Vitamins in Western diets

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Vitamins are now available at reasonable prices, much is known about modes of action and there is a rich literature on dietary surveys. Forty years ago the classical deficiency diseases were rampant and Edward Mellanby used to say that the difficulty was 'to get vitamins down the throats of those who need them'. Since that time normal diets have improved and welfare schemes have been adopted so that beriberi, pellagra, ariboflavinosis, rickets and scurvy have become rare in many countries. Free orange-juice and cod-liver oil for infants, the enrichment of white flour and the vitaminization of margarine contributed significantly to the improved situation (cf. Aykroyd, Joliffe, Lowry, Moore, Sebrell, Shank, Tisdall, Wilder & Zamecnik, 1949; Goldsmith, Darby, Steinkamp, Beam & McDevitt, 1950). Enrichment suits countries where staple foods are processed commercially, but is difficult where subsistence farming predominates. Nevertheless, even in India, fortification is in the short run the best way to reach a large number of people at little cost (Berg, 1973).

The price of synthetic vitamins decreases sharply with increasing utilization, and enrichment affords a cheap insurance, with practically no risk of hypervitaminosis. Margarine, once an inferior substitute for butter, is now in some respects a superior product; it contains vitamins A and D in stated amounts with no seasonal variations and is a good source of tocopherols and of essential fatty acids.

In the UK average intakes of vitamins exceed recommended daily allowances for all age groups and overt deficiency is very rare. This is true for many advanced countries but increasing heterogeneity of populations and diets raises complex issues, especially concerning the adaptation of immigrants to new surroundings. There is a great deal of evidence that despite satisfactory average intakes a significant minority of the population obtains less than the recommended allowance of one or more of the vitamins used as nutritional yardsticks.

Woodhill (1970) reported from Australia that intakes of vitamin A, \( \beta \)-carotene and thiamin were satisfactory but ascorbic acid intakes were low for a group of nineteen men (see Table 1). The difference between men and women needs further study (see also Brin, Dibble, Peel, McMullen, Bourquin & Chen, 1965). Women may possess a limited capacity for ascorbic acid synthesis.

In the USA a national appraisal of nutritional status has been undertaken. This is an immense task full of difficulties which make it necessary to revise procedures. Coursin (1970) reported briefly on the basis that dietary intakes below 70% of the
Table 1. **Ascorbic acid (AA) status of four groups of Australians investigated by Woodhill (1970)**

<table>
<thead>
<tr>
<th>Group</th>
<th>Description</th>
<th>Mean AA intake (mg/d)</th>
<th>Mean plasma AA (mg/l)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Women in a Church home</td>
<td>20</td>
<td>3.15</td>
</tr>
<tr>
<td>2</td>
<td>Women in their own homes</td>
<td>51</td>
<td>4.88</td>
</tr>
<tr>
<td>3</td>
<td>Men in hospital</td>
<td>54</td>
<td>2.78</td>
</tr>
<tr>
<td>4</td>
<td>Men receiving ‘Meals-on-wheels’</td>
<td>19</td>
<td>0.76</td>
</tr>
</tbody>
</table>

Recommended allowances could be 'identified as inadequate'. Frank deficiency was not seen and only 1–2% of the groups studied (and pre-selected as potentially vulnerable) showed any signs consistent with specific deficiency. (Dental surveys revealed a considerable incidence of tooth decay. Many children under 6 years of age were below expected height.) In five states, sample groups showed varied incidences of 'unacceptable' low values for serum vitamin A, serum ascorbic acid, urinary thiamin and urinary riboflavin (Table 2). Coinciding multiple unacceptable values were correlated with poverty (Table 3).

Table 2. **Vitamin status of selected groups of subjects in five states of the USA**

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Range of incidence of unacceptable† (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serum vitamin A</td>
<td>4–41</td>
</tr>
<tr>
<td>Serum ascorbic acid</td>
<td>0–3–16</td>
</tr>
<tr>
<td>Urinary thiamin</td>
<td>3–10</td>
</tr>
<tr>
<td>Urinary riboflavin</td>
<td>10–20</td>
</tr>
</tbody>
</table>

*From Coursin (1970).
†The customary categories of 'deficient levels' and 'low levels' were incorporated into 'unacceptable values' compared to 'acceptable values'.

These and similar investigations are not easy to interpret fully. Recommended daily allowances are meant to exceed bare requirements but the desired margin of safety is clearly eroded. More research is needed but in my view the primary requirement is for better education in nutritional fundamentals, particularly among vulnerable groups.
What has been said so far is probably familiar to this audience, but like Oscar Wilde I can plead that there is latitude for platitude especially when it comes to passing on knowledge.

