at varying intervals over 72 h. Urine, serum and an aliquot of the dose solutions were analysed for total Si. Orthosilicic acid and ultrafilterable Si in the dose solutions, at the time of ingestion, were determined as described above.

2.4. Sample preparations

2.4.1. Urine samples

Urine collections were immediately weighed and prepared for ICPOES analysis as before [31,32]. Briefly, the individual urine collections were shaken and a homogenous 50 g aliquot was removed and diluted (1 + 1) with 50 g 0.7% (v/v) HNO₃ in a pre-weighed polypropylene bottle and refrigerated at 4°C until analysis. Prior to analysis the diluted urine samples were warmed to 40°C overnight in an oven, and then allowed to cool to room temperature. This allows re-solubilisation of any precipitated material with no evaporation [31,32].

2.4.2. Blood samples

All blood samples were collected into cleaned polypropylene transport tubes (10 ml; Elkay), without anticoagulant. The capped blood samples were left to clot overnight in the laminar air flow workstation, then centrifuged for 20 min at 400 g (IEC Centra-3; Dunstable, Bedfordshire, UK) at 16°C, and serum fractions (2–5 ml) were collected. Serum samples were kept frozen at −20°C until elemental analysis. Prior to analysis, sera were thawed at room temperature and 1 ml removed into a separate, pre-weighed transport tube and diluted (1 + 5) with 5 ml 0.7% (v/v) HNO₃.

2.5. Elemental analysis

Total elemental analysis for Si and Al was carried out on an inductively coupled plasma optical emission spectrometer (Jobin-Yvon JY24, Instruments SA, Longjumeau France) with a v-groove nebuliser and conventional Scott-type double-pass spray chamber, as before [32], at 251.611 and 396.152 nm, respectively, and with a sample flow rate of 1 ml/min. Peak profiles were used as before [32,33], using a window size of 0.1 nm (0.05 nm either side of the peak) with 54 increments per profile. Integration times were 1 and 3 s per increment for Si and Al, respectively (i.e. analysis time of 1 and 3 min for Si and Al, respectively). Accurate determination of peak height from the ICPOES profile was determined with the program PEAKFIT, as described previously [33]. All samples were analysed in duplicate for each element.

Detection limits were 0.9 and 6.5 µg/l for Al and Si, respectively, in aqueous samples. Urine and serum samples were diluted 1 + 1 and 1 + 5, respectively, so limits of detection were higher for these samples, being ×2 and ×6 of those shown above, for urine and serum, respectively. Using individual sample based standards, accuracy (determined by spiking and recovery experiments) was 99.3 ± 4.1% for Al in urine, 102.0 ± 5.7% for Si in urine and 101.4 ± 3.7% for Al in serum. For Si, precisions in measurements were 107.8 ± 7.9 and 102.7 ± 4.1% in serum and urine, respectively. For Al in urine, precision was 101.8 ± 2.8%.

2.5.1. Serum and urine samples

The diluted serum and urine samples were analysed in batches. One batch consisted of all serum or urine collections from one volunteer. Batches were analysed with individual sample based standards, prepared using a pooled sample of the relevant volunteer’s pre-dose serum samples or urine collections [32]. The diluted serum samples and urine samples in study 2 were analysed for Si alone, using the same methods. Serum Al concentrations were not determined as they would not have been below the limit of analytical detection (in our laboratory baseline serum Al is normally consistently below 2 µg/l).

2.5.2. Dose solutions

Aliquots (10 ml) of the orthosilicic acid dose solutions were acidified to 0.7% (v/v) HNO₃ and analysed sequentially for Si and Al, as were the acid rinses of the polystyrene beakers.

2.5.3. Contamination

Sample blanks were also investigated. Nitric acid, 0.7% (v/v), handled and stored in the appropriate containers and mimicking preparation of urine, serum and dose solutions, was used. These were analysed for Si and Al to determine contamination introduced into the samples.

2.6. Statistical analysis

Data from study 1 was used to assess the effect of orthosilicic acid on excretion of endogenous Al, whereas data from studies 1 and 2 were used to measure the kinetics of Si uptake and excretion. Al excretion (pre- versus post-orthosilicic dose) was assessed non-parametrically (due to low sample numbers) by the Wilcoxon matched-pairs sign-rank test (two-tailed). Correlations were also assessed non-parametrically using Spearman’s rank correlation. A P < 0.05 was
considered to be significant. Data are shown as mean ± SD, unless otherwise stated.

