

Selenium balance studies in apparently healthy and housebound elderly people eating self-selected diets

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1. Metabolic balance studies (5 d) for Se were conducted in twenty-four apparently healthy elderly people (age 69.6–85.4 years), and twenty housebound subjects (age 69.9–85.1 years) with chronic disease. During the study the subjects lived in their own homes, ate self-selected diets and continued their normal daily activities.

2. Geometric mean daily dietary intakes of the two groups were significantly different ($P < 0.01$), being 819 (range 310–1631) nmol for the healthy and 475 (range 233–1136) nmol for the housebound elderly.

3. Daily intake of Se significantly correlated with balance in both groups. Solution of the regression equations gave theoretical daily requirements of 447 nmol for the healthy and 419 nmol for the housebound subjects. The healthy elderly were in positive balance of 148 nmol/d for Se and the housebound were in equilibrium with an overall mean retention of 43 nmol/d.

4. Mean levels of Se in blood fractions for the healthy and housebound subjects were significantly different, being 1.65 and 1.40 $\mu\text{mol/l}$ whole blood, 1.45 and 1.21 $\mu\text{mol/l}$ plasma and 5.72 and 5.30 nmol/g haemoglobin in erythrocytes respectively. There was no difference in the whole-blood glutathione peroxidase (EC 1.11.1.9) activities between the two groups.

5. There were clear differences in Se status between the two groups of elderly people. The cause of the positive balance in the healthy subjects remains unexplained.

There is an increasing interest in the possible relation between selenium metabolism and the process of ageing (Csallany *et al.* 1981). Free radical formation and lipid peroxidation have been proposed as factors which cause membrane damage and accelerate the ageing process (Harman, 1956). Se as a component of glutathione peroxidase (EC 1.11.1.9; GSH-Px) has antioxidant activity and assists in the prevention of the accumulation of lipid peroxides and free radicals (Flohe *et al.* 1979). There have, however, been relatively few investigations into the Se status of the elderly.

The Se content of some animal tissues is reported to increase with age (Persigehl *et al.* 1977; Burch *et al.* 1979). In contrast human erythrocyte Se concentration (Thomson *et al.* 1977; Miller *et al.* 1983) and GSH-Px activity (Thomson *et al.* 1977) appear to fall; such changes may be due to the process of ageing itself, or may reflect an inadequate dietary intake or altered absorption, excretion or distribution of Se in the elderly. There is no firm recommended dietary allowance (RDA) for Se. The existing estimated safe and adequate daily dietary intake (ESADI) of 630–2530 nmol/d was derived mainly from animal studies (Food and Nutrition Board, 1980). In common with most RDA and ESADI it takes no special account of any effect that ageing or disease may have. Widely differing daily dietary Se intakes have been reported for the elderly, ranging from 2380 nmol in Canada (Gibson *et al.* 1983) to 300 nmol in Sweden (Abdulla *et al.* 1979). As the geographical distribution of soil Se content varies considerably (Burk, 1976), interpretation of such reports is difficult.

In order to learn more about the daily intake, retention and excretion of Se in old age

we have carried out metabolic balance studies on healthy, free-living elderly people eating self-selected diets. In addition, to assess the influence of the commoner chronic diseases of old age on these factors, we have investigated a group of housebound subjects.

METHODS

Subjects

The studies were carried out between August 1980 and September 1984. Exact details of the anthropometric characteristics of the subjects have been published elsewhere (Bunker *et al.* 1987*a*).

Healthy group. Eleven men (mean age 78.2, range 70.1–85.2 years) and thirteen women (mean age 75.8, range 69.6–85.4 years) were included. They were free from any apparent disease, although one woman was subsequently shown to have mild hyperglycaemia. They lived unsupported in their own homes, and purchased and prepared their own food.

Housebound group. Seven men (mean age 78.8, range 69.9–85.1 years) and thirteen women (mean age 78.8, range 70.5–82.9 years) took part. Subjects were classified as housebound if they were unable to go out unaccompanied. This was generally due to physical infirmity, although two subjects were suffering from severe depression. The participants were suffering from various stable chronic diseases such as arthritis, chronic obstructive pulmonary disease, etc. Individuals known to have hepatic, renal or gastrointestinal disease, malignancies or acute illness were not included. The subjects were receiving various medications including tranquilizers, diuretics and anti-inflammatory drugs as described previously (Bunker *et al.* 1987*b*). Food was bought by friends, relatives, home help or the subjects themselves when they were taken out. Three men and four women received meals-on-wheels 5 d/week.