Nutrition has its dogmas and one of them is that enough of a vitamin really is enough. Requirements are needed to replace losses due to side-reactions, as modes of action are mostly cyclic. A few scientists and many other people interested in nutrition hope (or believe) that extra vitamin intakes will do something more than ensure optimal ‘normal’ function. Official bodies have tended to revise recommended daily allowances downwards but enthusiasts want more. There is a widespread hankering after a ‘bonus’ role different from the recognized function, and this should not cause too much surprise. At one stage the use of vitamin A-rich fish-liver oils was encouraged so as to promote resistance to infection, and carrots were advocated to improve night vision. Multivitamin preparations were given to people already on a good mixed diet and indeed may have done good by suggestion! The latest development is the use of large doses of ascorbic acid against colds and of megavitamin therapy in psychiatry.

A majority of nutrition workers is increasingly anxious about misinformation in the popular literature and a recent set of appraisals in a special supplement of Nutrition Reviews (1974) expresses the situation. Bruch (1974) admits that ‘in some important aspects our rational scientific approach fails to fulfil the desperate needs of suffering people and it is to these needs that quacks and cultists address themselves’. Food faddism often appeals to well-to-do people reacting against excess or responding to emotional stress. They may find relief in a diversionary ‘cause’. Intelligent but scientifically unsophisticated people are game, with no off-season. Rynearson (1974) used the term hogwash (and defined it) and likened some of the marketing of special dietary preparations to the patent-medicine craze of the late 19th century. Even if propaganda borders on fraud there is no easy remedy, because incautious action could be counter-productive (see Henderson, 1974). Some of the books most criticized contain a beguiling mélange of true and false statements. They are easy to read and promise much, which may account for their vast sales.

Vitamin A

Intakes are still much too low for great numbers of people. Deficiency can arise from too little carotenoid provitamin in some ethnic groups or too little preformed vitamin, depending on the different dietary patterns. Moreover, the necessary carrier, retinol-binding protein in association with prealbumin, may not be synthesized in sufficient quantity when there is protein inadequacy. In developed countries a diversified diet permits substantial liver storage of vitamin A esters and deficiency should be rare. Unfortunately such a mixed diet is not obtained by everybody. Chase, Kumar, Dodds, Sauberlich, Hunter, Burton & Spalding (1971) studied 300 Mexican-American children under 6.5 years of age and found low serum vitamin A values (300 μg retinol equivalent/l) in half the group and very low values (100 μg retinol equivalent/l) in ten children. Sauberlich, Hodges, Wallace, Kolder, Canham, Hood, Raica & Lowry (1974) cite evidence that 10.2% of a
group of Negro-American children aged 1–5 years had values below 200 μg retinol equivalent/l.

Liver samples taken at autopsy in six American areas showed that about 40% of subjects aged 11–40 years had low or deficient levels (40 μg retinol equivalent/g liver). In five Canadian cities about 100 liver assays showed one in ten people with no liver reserves and many others having quite small stores. Moore (1957) found that about one-third of seventy-three persons killed in accidents had an average of 12.3 μg retinol equivalent/g liver.

It seems certain that a significant proportion of the population in Western countries is at some risk in respect of vitamin A status. The situation is much worse elsewhere. Pronounced deficiency often accompanies near-famine, and keratomalacia causes a vast amount of blindness and an even greater amount of permanent ocular disability (cf. Oomen, 1974). This is peculiarly sad because the effective remedies are obvious and attainable given some social, political and administrative competence.

**Vitamin D**

In 1919 one could see people with knock-knees and bandy legs every day in any large British city. Rickets is now rare and research on vitamin D deserves a big slice of the credit. Ergocalciferol is readily available and, in relation to the requirement, is cheap. Exposure of the body to sunlight is fashionable for cosmetic reasons, and anybody who considers the irradiation process carefully may well conclude that it is imperfectly understood, but that Nature has developed protection against hypervitaminosis resulting from sun-bathing.

Recommended intakes vary but 10 μg cholecalciferol equivalent/d is enough for most people. Fortification of milk, cereals, fruit drinks and margarine is permitted in one country or another. In fact the American Academy of Pediatrics found it not unusual for a child to consume as much as 50 μg cholecalciferol equivalent/d from all sources (White & Selvey, 1974). There is some risk of hypervitaminosis. In Britain the use of welfare cod-liver oil and the enrichment (0.25 μg/g) of baby foods helped to banish rickets, and the continued fortification of margarine is sound policy. Some immigrant groups have displayed rickets in recent years but remedial action has been taken.

Recent discoveries concerning 1,25-dihydroxycholecalciferol, calcitonin, parathyroid hormone and calcium-binding protein reveal interlocking systems which make naïve questions almost meaningless. Vitamin D requirement remains somewhat uncertain but important nutritional aspects of osteomalacia and osteoporosis are emerging. Vitamin D status may have a bearing on the age of puberty and on rates of growth, but by and large there seems to be as much danger of hypervitaminosis D as of deficiency.

**Vitamin E**

The (US) National Research Council advises that 'the apparent absence of vitamin E deficiency in the general population suggests that the amount of the
vitamin in foods is adequate' (Food and Nutrition Board, (US) National Research Council, 1974). The recommended daily allowance for adults (8–10 mg α-tocopherol equivalent/d) probably exceeds the normal intake (see Losowsky & Kelleher, 1975). An extended study carried out at the Elgin State Hospital, Illinois (Horwitt, Harvey, Century & Witting, 1961; Horwitt, 1962) showed that patients kept for some years on a diet very low in vitamin E had no clear signs of adverse changes in physical or mental health.