3. Results

3.1. Orthosilicic acid dose solutions

The orthosilicic acid dose solutions were palatable and, although some of the subjects complained of headache, perhaps due to caffeine omission, no adverse effects were associated with their ingestion.

The amount of Si ingested by the subjects in the two studies is shown in Table 1. Negligible amounts of the dose solution remained adherent to the beakers (0.06 ± 0.03% Si) from which the dose solutions were ingested. Similarly, no Al contamination (< 0.9 µg/l) and no significant Si contamination (< 6.5 µg/l) was introduced into the solutions by handling and storage of the blank solutions. Speciation of the Si solution, showed that 98% was orthosilicic acid (Table 1) by molybdic acid [30] and 84–97% ultrafilterable (i.e. < 5000 MW) at the time of ingestion.

3.2. Baseline Si and Al levels

Baseline 24 h urinary excretions of Si and Al are shown in Fig. 1. Baseline serum Si concentrations were between 86.3 and 475.4 µg/l (median value was 146.6 µg/l; n = five subjects), again obtained from pre-dose collections.

3.3. Ingestion of orthosilicic acid dose solutions

3.3.1. Urinary Si and Al

Urinary excretion of Si was markedly increased above baseline following ingestion of the orthosilicic acid dose solu-

Fig. 1. Baseline urinary Si (a) and Al (b) levels in 24 h collections; each point represents the 24 h urinary excretion of a different volunteer. Solid line denotes mean values of all volunteers, being 20.12 ± 6.40 mg and 5.06 ± 1.15 mg, respectively, for Si and Al. Data are from studies 1 and 2 (a) or study 1 (b).

24 h urinary Si (a) and Al (b) levels in 24 h collections; each point represents the 24 h urinary excretion of a different volunteer. Solid line denotes mean values of all volunteers, being 20.12 ± 6.40 mg and 5.06 ± 1.15 mg, respectively, for Si and Al. Data are from studies 1 and 2 (a) or study 1 (b).

Table 1

<table>
<thead>
<tr>
<th>Subject</th>
<th>Dose a (mg)</th>
<th>Serum Si absorption b (%)</th>
<th>Urinary Si excretion c (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Peak (min) AUC (mg/l)</td>
<td></td>
</tr>
<tr>
<td>M1</td>
<td>55.2</td>
<td>98.3 60 1.17 21.8 48.5</td>
<td></td>
</tr>
<tr>
<td>M2</td>
<td>54.7</td>
<td>95.3 80 1.01 16.5 52.1</td>
<td></td>
</tr>
<tr>
<td>M3</td>
<td>55.8</td>
<td>98.7 80 1.01 16.5 44.9</td>
<td></td>
</tr>
<tr>
<td>F1</td>
<td>54.7</td>
<td>98.8 80 1.01 16.5 57.6</td>
<td></td>
</tr>
<tr>
<td>F2</td>
<td>55.7</td>
<td>97.6 80 1.01 16.5 40.2</td>
<td></td>
</tr>
<tr>
<td>M4</td>
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<td>99.3 40 0.46 16.3 54.6</td>
<td></td>
</tr>
<tr>
<td>M5</td>
<td>27.1</td>
<td>99.1 84 0.58 19.5 74.7</td>
<td></td>
</tr>
<tr>
<td>M6</td>
<td>27.5</td>
<td>97.6 60 0.26 6.1 21.9</td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td></td>
<td>98.1 65 16.0 49.3</td>
<td></td>
</tr>
<tr>
<td>(SD)</td>
<td></td>
<td>(1.3) 18 (6.0) 15.2</td>
<td></td>
</tr>
</tbody>
</table>

a Each dose ingested was 600–621 ml. Si T = total Si, measured by ICPOES; Si m = monomeric Si (measured by molybdate) in dose solution as percentage of the total Si.

b AUC was calculated by mass, between 0 and 5 h (subjects M1 and M3) or 0 and 4 h (subjects M4–M6) post-dose. Figures are baseline corrected and shown as a percentage of ingested dose. Peak Si concentration is not baseline corrected.

c Si excreted in urine by 8 h (subjects M1–M3 and F1 and F2) or 4 h (subjects M4–M6) post-dose. Figures are baseline corrected and shown as a percentage of ingested dose.
1). Similarly, in study 2, peak excretion was 50% of ingested Si dose and occurred within the first 4 h of ingestion (Table 1).

