

Trace nutrients. Selenium in British food

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1. The amount of selenium in nationally representative samples of prepared and cooked groups of foods, and in a variety of raw individual foods, was determined fluorimetrically.
2. The average British diet was calculated to provide approximately $60 \mu\text{g Se/d}$, of which half was derived from cereals and cereal products and another 40% from meat and fish. Milk, table fats, fruit and vegetables provided little or no Se.
3. Individual foods which were particularly rich in Se ($>0.2 \text{ mg/kg}$) included 'bread-making' and wholemeal flours, kidney, fatty fish, brazil nuts (*Bertholletia excelsa*) and several other varieties of nut. In contrast, breast milk and other foods for babies (except some cereal products) contained little Se.
4. The total intake, and the amounts of Se in major foods, were lower than in most other studies. This is probably the result of the comparatively low levels of this element in British soil.

The trace element selenium is an essential dietary constituent for experimental animals such as the rat, and for farm animals, but in excess it is toxic. The intakes which are necessary for health and those which are toxic are of considerable economic importance because the world-wide variation in the Se content of soil is so great that, for animals fed on local plants, both deficiency diseases (white muscle disease in sheep and calves, exudative diathesis in poultry and hepatosis dietetica in pigs) and toxicity diseases (blind staggers and alkali disease) are well known (Underwood, 1971). Symptoms of Se deficiency have also been induced in squirrel monkeys (Muth, Weswig, Whanger & Oldfield, 1971), and it now appears that Se is essential for man as a constituent of erythrocyte glutathione peroxidase (EC 1.11.1.9) (Awasthi, Beutler & Srivastava, 1975).

The intake of Se by man must also vary widely throughout the world, but uncomplicated Se deficiency has not been demonstrated; some symptoms of toxicity have, however, been noted in people living in seleniferous areas (Burk, 1976). The United Nations were unable to make any recommendations about dietary intakes (WHO, 1973), but the (US) National Research Council has recently interpreted information from animals as suggesting a human requirement of approximately $60\text{--}120 \mu\text{g Se/d}$ with toxicity occurring after prolonged ingestion of $2400\text{--}3000 \mu\text{g Se/d}$ (Food and Nutrition Board, 1976).

Little information is available for calculating Se intakes in Britain. The Se content of a wide variety of foods in America and Canada and some foods in New Zealand, which we may import, is known (Morris & Levander, 1970; Schroeder, Frost & Balassa, 1970; Arthur, 1972; Robinson, 1976), but no reliable analyses of foods grown or produced in Britain have been published. We therefore obtained typical samples of foods and determined their total Se content, and have also estimated the average daily intake in the United Kingdom from samples of the 'total diet' (Harries, Jones & Tatton, 1969). The results are presented and discussed in this paper.

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METHODS

Food samples

The average daily intake of Se was estimated by means of the food samples collected for the Ministry of Agriculture, Fisheries and Food's 'total diet' study. The organization of this study, and the foods included, have been described in detail elsewhere (Harries *et al.* 1969); in brief, selected colleges throughout the United Kingdom buy some 80 major foods in local shops, in amounts appropriate to their region and the season of the year, then prepare and cook them as for eating. The foods are subsequently combined into eight groups (including a new one of fish alone) for analytical convenience. The six colleges which provided samples for this particular study during the first quarter of 1974 were as geographically representative of Britain as possible, being in Cardiff, Edinburgh, Glasgow, Shrewsbury, Newcastle and Uxbridge (Middlesex).

In addition, we bought a variety of individual foods in the London area and the Se content of the raw edible portions was determined within 24 h. In some cases, more than one sample of the food was obtained for analysis. The foods were selected both on the basis of their importance in the diet and also to indicate the types of food which might be expected to contain particularly large or small amounts of Se; this helped to indicate which diets were likely to be higher or lower than average in this element. Although some subsequent loss of Se was expected from foods to be boiled, dry cooking would have resulted in comparatively little reduction (Higgs, Morris & Levander, 1972).