Absence of such a deficiency syndrome in man has not prevented large claims being made for heavy dosage with vitamin E. Official opinion in the USA rejects ‘miraculous’ effects attributed to vitamin E. The Food and Nutrition Board, (US) National Research Council (1974) lists many claims which ‘are not backed by sound experimentation or clinical observations’. White & Selvey (1974), writing under the heading ‘Vitamin E for everything’, conclude that ‘we’ve probably been handed an overdose of bunk about vitamin E’. Yet in sprue and malabsorption syndromes some benefit probably occurs and reports of benefit in intermittent claudication after high dosage cannot be discounted altogether (see also Mason & Horwitt, 1972).

Properly controlled trials are expensive and difficult but there is no other way to settle some disputes about vitamin E.

**Vitamin B₁₂ and folic acid**

Strict vegetarians in Western countries are at risk since vitamin B₁₂ comes from animal foods. Life-long Hindu vegetarians seem to remain healthy with abnormally low serum vitamin B₁₂ values.

Folic acid and vitamin B₁₂ are functionally interrelated, and premature babies, some pregnant women and some old people are at risk. Breast-fed babies exhibit better folic acid status than bottle-fed babies. Milk loses folic acid on pasteurization and after reheating only 30% remains.

Alcohol intake exacerbates marginal folic acid status and some drugs and contraceptive agents inhibit the enzyme folate conjugase and tend to produce deficiency.

**Ascorbic acid**

Recommended daily allowances (30–45 mg/d) are less than the average amount of ascorbic acid purchased (52 mg) in the UK after assuming that three-quarters of the vitamin in green vegetables and half that in root vegetables is lost in cooking. As little as 10 mg/d prevents scurvy, but a deficiency syndrome (lassitude, fatigue etc.) occurs well before scurvy or impaired wound healing appears. There is evidence that some elderly people are at risk. A body pool of about 1.5 g can be attained but thereafter large intakes are mainly excreted and doses above 0.5 g are not properly distributed over the pool. Ascorbic acid is ‘mobilized’ (on an ascorbic acid-free diet) at the rate of 3% per d. Thus initially it is used up at 45 mg/d but when the pool has fallen to 300 mg the loss is only 9 mg/d. Mental dysfunction has been recorded when the pool was halved, whereas scurvy appeared only when it
had fallen to 20% or less. An intake of 45 mg/d would probably maintain the body pool for most people.

Cigarette smokers have lower leucocyte ascorbic acid levels than non-smokers. Houston & Levy (1975) found that salicylamide, which is metabolized via sulphate and glucuronide, exhibits after intake of 2 g ascorbic acid a decline in the proportion converted to sulphate and an increase in the proportion forming the glucuronide. Tetracycline therapy depletes ascorbic acid stores and this may presage more difficulties. Pauling’s book, Vitamin C and the Common Cold (Pauling, 1970), has generated much controversy and greatly boosted the sales of ascorbic acid. The (British) Common Cold Research Unit (see Andrewes, 1973) found that ascorbic acid had no demonstrable effect in culture on several of the viruses implicated and no effects on trials with volunteers (Walker, Bynoe & Tyrrell, 1967). Some benefit was recorded by Wilson & Loh (1973) and by Anderson, Reid & Beaton (1972). At the onset of a cold Pauling (1970) recommends the use of one 500 mg tablet/h and if symptoms persist on the second day the regimen should continue with 4–10 g/d.

Careful double-blind tests using massive doses confirmed earlier findings with lower doses, but another test showed lessened disability. Nevertheless professional opinion (cf. White & Selvey, 1974) is pretty clear that no objective, conclusive evidence supports the theory that ascorbic acid is at all helpful: ‘Until such data are available ascorbic acid should not be used for this purpose’.

Performance in athletic and sporting events

Large numbers of people would be affected and some would profit financially if athletes, horses or dogs could by ingesting supplementary vitamins improve their performances by a very small margin over that of their competitors.

White & Selvey (1974), however, assert plainly that ‘it has not been shown that vitamin supplements are useful in improving the performance of a healthy athlete’. Comparison of ascorbic acid and placebo supplements using endurance tests and recovery from injury showed no difference (Gey, Cooper & Bottenburg, 1970).

When one class of ‘biochemical’ (e.g. anabolic steroids) works dramatically, persons lacking biochemical expertise may, understandably enough, expect vitamins to ‘work’ too. A valuable summary (Dairy Council Digest, 1975) has recently appeared.

The difficulty of this problem is very real. Suppose that supplementary vitamins could improve the performance of an athlete or a horse by 1%. How could that be proved? Or if it were not true, how could the claim be falsified? What degree of priority should be accorded to such tests? We must denounce false claims which delude the innocent, part them and their money or delay them from consulting a doctor. But what should we do about claims that are highly dubious but have not been (or cannot readily be) properly tested?

REFERENCES

Some aspects of diet and health


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