The study was approved by the Joint Ethical Sub-Committee of the Faculty of Medicine of the University of Southampton and Southampton and South West Hampshire District Health Authority.

Metabolic balance studies (5 d) were carried out as previously described (Bunker *et al.* 1984*a*). This involved the collection of duplicate diets, faeces and urine into plastic containers taking precautions to avoid contamination of the samples. Where a subject received meals-on-wheels two portions were delivered; these were combined and divided, any of the food not eaten being saved separately. It was necessary to spend much time with the subjects, particularly those who were housebound, to ensure reliable collection of samples, but great care was taken not to encourage the subjects to alter their normal eating habits.

Blood samples were obtained from all the healthy and eighteen of the housebound individuals.

Analyses

Balance material. Samples were analysed by a modified hydride-generation atomic absorption spectrophotometric (HG-AAS) technique used for blood and plasma (Lloyd *et al.* 1982). Portions of diet (5 g), faecal (5 g) and urine (7.5 g) homogenate were weighed into glass cylinders. Approximately 15 ml 18 M-sulphuric acid–16 M-nitric acid (1:1, v/v) solution were added and the samples left to stand for 16 h. The volume was made up to 25 ml with the acid solution and mixed well. Triplicate 1 ml portions were transferred into glass tubes, a 2 ml portion of the acid mixture added and the tubes heated at 150–155° for 2.5 h. After cooling, 2 ml 6 M-hydrochloric acid was added to the tubes and they were reheated at 95° for 30 min. The contents were transferred into hydride-generation reaction vessels, diluted to 20 ml and 200 μ l antifoam reagent added (Antifoam emulsion DB 110A;

Table 1. Analysis of standard reference materials following digestion with nitric and sulphuric acids

Sample	No. of analyses	Value obtained (nmol/g dry wt)		Certified value* (nmol/g dry wt)	
		Mean	1 SD	Mean	Estimated uncertainty
Bovine liver 1577a†	10	9.13	0.22	8.99	0.89
Wheat flour 1567†	2	12.66	—	13.93	2.53
Rice flour 1568†	2	4.56	—	5.07	1.27
NEA horse kidney H-8‡	2	61.6	—	59.1	3.8

* Certified value converted to SI units.

† Obtained from National Bureau of Standards, Washington, DC.

‡ Obtained from International Atomic Energy Agency, Vienna.

Dow Corning, Glamorgan; 10 ml/l). Standards (0–2.53 $\mu\text{mol/l}$) were prepared by taking 100 μl volumes, adding 3 ml of the acid mixture and proceeding as for the test samples.

Hydrogen selenide vapour was produced by adding 6 ml sodium tetrahydroborate (III) (0.16 M (60 g/l) in 0.025 M-sodium hydroxide solution) to the reaction vessel. Se concentration was measured using a Perkin Elmer atomic absorption spectrophotometer (model 2380) with an electrode-less discharge lamp and a mercury-hydride system (MHS 20). The following instrumental conditions were used: tube temperature 950°, purge I (argon) 38 s, reaction time 10 s, purge II (argon) 25 s. Integrated peak area was measured with a read time of 19 s.

It has been suggested (Janghorbani *et al.* 1982; Neve *et al.* 1982) that the inclusion of perchloric acid in the digestion procedure is essential for the complete decomposition of the organic matrix and conversion of organoselenium to Se IV. We have found (Bunker & Delves, 1987) no difference in the digestion efficiencies of a nitric–sulphuric acid mixture and a nitric–sulphuric–perchloric acid mixture for dietary faecal and urinary samples subsequent to analysis by HG-AAS. The attendant potential hazards associated with the use of perchloric acid and the need for special venting facilities makes the avoidance of this acid desirable.

Four different standard reference materials were analysed by the method described and the results are given in Table 1. Mean within-batch precision of 3.0, 3.1 and 2.5% and mean recoveries of Se added to the acid chemical homogenate of 98, 103 and 102% were obtained for the analysis of ten samples of urinary, dietary and faecal homogenates respectively.