A non-significant ($P = 0.5; n = 5$) increase in Al excretion ($1.87 \pm 0.36 \mu g/8 h$ (post-dose) versus $1.69 \pm 0.38 \mu g/8 h$ (baseline, pre-dose); Fig. 2) was observed at the peak excretion of Si in urine, following ingestion of dose solutions in study 1.

### 3.3.2. Serum Si

A typical example of the uptake of Si into serum, following ingestion of the orthosilicic acid dose solutions, is shown in Fig. 3. Ingestion of the orthosilicic acid markedly increased serum Si levels and peak concentration was observed between 60 and 84 min (Fig. 3 and Table 1). Si was rapidly cleared from serum and levels quickly returned to baseline (Fig. 3(a)). A peak in serum Si was also observed following ingestion of the control dose solution in study 2 (ultrapure water alone; Fig. 3(b)), but this occurred at 8 h post-dose and is consistent with an increase in serum Si following the ingestion of lunch at 4 h post-dose. An identical peak was also observed at this time following ingestion of the Si dose in study 2 (Fig. 3(b)), again consistent with lunch at 4 h,

masking the expected rapid return of serum Si levels to baseline as observed in study 1 (Fig. 3(a)). The area under the serum curve (AUC) for Si, following dosing, was determined by mass (‘cutting and weighing’ technique) over the period that subjects were fasted to avoid the influence of dietary Si ($0\sim5\text{ h in study 1 and 0\sim4\text{ h in study 2}}$). AUC, shown as a percentage of the ingested dose, while assuming a plasma volume of 2.46 l [34], has a median value of $16.5\%$ (range: $6.1\sim21.8\%$; $n = five\ subjects$) (Table 1).

### 3.4. Si kinetics

Baseline urinary elimination of Si correlated with baseline serum Si concentration ($r = 0.78, P < 0.01$; Fig. 4). Urinary and serum Si levels also correlated with creatinine clearance ($r = 1.0, P < 0.01$, and $r = 1.0, P < 0.01$, respectively). The renal clearance of Si was $82\sim96\text{ ml/min as calculated using the equation reported in the literature}$ [35].
4. Discussion

Urinary excretion of Si in man is variable, being dependent upon the amount and form of Si in the diet [8,9,29,36]. Here the baseline urinary excretion of Si was 20.1 ± 6.4 mg/24 h (Fig. 1), which is in agreement with literature values of 8.7 ± 4.2 mg [36] to 33.1 ± 3.9 mg [37]. This suggests that dietary Si is fairly well absorbed, since its urinary excretion is a relatively high percentage of the 20–50 mg Si ingested per day in the British diet [6,7]. Indeed, the intake of food alone causes a rise in serum Si levels (Fig. 3(b) and [8]), confirming that at least some Si is readily available from the diet. Interestingly, there was a 4 h delay before peak Si absorption appeared in serum from food, compared to 1 h with orthosilicic acid (Fig. 3), showing that the former is broken down more slowly in the gastrointestinal tract. Others have also noted that orthosilicic acid (Si(\(\text{OH}_2\))\(_4\)), which is a small neutral species under physiological conditions (pK\(_a\) = 9.6), is most readily available for gastrointestinal absorption. Orthosilicic acid is mainly found in the ‘liquid’ portion of the diet and was used here, in chemically defined solutions, for dosing studies in man.