Analytical methods

Each food sample was digested with nitric and perchloric acids. Only when the organic matter was destroyed was sulphuric acid added, and the solution was then boiled until the nitric and perchloric acids had been removed. Hydrogen peroxide was added to ensure reduction of all Se to the tetravalent state. The Se was complexed with 2,3-diaminonaphthalene, extracted into cyclohexane, and estimated fluorimetrically at 518 nm. Full details of the analytical procedure have been published (Michie, Dixon & Bunton, 1978).

With this procedure recoveries of approximately 95% are normally obtained. The determination of the Se content of the National Bureau of Standards reference material no. 1577 (bovine liver) gave results of 1.1, 1.0, 1.2, 1.1, 1.0, 1.4, 1.0 and 1.1 mg/kg which are, with one exception, in agreement with the certified value of 1.1 mg/kg (95% confidence limits 1.0 to 1.2 mg/kg).

RESULTS

'Total diet' study

The ranges and mean Se contents of each food group are shown in Table 1. Values for individual colleges in the different geographical areas are not presented because the variations were as likely to reflect individual differences in sampling, washing, preparing and cooking the components (which the study was designed to include), as they were to reflect real regional variations in Se intake.

The fish samples, which included representative quantities of white fish, fatty fish, fish products, shell fish, canned salmon and other canned fish, were richest in Se; the small amounts eaten, however, mean that fish would have contributed only 5 µg/d to the average diet. The combined meat and fish samples and the cereal samples also contained significant amounts of the element, but the milk, fats, vegetables, and fruit and preserves, contained little or none. From the concentrations found, and the average amounts of these groups of food eaten in Britain (Ministry of Agriculture, Fisheries and Food, 1972), it was possible to

Table 1. Average daily intake of selenium in Britain, as estimated from 'total diet' samples,* January–March 1974

(Values given in parentheses are the no. of centres participating in the 'total diet' study)

Food group	Se content (mg/kg)		Estimated wt of food eaten (kg/d)†	Estimated intake of Se ($\mu\text{g}/\text{person per d}$)
	Range	Mean‡		
Cereals	0.03–0.23 (6)	0.11	0.27	30
Meat and fish	0.09–0.16 (6)	0.12	0.18	22
Fish	0.28–0.38 (5)	0.32	0.02§	5§
Milk	<0.01–0.01 (6)	0.01	0.40	4
Fats	0.01 (6)	0.01	0.08	1
Green vegetables	<0.01–0.02 (6)	0.01	0.11	1
Root vegetables	<0.01 (6)	0.005	0.21	1
Fruit and preserves	<0.01 (6)	0.005	0.25	1
Total			1.50	60

* For details see p. 392.

† Values <0.01 were taken as 0.005 when calculating mean values.

‡ From Ministry of Agriculture, Fisheries and Food (1972).

§ Values excluded from totals, where the contributions from fish are derived from the combined meat and fish group.

estimate that the average intake of Se in Britain is approximately 60 $\mu\text{g}/\text{person per d}$, of which 50% was derived from cereals and cereal products and approximately 40% from meat and fish together (Table 1).

Individual foods

The Se contents of the foods selected are shown in Table 2. Although the amount of Se would be expected to vary according to the geographical origin of each sample, no attempt was made in this study to quantify such variations. Instead these typical values were used simply as indicators of the most likely sources of Se in common diets.