The dietary samples were also analysed for nitrogen (semi-automated Kjeldahl; Tecator, Bristol) and gross energy (bomb calorimeter; Gallenkamp, London). The protein content was calculated assuming a 160 g N/kg protein concentration and metabolizable energy was calculated using the formula of Miller & Payne (1959). These results have been reported previously (Bunker *et al.* 1987a).

Blood samples

Haematological analyses were carried out using a Coulter-counter model S+4. Whole blood and plasma samples were analysed for Se by the method of Lloyd *et al.* (1982). Erythrocyte Se concentration was calculated from the difference between the concentration in whole blood and plasma, taking the packed cell volume into account. GSH-Px activity was measured in whole blood by the method of Beutler (1979), using *t*-butyl hydroperoxide as a substrate.

Table 2. *Daily dietary selenium intake in elderly healthy and housebound men and women*
(Geometric means, with arithmetic means in parentheses, and 95% confidence interval (95% CI))

	<i>n</i>	Se intake					
		nmol/d		nmol/kg body-wt per d		nmol/10 MJ	
		Mean	95% CI	Mean	95% CI	Mean	95% CI
Healthy:							
Men	11	879 (895)	805-959	12.6 (12.9)	11.5-13.9	973 (984)	909-1041
Women	13	773 (859)	626-953	12.2 (13.7)	9.9-15.0	1125 (1223)	939-1349
All	24	819 (876)	695-966	12.4 (13.4)	10.5-14.6	1053 (1114)	914-1213
Housebound:							
Men	7	484 (512)	411-570	7.2 (7.5)	6.4-8.2	760 (795)	651-889
Women	13	470 (513)	385-575	7.7 (8.3)	6.3-9.4	951 (1045)	772-1172
All	20	475 (513)	395-571	7.5 (8.0)	6.3-8.9	879 (958)	724-1068
Probability values for the independent effect of sex and health:							
Sex	—	NS	—	NS	—	NS	—
Health	—	<i>P</i> < 0.001	—	<i>P</i> < 0.001	—	<i>P</i> < 0.01	—

NS, not significant.

Statistical analysis

Each group of results was examined to assess their closeness to a normal distribution. All values relating to blood analyses approximated such a distribution; those for Se intake, urinary, faecal and total excretion were positively skewed, and were therefore log-arithmically transformed to achieve approximate normality. The log-transformed mean and standard deviation were used to calculate the geometric mean and 95% confidence interval (95% CI). To facilitate comparison with other published work, the arithmetic mean values, where appropriate, were calculated. Multiple regression analysis was used to assess the effects of health, sex and body-weight on the results. Association between variables was determined using Pearson's correlation coefficient, a paired *t* test was used to assess the significance of the retention values obtained and an unpaired *t* test used to compare the Se concentration in the blood fractions and the whole blood GSH-Px activity between the two groups of elderly (Armitage, 1971).

RESULTS

Table 2 gives the results of daily Se intake for both groups of men and women. The daily dietary Se intake correlated with that of protein (healthy *r* 0.62, *P* < 0.01; housebound *r* 0.41, *P* < 0.05). In the healthy group, only two women and one man had intakes below the lower level of the ESADI of 630 nmol, while eleven women and five men in the housebound group consumed less than this amount. The meals-on-wheels provided a mean level of 166 (95% CI 136-196) nmol, of which 139 (95% CI 94-184) nmol were consumed. This was equivalent to 38% of the total daily Se intake of the seven recipients.

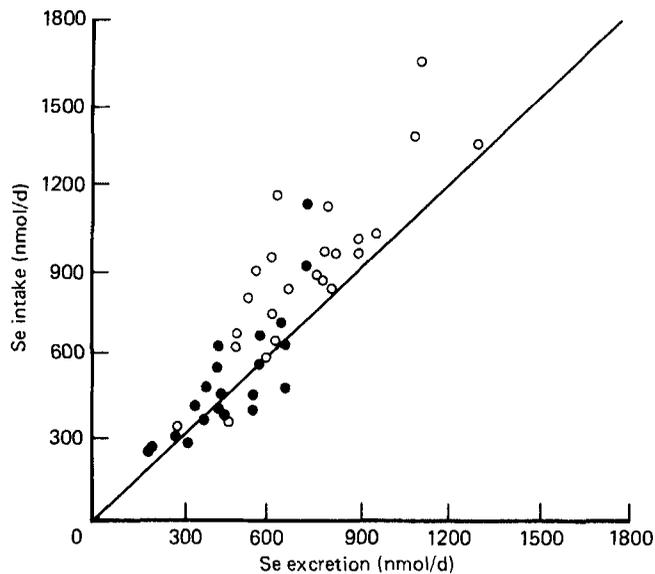
The results of the balance studies are given in Table 3 and Fig. 1. The healthy were in positive balance of 148 nmol, a value which significantly differed from zero (*t* 4.52). The housebound were in equilibrium (*t* 1.41) for Se, with a mean retention of 43 nmol. There was no difference in the intake, excretion or retention of Se for men or women in either group. Se retention was significantly correlated with dietary intake (healthy *r* 0.66, *P* < 0.001; housebound *r* 0.75, *P* < 0.001) as shown in Fig. 2. Balance was not affected by