Following dosing, Si was readily eliminated in urine, so 50.3% (median; range: 21.9–74.7%; eight subjects) of the ingested dose was excreted within the first 4–8 h, while serum concentrations quickly returned to baseline (< 24 h). These data are consistent with previous studies where silicic acid was probably ingested in its monomeric form (orthosilicic acid), although this was not confirmed chemically. Thus, Popplewell et al. [10], using low doses of radiolabelled silicic acid reported that 30% of Si was excreted in urine within 8 h of ingestion, while Bellia et al. [20] showed, over the same time period, that 42–75% of Si in beer was also excreted in urine. In the most recent study, Calomme et al. [8] investigated the ingestion of 1 ml of 20 g/l ‘stabilised orthosilicic acid’, and found 8.1 ± 1.2% Si in serum when considering AUC between 0 and 8 h, which compares to our figures of 6.1–21.8% (Table 1). It should be stressed, however, that AUC is not a measure of absorption, but rather represents the ‘circulating balance’ of gut transfer, excretion and tissue uptake. Nonetheless, AUC is a widely used measure as it allows relative comparisons of bioavailability to be made between individuals and between studies, as for example, in the comparison of results from this study with those of Calomme et al. [8].

Baseline 24 h urinary Si excretion and baseline serum Si concentrations correlated significantly (Fig. 4) and both correlated positively with creatinine clearance as previously suggested [38,39]. Similarly, Si levels in serum following dosing also correlated with Si levels in urine (data not shown). These correlations and the high renal clearance of Si (see below) strongly suggest that the kidney is the major route of Si excretion. Renal clearance of Si was 82–96 ml/min, again confirming the reported value of 88.6 ± 7.9 ml/min [37]. This relatively high renal clearance, compared for example to Al (4–7 ml/min [35]), suggests that the majority of Si in serum is filterable by the kidney and that there is a low renal re-absorption of Si in the nephron [20]. Taken together, these data suggest that Si from orthosilicic acid is readily absorbed in man and readily excreted in urine, so there appears to be no specific mechanism to retain Si in man [40].

Finally, we investigated the possibility that orthosilicic acid may mobilise Al from body stores into urine [20]. The mean baseline Al excretion was 5.06 ± 1.15 µg/24 h (Fig. 1(b)), in keeping with other reported results in man [32,41]. Following ingestion of 55 mg/l orthosilicic acid, an insignificant (\(P = 0.5\)), but similar increase in the excretion of Al was observed with the peak urinary excretion of Si (0–8 h post-dose). This could easily be accounted for by absorption of a fraction (0.73 ± 0.82%) of the contaminant Al ingested (51 µg) in the orthosilicic acid solutions or by the increased urinary output following ingestion of a large volume (600 ml) of fluid. Thus, we found no evidence for the mobilisation of Al from body stores following ingestion of orthosilicic acid. In contrast, Bellia et al. [20] observed a significant concomitant increase in urinary elimination of Al (or an unlikely absorption of 26% of the ingested Al (72.1 µg)) following the intake of beer which contained silicic acid. They suggested an interaction, in the proximal convoluted tubule of the kidney, between Si and Al leading to their co-elimination. It is not clear why our results differ, but a number of explanations should be considered. First, a small peak of Al may have occurred in urine following ingestion of orthosilicic acid that would only be apparent in urine collections of much shorter time intervals (e.g. in catheterised subjects). However, Bellia et al. [20] also used 8 h collection periods for urine and were able to observe a difference. Secondly, alcohol may increase gut permeability as it is known to affect lipid bilayer permeability [42], which would then increase Al uptake from beer. However, a 26% increase seems unlikely. Thirdly, Si and alcohol may have a synergistic effect on the excretion of Al, which is not observed with alcohol.
[20] or Si alone (Fig. 2). Fourthly, a component of beer other than alcohol or silicic acid may be responsible for the excretion of Al from body stores. Finally, the body pool of Al of the subjects in the Bellia study [20] may have been higher, as volunteers were older than the subjects in this study.

In conclusion, orthosilicic acid [Si(OH)₄] was readily absorbed in the gastrointestinal tract of man and eliminated by the kidney into urine. At least 49% Si was taken up and, although it is not possible to estimate how much was retained without radio-isotopic studies, this was probably less than 10% of the absorbed dose [10]. There was no evidence to suggest that orthosilicic acid facilitates the excretion of Al from body stores as there was no difference in the urinary output of Al following ingestion of orthosilicic acid.

5. Abbreviations

ICPOES inductively coupled plasma optical emission spectrometry
AUC area under the curve

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