DISCUSSION

The amount of Se in a food varies largely with its protein content and with the area in which it is grown (Burk, 1976). Half of the food we eat in Britain is home-produced (Ministry of Agriculture, Fisheries and Food, 1977) but although Se has been found in the soil of Devon, Staffordshire and North Wales (Webb, Thornton & Fletcher, 1966), there is little other direct information on the Se content of the soil in Britain or of the food crops grown upon it. Indirect evidence from the level of erythrocyte glutathione peroxidase in sheep indicates, however, that substantial areas of England and Wales are comparatively poor in Se (Anderson & Patterson, 1977). It is therefore not surprising that the estimated average intake of Se in Britain is, at 60 $\mu\text{g}/\text{d}$, somewhat lower than Canadian and American estimates, although higher than in New Zealand where much of the farmed soil contains inadequate Se. Average daily Se intakes in these countries have been reported as ($\mu\text{g}/\text{person}$): north-eastern United States 60–150 (Schroeder *et al.* 1970), Canada 110–220 (Thompson, Erdody & Smith, 1975), and New Zealand 25 (Robinson, 1976). In Europe, Swedish hospital diets provide from 23–210 $\mu\text{g Se/d}$ (Boström & Wester, 1968; Wester, 1971, 1974), Sweden having comparatively little Se in the soil, while Cresta (1976) has estimated the following intakes from dietary patterns without allowing for regional differences in the Se content of individual foodstuffs ($\mu\text{g}/\text{person per d}$): Netherlands 110, France 166, Italy 141.

An intake of 60 $\mu\text{g Se/d}$ in Britain would barely meet the tentative recommendation of 60–120 $\mu\text{g}/\text{d}$ (Food and Nutrition Board, 1976). But, as the condition most likely to

Table 2. *Selenium content (mg/kg) of selected foods included in the British diet*

Cereals	Se content*
Flour: Plain and self-raising	0.04
Bread-making	0.42
Wholemeal	0.53
Macaroni	0.16
Rice, long grain	0.10
Porage oats	0.03
Cornflakes	0.02
Soya-bean flour	0.09
Meat	
Beef	0.03
Lamb	<0.01
Pork	0.14
Liver: Ox	0.20
Lamb's	0.06
Kidney: Ox	0.91, 0.98, 1.28
Lamb's	0.63, 0.93, 1.22
Pig's	2.16, 2.40, 2.81
Heart, Ox	0.03
Fish	
Cod (<i>Gadus morhua</i>)	0.10
Mackerel (<i>Scomber scombrus</i>)	0.35
Herring (<i>Clupea harengus</i>)	0.45, 0.76
Kipper (<i>Clupea harengus</i>)	0.32
Milk and dairy products	
Milk	<0.01
Cheese, cheddar	0.12
Egg white	0.06
Egg yolk	0.20
Fats	
Butter	<0.01
Margarine	<0.01
Vegetables	
Potatoes (<i>Solanum tuberosum</i>)	<0.01
Carrots (<i>Daucus carota</i>)	<0.01
Cabbage (<i>Brassica oleracea</i>)	<0.01
Mushrooms (<i>Agaricus campestris</i>)	0.08, 0.10
Garlic (<i>Allium sativum</i>)	0.02
Sugar, fruit and nuts	
Sugar: White	<0.01
Brown	<0.01
Oranges (<i>Citrus aurantium</i>)	<0.01
Satsumas (<i>Citrus reticulata</i>)	<0.01
Apples (<i>Malus pumila</i>)	<0.01
Peanuts: Fresh (<i>Arachis hypogaea</i>)	0.03
Roasted (<i>Arachis hypogaea</i>)	0.04, 0.45, 0.66
Walnuts (<i>Juglans regia</i>)	0.19
Brazil nuts (<i>Bertholletia excelsa</i>)	2.3, 4, 9.5, 16, 17, 33, 42, 53
Hazel nuts (<i>Corylus avellana</i>)	<0.01
Cashew nuts (<i>Anacardium occidentale</i>)	0.27, 0.41
Sweet chestnuts (<i>Castanea sativa</i>)	<0.01
Almonds (<i>Prunus amygdalis</i> var. <i>dulcis</i>)	0.04
Beer	
Bitter	<0.01
Lager	<0.01
Baby foods	
Human milk	0.01–0.02
Infant milk, dried	0.08
Baby cereal	0.08, 0.35, 0.41
10 samples, various, canned or bottled	<0.01–0.06
Water	<0.001

* Where more than one value is shown, the additional values are for separate samples and not replicates for the same sample.

predispose to Se deficiency, protein-energy malnutrition in children (Burk, 1976), is not found, and New Zealand women with intakes of the order of 20–25 µg/d do not report any improvement in health even after doses of 1–2 mg (Robinson, 1976), it is unlikely that Se deficiency occurs. Indeed, the concentration in blood in England is comparatively high (WHO, 1973).