Table 3. Daily intake, excretion and retention of selenium (nmol/d) in healthy and housebound men and women

(Mean values and 95% confidence interval (95% CI))

	n	Intake		Total excretion		Urinary excretion	
		Mean	95% CI	Mean	95% CI	Mean	95% CI
Healthy:							
Men	11	878	805-959	721	661-785	368	327-415
Women	13	773	626-953	667	559-796	334	286-389
All	24	819	695-966	691	600-796	349	305-401
Housebound:							
Men	11	484	411-570	505	450-566	244	207-288
Women	13	470	385-575	417	349-498	200	165-243
All	20	475	395-571	446	379-524	214	178-258

	n	Faecal excretion		Apparent absorption		Net retention	
		Mean	95% CI	Mean	95% CI	Mean	95% CI
Healthy:							
Men	11	334	288-388	543	473-613	161	93-229
Women	13	324	258-407	492	386-598	137	67-207
All	24	329	271-398	515	426-605	148	80-216
Housebound:							
Men	11	239	196-292	251	186-316	-5	-65-54
Women	13	211	174-257	285	202-368	69	4-134
All	20	221	182-268	273	197-349	43	-21-107

Fig. 1. Daily selenium balance in healthy (○) and housebound (●) elderly. The line of equality ($y = x$) is shown.

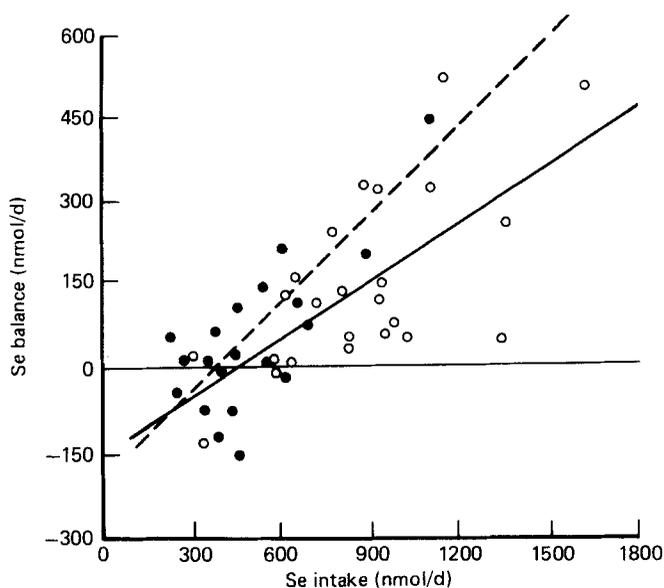


Fig. 2. Relation between selenium intake and Se balance in healthy (○) and housebound (●) elderly people. (—), (---), Regression lines for healthy and housebound elderly respectively.

Table 4. Selenium concentration in blood and plasma and whole-blood glutathione peroxidase (EC 1.11.1.9) activity in healthy and housebound elderly and younger individuals

(Arithmetic mean and 95% CI)

	Healthy elderly (n 24)		Housebound elderly (n 18)		Younger adults† (n 338)	
	Mean	95% CI	Mean	95% CI	Mean	95% CI
Whole-blood Se (μmol/l)	1.65	1.57–1.73	1.40***	1.28–1.52	1.75	1.72–1.78
Plasma Se (μmol/l)	1.45	1.38–1.53	1.21**	1.08–1.35	1.47	1.45–1.49
Erythrocyte Se (nmol/g Hb)	5.72	5.54–5.91	5.30*	4.99–5.61	6.25	6.13–6.37
Whole-blood glutathione peroxidase (U/g Hb)	21	19–23	19	17–22	19.6	19.1–20.1

Hb, haemoglobin.

Mean values were significantly different from those of the healthy elderly: * $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$.

† Published values of Lloyd *et al.* (1983) converted to SI units.

body-weight. Analysis of covariance showed that a single straight line could be used to describe the values for men and women as there was no significant difference between the slopes. Statistical analysis also showed that the slopes obtained for the healthy and housebound groups were similar. The following equations were derived to describe the association between Se intake (nmol/d) and balance (nmol/d) for healthy and housebound elderly respectively: Se balance = 0.34 intake – 152 and Se balance = 0.47 intake – 197. Expressed in terms of body-weight (nmol/kg per d) the following equations were obtained for the healthy and housebound subjects respectively: Se balance = 0.39 intake – 2.76 and Se balance = 0.53 intake – 3.55. Se retention did not correlate with any other measurements of Se status.

Table 4 gives the results of the analyses of Se concentration in whole blood, erythrocytes and plasma and GSH-Px activity in blood. There was no difference in any of the values obtained between men and women and therefore the values have been combined. There was no correlation between GSH-Px activity and any measurement of Se status.

DISCUSSION

The mean (geometric) daily Se intake of 819 nmol in the healthy elderly exceeded the lower level of the ESADI of 630–2530 nmol/d (Food and Nutrition Board, 1980) and was similar to the average estimated intake for this country of 760 nmol (Thorn *et al.* 1978). The value was lower than the mean values of 1380 and 1190 nmol/d calculated for elderly people eating self-selected diets in Canada (Gibson *et al.* 1983) and America (Lane *et al.* 1983) respectively, but is similar to the median intake of 881 nmol determined by analysis for the diets of elderly Canadian women (Gibson *et al.* 1985). The dietary Se intake of our healthy subjects was considerably higher than the median value of 304 nmol/d found by duplicate diet analysis for a group of Swedish pensioners (Abdulla *et al.* 1979). It must be borne in mind, however, when making such comparisons, that we have reported geometric mean intakes whilst other authors have given arithmetic mean or median values. Additionally, as a result of the geographical distribution of soil Se levels, the dietary intake of this element varies greatly in different parts of the world (Burk, 1976).

The mean Se intake of the housebound subjects of 475 nmol/d was only 58% of that found in the healthy people. Differences in energy consumption do not entirely account for this finding. The housebound subjects consumed a significantly-less Se-dense diet than the healthy elderly (879 v. 1053 nmol/10 MJ; $P < 0.01$). This observation contrasts with the fact that the densities of the diets eaten by both groups of elderly were comparable with respect to several nutrients, including protein (Bunker *et al.* 1987*a*), zinc, copper (Bunker *et al.* 1984*a*, 1987*b*), calcium, phosphorus and magnesium (Bunker *et al.* 1988). In assessing the dietary intake of Se it is essential to consider the contribution that different food groups make to the total intake. Thorn *et al.* (1978), in a study of diet samples collected in 1974, reported that half the Se in the average British diet was derived from cereals and cereal products, with meat and fish providing another 40%. Since this report there has been a reduction in the import of wheat from Canada and the USA (Ministry of Agriculture, Fisheries and Food (MAFF), personal communication). The average Se concentration of wheat obtained from North America is at least ten times greater than that of home-produced wheat (Thorn *et al.* 1978). Consequently the proportion of American wheat included in milling has a profound influence on the Se content of flour. The duplicate-diet collections for the healthy subjects were carried out between August 1980 and July 1982, while those for the housebound elderly were obtained between July 1983 and September 1984. During this time the contribution of imported wheat from North America to the total amount of wheat milled in this country decreased from approximately 35% in 1980 to approximately 17% in 1984 (MAFF, personal communication). This fact may go some way to explain the differences in the Se densities of the diets eaten by the two groups of elderly.