Individual intakes will vary considerably about the calculated mean, depending on the amount of protein in the diet, and in particular on the amounts of Se-rich foods which are eaten. Table 2 shows that foods which contained more than 0·2 mg Se/kg were 'bread-making' and wholemeal flours, some baby cereals, kidney, fatty fish and certain nuts, especially brazil nuts (*Bertholletia excelsa*). Certain people, including vegetarians and 'health food' advocates, might thus obtain substantially more than average, but still far below those intakes postulated as toxic (Food and Nutrition Board, 1976). In contrast, infants in Britain might have intakes of little more than 5 µg Se/d unless certain cereal products were eaten, for breast milk, cow's milk and other prepared infant foods all contained very little Se.

The values in Table 2 can provide only general indications of Se intakes in Britain, for the origins of the samples were in general unknown. Nevertheless, they are broadly compatible with published values if allowance is made for the low Se concentrations apparent in British soil.

Cereals. The Se in 'bread-making' flour was similar to North American values (Morris & Levander, 1970; Arthur, 1972) as it would probably have been imported from Canada or the USA, but the self-raising and plain flours, being largely indigenous, contained much less. As higher proportions of home-produced wheat are being used in bread, the Se content of bread must decrease. The values for macaroni, rice, porage oats and Cornflakes were all among the lowest in the literature (Arthur, 1972; Schlettwein-Gsell & Mommsen-Straub, 1972). The substantial difference in Se content between 'bread-making' flour and the other cereals probably accounts for the great variability found in the six cereal samples of the 'total diet' (Table 1), 0·27 kg of which would have contained from 8 to 62 µg of selenium.

Meat and fish. As in other studies (Schlettwein-Gsell & Mommsen-Straub, 1972; Arthur, 1972), kidney was one of the richest sources of Se. Nevertheless the values we obtained for offal, and for carcase meat and fish, were lower than in most other reports. The very low values for carcase meats must reflect the small quantities of Se provided by grass and feed-stuffs in Britain but it is not clear why the fish contained much less than the 0·9 mg/kg found in Canadian sea fish (Arthur, 1972). Despite this, meat and meat products (together with small amounts of fish) remained the other major contributors of Se to the average British diet.

Fruit and vegetables. Mushrooms (*Agaricus campestris*) and garlic (*Allium sativum*) are known to contain Se, but the values found were lower than those reported elsewhere. Se was otherwise not detected in any of the fruit or vegetables analysed.

Nuts. The Se content of brazil nuts averaged 22 µg/kg, far higher than the only other reported value of 1·03 µg (Schroeder *et al.* 1970). Se was also detected in peanuts, cashew nuts and walnuts, which shows that nuts could provide a significant quantity of Se for some people.

Milk, dairy products and fats. Se content was below the limit of detection in milk, as would be expected in a country whose soil and herbage are comparatively low (Underwood, 1971). Nor could any be found in butter or margarine. Cheese and egg yolk, while containing similar amounts to those found in America and Canada (Morris & Levander, 1970; Arthur, 1972) were not a significant source of Se in the British diet as a whole.

Beer. The absence of Se was expected, since little or no Se can be detected in drinking water in Britain.

Baby foods. Little Se was found in breast milk (see also, Department of Health and Social Security, 1977) or cow's milk, reflecting the low intakes of the mothers (from food) and cows (from feedstuffs). Neither was there much Se in canned or bottled baby foods, so, unless certain cereal products were eaten, infants in Britain, as in the Americas and New Zealand (Morris & Levander, 1970; Arthur, 1972; Millar & Sheppard, 1972; Shearer & Hadjimarkos, 1975), would ingest comparatively little Se.