The healthy elderly were in positive balance of 148 nmol for Se. This high apparent positive retention is difficult to explain. It is unlikely that inaccuracies have occurred in the measurement of intake or excretion. The precision and accuracy of the analytical method have been validated, and good agreement was obtained between the analysed and certified values for a range of different organic quality control materials. Analysis of the same samples has shown these subjects to be in equilibrium for N, Zn and Cu, iron and chromium (Bunker *et al.* 1984*a,b,c*, 1987*a*). The balance values do not, however, take

account of losses via perspiration or expired air. Studies from New Zealand using ^{75}Se -labelled selenite (Thomson & Stewart, 1974) and selenomethionine (Griffiths *et al.* 1976) found dermal and respiratory losses to be negligible. Actual measurements of sweat (Levander *et al.* 1981) and skin (Molin & Wester, 1976) Se content suggest that daily losses from each are less than 13 nmol.

Some workers (Janghorbani *et al.* 1982; Neve *et al.* 1982) believe that the trimethylselenium ion, which may be a major excretory form of Se in urine, is extremely difficult to digest without the use of perchloric acid. Other authors (Reamer & Veillon, 1981, 1983*a,b*) do not agree with this suggestion and we (Bunker & Delves, 1987) have found no difference in urinary Se concentration determined with and without the use of perchloric acid. The method described here has also been used to analyse urine in an International Union of Pure and Applied Chemistry (IUPAC) inter-laboratory study. The mean value obtained was within the range of reported observations from other laboratories and agreed particularly well with those results derived using neutron-activation analysis with radiochemical separation and isotope-dilution mass spectrometry following acid digestion.

To date there is only one report in the literature of balance studies for Se being carried out in the elderly (Stead *et al.* 1985). Unfortunately details relating to study design and analytical techniques were minimal. Five of the six men studied did not live independently in the community, and subjects were admitted to the metabolic unit only 24 h before the commencement of the study. The subjects consumed a mean of 760 nmol Se/d and were in positive balance to the extent of 282 nmol. This value represented a retention of 37% of the dietary intake compared with 14% by the subjects in the present study. Obviously the six men studied were not in equilibrium with their hospital diets and the validity of the findings is questionable. Levander & Morris (1984) have carried out balance studies on young North American adults eating self-selected diets providing an average of 1025 nmol Se/d. The subjects were in positive balance of 129 nmol/d, which represented 13% of their daily intake. Two other studies carried out on subjects eating self-selected diets are also of interest. Four New Zealand women have been shown (Stewart *et al.* 1978) to be in equilibrium for Se with an average daily intake of 306 nmol, while ten Chinese men were found to be in positive balance to the extent of 23 nmol, with a mean daily intake of only 114 nmol (Luo *et al.* 1985). It is important to note that perchloric acid digestion was used in the sample preparation for the three studies (Stewart *et al.* 1978; Levander & Morris, 1984; Luo *et al.* 1985) on younger people. Yet in two of them (Levander & Morris, 1984; Luo *et al.* 1985) the subjects were in overall positive balance. The reason for these results and the positive balance of the subjects in the present study remains unexplained.

The housebound subjects were in equilibrium for Se with a mean retention of 43 nmol. This finding contrasts with the overall negative N, Zn and Cu (Bunker *et al.* 1987*a,b*) balances reported by us for this group. The housebound appear to be in balance for Se but their status is quite different from that of the healthy elderly.

Absorption of Se was similar in the healthy and housebound subjects (mean 0.57 (95% CI 0.53–0.62) *v.* 0.50 (95% CI 0.43–0.57)). These values are comparable to those of 0.64, 0.55 and 0.57 found for younger subjects eating self-selected diets in North America (Levander & Morris, 1984), New Zealand (Stewart *et al.* 1978) and China (Luo *et al.* 1985) respectively. Urinary Se excretion accounted for 50 and 48% of the total excretion in the healthy and housebound subjects respectively, a percentage similar to that found in the aforementioned studies.

There was a highly significant correlation between Se intake and retention in both groups of elderly people. Solution of the appropriate regression equations provided theoretical daily requirements of 447 and 419 nmol Se to maintain balance in healthy and housebound subjects respectively. These values are less than the values of 1013 and 722 nmol reported

by Levander & Morris (1984) for young men and women, but greater than the level of 304 nmol found by Stewart *et al.* (1978) for young women in New Zealand. This reinforces the suggestion of Levander & Morris (1984) that Se requirement is a function of previous dietary intake. The same authors, in explaining an observed difference between the requirement for men and women, speculated that requirement is also dependent on lean body mass (LBM). We found no difference between the needs of men and women. Men lose LBM more rapidly with age than women, and therefore it is possible that the differences between the sexes are less marked in the elderly. Solution of the weight-adjusted regression equations gave values for theoretical daily requirement of 7.08 and 6.70 nmol/kg per d for the healthy and housebound people respectively.

The level of Se found in the whole blood and erythrocytes of the healthy elderly was slightly lower than the values reported by this laboratory (Lloyd *et al.* 1983) for younger adults. Since the studies of the healthy elderly and younger subjects were carried out simultaneously, a comparison of the two groups is valid. There was no difference between the two groups in the plasma Se concentration or the whole-blood GSH-Px activity. A fall in erythrocyte Se concentration with age has been reported previously by some authors (Thomson *et al.* 1977; Miller *et al.* 1983) while others found that plasma but not erythrocyte levels fell with age (Verlinden *et al.* 1983). Thomson *et al.* (1977) also reported a concomitant fall in plasma Se levels and also erythrocyte GSH-Px activity. Certain human and animal tissues are reported to accumulate Se with progressive age (Persigehl *et al.* 1977; Burch *et al.* 1979), and an increase in liver GSH-Px activity per unit protein with age has also been shown in normal human liver (Corrocher *et al.* 1980). It is possible that a redistribution of Se from the erythrocyte to other tissues may occur with ageing.

The housebound elderly had significantly lower whole blood, erythrocyte and plasma Se concentrations than did the healthy subjects. This can probably be attributed to the lower Se consumption in this group. There was, however, no significant difference in whole blood GSH-Px activities between the two groups.

There were clear differences between the healthy and housebound groups. The former were in positive balance for reasons which remain unexplained, and the latter were in equilibrium. The mean dietary intake in the housebound elderly of 475 nmol was marginally in excess of the theoretical requirement of 419 nmol, but failed to reach the ESADI (Food and Nutrition Board, 1980). Plasma, whole blood and erythrocyte Se concentrations were lower than found in the healthy people, although whole blood GSH-Px activity was unaffected. Although we cannot prove that the Se status in this group was suboptimal, it is apparent that it was different from that of healthy elderly people.

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REFERENCES

- Abdulla, M., Kolar, K. & Svensson, S. (1979). *Scandinavian Journal of Gastroenterology* **14** Suppl. 52, 181-184.
- Armitage, P. (1971). *Statistical Methods in Medical Research*. Oxford: Blackwell Scientific Publications.
- Beutler, E. (1979). *A Manual of Biochemical Methods*, 2nd ed, pp. 71-73. New York: Grune and Stratton.
- Bunker, V. W. & Delves, H. T. (1987). *Analytica Chimica Acta* **201**, 331-334.
- Bunker, V. W., Hinks, L. J., Lawson, M. S. & Clayton, B. E. (1984a). *American Journal of Clinical Nutrition* **40**, 1096-1102.
- Bunker, V. W., Lawson, M. S. & Clayton, B. E. (1984b). *Journal of Clinical Pathology* **37**, 1353-1357.
- Bunker, V. W., Lawson, M. S., Delves, H. T. & Clayton, B. E. (1984c). *American Journal of Clinical Nutrition* **39**, 797-802.