Further interpretation of these low results must await studies of the forms in which Se occurs in British foods, for their rates of absorption into the body and metabolic effects differ (WHO, 1973). It must also await clarification of epidemiological relationships between high or low intakes of Se and the incidence of a number of diseases (Burk, 1976). It is apparent that there is no general likelihood of excessive intakes in this country, but because of the physiological interrelationships between Se, vitamin E and polyunsaturated fatty acids (Hoekstra, 1975), it is of interest that intakes of vitamin E (Smith, Kelleher, Losowsky & Morrish, 1971) and polyunsaturated fatty acids (Ministry of Agriculture, Fisheries and Food, 1976) also appear lower than in several other countries.

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REFERENCES

- Anderson, P. H. & Patterson, D. S. P. (1977). *J. Flour & Anim. Feed Mill.* **160**, 20.
 Arthur, D. (1972). *Can. Inst. Fd Sci. Tech.* **5**, 165.
 Awasthi, Y. C., Beutler, E. & Srivastava, S. K. (1975). *J. biol. Chem.* **250**, 5144.
 Boström, H. & Wester, P. O. (1968). *Acta Med. scand.* **183**, 209.
 Burk, R. F. (1976). In *Trace Elements in Human Health and Disease*, vol. 2, p. 105 [A. S. Prasad, editor]. London: Academic Press.
 Cresta, M. (1976). *Fd and Nutr. (FAO)* **2** (2), 8.
 Department of Health and Social Security (1977). *Rep. Hlth Soc. Subj.* no. 12.
 Food and Nutrition Board (1976). *Nutr. Rev.* **34**, 347.
 Harries, J. M., Jones, C. M. & Tatton, J. O'G. (1969). *J. Sci. Fd Agric.* **20**, 242.
 Higgs, D. J., Morris, V. C. & Levander, O. A. (1972). *J. agric. Fd Chem.* **20**, 678.
 Hoekstra, W. G. (1975). *Fedn Proc. Fedn Am. Soccs exp. Biol.* **34**, 2083.
 Michie, N. D., Dixon, E. J. & Bunton, N. G. (1978). *J. Ass. off. analyt. Chem.* **61**, (1).
 Millar, K. R. & Sheppard, A. D. (1972). *N. Z. Jl Sci.* **15**, 3.
 Ministry of Agriculture, Fisheries and Food (1972). *Survey of Lead in Food*, p. 26. London: HM Stationery Office.
 Ministry of Agriculture, Fisheries and Food (1976). *Household Food Consumption and Expenditure*: 1974. London: HM Stationery Office.
 Ministry of Agriculture, Fisheries and Food (1977). *Food Facts* no. 3.
 Morris, V. C. & Levander, O. A. (1970). *J. Nutr.* **100**, 1383.
 Muth, O. H., Weswig, P. H., Whanger, P. D. & Oldfield, J. E. (1971). *Am. J. vet. Res.* **32**, 1603.
 Robinson, M. F. (1976). *J. hum. Nutr.* **30**, 79.
 Schlettwein-Gsell, D. & Mommesen-Straub, S. (1972). *Int. J. Vitam. Nutr. Res.* **42**, 607.
 Schroeder, H. A., Frost, D. V. & Balassa, J. J. (1970). *J. chron. Dis.* **23**, 227.
 Shearer, T. R. & Hadjimarkos, D. M. (1975). *Arch. Environ. Hlth* **30**, 5.
 Smith, C. L., Kelleher, J., Losowsky, M. S. & Morrish, N. (1971). *Br. J. Nutr.* **26**, 89.
 Thompson, J. N., Erdody, P. & Smith, D. C. (1975). *J. Nutr.* **105**, 274.
 Underwood, E. J. (1971). *Trace Elements in Human and Animal Nutrition*, 3rd ed. London: Academic Press.
 Webb, J. S., Thornton, I. & Fletcher, K. (1966). *Nature, Lond.* **211**, 327.
 Wester, P. O. (1971). *Acta Med. scand.* **190**, 155.
 Wester, P. O. (1974). *Atherosclerosis* **20**, 207.
 WHO (1973). *Wld Hlth Org. Techn. Rep. Ser.* no. 532, Geneva.