- Bunker, V. W., Lawson, M. S., Stansfield, M. F. & Clayton, B. E. (1987*a*). *British Journal of Nutrition* **57**, 211–221.
- Bunker, V. W., Lawson, M. S., Stansfield, M. F. & Clayton, B. E. (1987*b*). *American Journal of Clinical Nutrition* **46**, 353–359.
- Bunker, V. W., Lawson, M. S., Stansfield, M. F. & Clayton, B. E. (1988). *Journal of Gerontology* (In the Press).
- Burch, R. E., Sullivan, J. F., Jetton, M. M. & Hahn, H. K. J. (1979). *Age* **2**, 103–107.
- Burk, R. F. (1976). In *Trace Elements in Human Health and Disease*, pp. 105–133 [A. S. Prasad and D. Oberleas, editors]. New York: Academic Press.
- Corrocher, R., Casaril, M., Guidi, C., Cabrielli, B., Mialto, O. & De Sandre, G. (1980). *Scandinavian Journal of Gastroenterology* **15**, 781–786.
- Csallany, A. S., Zaspel, B. J. & Ayaz, K. L. (1981). In *Selenium in Biology and Medicine*, pp. 118–131 [J. E. Spallholz, J. L. Martin and H. E. Ganther, editors]. Westpoint, CT: AVI Publishing Co.
- Flohe, L., Gunzler, W. A. & Loschen, G. (1979). In *Trace Elements in Health and Disease*, pp. 263–286 [N. Kharasch, editor]. New York: Raven Press.
- Food and Nutrition Board (1980). *Recommended Dietary Allowances*, 9th revised ed. Washington, DC: National Academy of Sciences.
- Gibson, R. S., Anderson, B. M. & Sabry, J. H. (1983). *Journal of the American Dietetic Association* **82**, 246–250.
- Gibson, R. S., Martinez, O. B. & MacDonald, A. C. (1985). *Journal of Gerontology* **40**, 296–302.
- Griffiths, N. M., Stewart, R. D. H. & Robinson, M. F. (1976). *British Journal of Nutrition* **35**, 373–382.
- Harman, D. (1956). *Journal of Gerontology* **11**, 298–300.
- Janghorbani, M., Ting, B. T. G., Nahapetian, A. & Young, V. R. (1982). *Analytical Chemistry* **54**, 1188–1190.
- Lane, H. W., Warren, D. C., Taylor, B. J. & Stool, E. (1983). *Proceedings of the Society for Experimental Biology and Medicine* **173**, 87–95.
- Levander, O. A. & Morris, V. C. (1984). *American Journal of Clinical Nutrition* **39**, 809–815.
- Levander, O. A., Sutherland, B., Morris, V. C. & King, J. C. (1981). In *Selenium in Biology and Medicine*, pp. 256–258 [J. E. Spallholz, J. L. Martin and H. E. Ganther, editors]. Westpoint, CT: AVI Publishing Co.
- Lloyd, B., Holt, P. & Delves, H. T. (1982). *Analyst* **107**, 927–933.
- Lloyd, B., Lloyd, R. S. & Clayton, B. E. (1983). *Journal of Epidemiology and Community Health* **37**, 213–217.
- Luo, X., Wei, H., Yang, C., Xing, J., Qiao, C., Feng, Y., Liu, J., Liu, Z., Wu, Q., Liu, Y., Stoecker, B. J., Spallholz, J. E. & Yang, S. P. (1985). *American Journal of Clinical Nutrition* **42**, 31–37.
- Miller, D. S. & Payne, P. R. (1959). *British Journal of Nutrition* **13**, 501–508.
- Miller, L., Mills, B. J., Blotcky, A. J. & Lindeman, R. D. (1983). *Journal of the American College of Nutrition* **4**, 331–341.
- Molin, L. & Wester, P. O. (1976). *Scandinavian Journal of Clinical and Laboratory Investigation* **36**, 679–682.
- Neve, J., Hanocq, M., Molle, L. & Lefebvre, G. (1982). *Analyst* **107**, 934–941.
- Persigehl, M., Schicha, H., Kasperek, K. & Klein, H. J. (1977). *Beitrage zur Pathologie* **161**, 209–220.
- Reamer, D. C. & Veillon, C. (1981). *Analytical Chemistry* **53**, 1192–1195.
- Reamer, D. C. & Veillon, C. (1983*a*). *Analytical Chemistry* **55**, 1605–1606.
- Reamer, D. C. & Veillon, C. (1983*b*). *Journal of Nutrition* **113**, 786–792.
- Stead, N. W., Leonard, S. & Carroll, R. (1985). *American Journal of Medical Science* **290**, 228–233.
- Stewart, R. D. H., Griffiths, N. M., Thomson, C. D. & Robinson, M. F. (1978). *British Journal of Nutrition* **40**, 45–54.
- Thomson, C. D., Rea, H. M., Robinson, M. F. & Chapman, O. W. (1977). *Proceedings of the University of Otago Medical School* **55**, 18–19.
- Thomson, C. D. & Stewart, R. D. H. (1974). *British Journal of Nutrition* **32**, 47–57.
- Thorn, J., Robertson, J., Buss, D. H. & Bunton, N. G. (1978). *British Journal of Nutrition* **39**, 391–396.
- Verlinden, M., van Sprundel, M., Van der Auwera, J. C. & Eylenbosch, W. J. (1983). *Biological Trace Element Research* **5**, 103–113.