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Letter to the Editor

Pro-vitamin A carotenoid conversion factors: retinol equivalents — fact or fiction?

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Abstract

The vitamin A potential of a food is conventionally expressed as retinol equivalents, i.e. retinol plus the corresponding retinol equivalent of the provitamin A carotenoids. 1 mg of β-carotene, for example, is taken to have the same biological activity as 0.167 mg of retinol. However, this figure is based on certain assumptions regarding the amount of β-carotene absorbed and subsequently converted to retinol. This commentary considers the numerous food and host related factors which may ultimately determine carotenoid bioavailability and conversion to retinol. The authors endorse suggestions that until more definitive data on the potential absorption of carotenoids from foods is available, any calculated value of the retinol equivalent of the provitamins must be treated with caution. Furthermore, we would suggest that the use of current data on retinol equivalents in tables of food composition should be abandoned.

Keywords: Retinol equivalents; Carotenoids; Conversion factors

Vitamin A deficiency is a major nutritional problem in many underdeveloped populations throughout the world.

Its consequences, xerophthalmia, blindness and premature death, are common especially in children. Vitamin A is also important in maintaining growth and reproductive efficiency, maintenance of epithelial tissues and prevention of keratinization, and has an important action in immune response.

The problem in these populations results from a low or negligible intake of preformed vitamin A (retinol) from animal sources. The primary source of vitamin A in these populations is the provitamin A carotenoids from fruits and vegetables.

A number of carotenoids, the so-called provitamin A carotenoids which include β and α-carotene and β-cryptoxanthin, have the capacity, although not the same capacity, to be converted to vitamin A.

Current evidence indicates that not only is the concentration of provitamins and other carotenoids in foodstuffs very different but large variations can occur even in the same type of food, due to varietal differences, stage of maturity, climatic conditions, processing and/or cooking.

The efficiency of absorption of carotenoids is affected by the amount of the carotenoid ingested, processing and/or cooking of the food, other dietary ingredients which may stimulate (e.g. type and amount of dietary fat) or inhibit (fibre) absorption, matrix effects, interactions between carotenoids, between individual variation, parasitic infestation, current nutritional status, etc.

The provitamin A carotenoids are converted enzymatically to retinol mainly in the intestinal mucosa. The enzyme activity is dependent on, amongst other things, the level of protein in the diet. The retinol formed is then handled in the same way as dietary retinol. On the basis of central enzymatic cleavage, one molecule of β-carotene gives two molecules of retinol; expressed in μgs, 1 μg β-carotene is theoretically equal to 1 μg retinol.

However, those factors which affect absorption of the provitamins must also have an effect on the amount of the provitamin which can be subsequently converted to Vitamin A, a major factor being the amount of provitamin from the diet which is presented to the intestinal mucosa.

Under normal conditions of vitamin A status, the serum concentrations of vitamin A are homeostatically

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controlled and the concentration remains relatively constant. The extent and mechanism of regulation of the conversion of provitamins to vitamin A is unclear at the present time.

Hume and Krebs (1949; the Sheffield Experiment) found that the absorption of β-carotene from vegetables and carrots was on average only one third that of β-carotene in oil.

In 1967, an FAO/WHO group recommended "that in the absence of more specific data for foods, the availability of β-carotene be taken as one-third (ie approximately 33% of the β-carotene from the diet was available for absorption) and that the utilisation efficiency in human beings be taken as one-sixth, (assuming 50% conversion to Vitamin A)". That is, 1 mg β-carotene in the diet is taken to have the same biological activity as 0.167 mg retinol.

In 1974, the NAS/NRC *Recommended dietary allowances* proposed "that in addition to any expression as international unit activity, Vitamin A should also be given in terms of retinol equivalents defined as follows

1 retinol equivalent (RE)	= 1 μg retinol
	= 6 μg β-carotene
	= 12 μg other carotenoid Vitamin A precursors.

These figures are generally still in use at this time.

Simpson and Tsou (1986), however, considered the NAS/NRC ratios were probably on the conservative side. In 1988, FAO/WHO suggested factors based on β-carotene intake; at <1000 μg β-carotene intake then 1 μg retinol (or 1 RE) = 4 μg β-carotene, 1000–4000 μg intake then 1 μg retinol = 6 μg β-carotene and >4000 μg intake 1 μg retinol = 10 μg β-carotene.

The retinol potential of a food is conventionally expressed as retinol equivalents, i.e. retinol plus the corresponding retinol equivalent of the provitamin.

In a 1942 article, Graves discussed the vitamin A value of carotene in vegetables in relation to their carotenoid content and apparent biological activities. He concluded that "where matters of policy affecting the nutrition of the population are under consideration, no results [chemical or physical determinations of the carotene content] should be accepted [to define potential Vitamin A activity] unless they are supported by biological findings". Nearly 60 years on we are still struggling with the same problem.

Difficulties in interpreting population studies were demonstrated in a study by de Pee, West, Muhilal, Karyadi and Hautvast, 1995 who reported on the feeding of stir fried vegetables supplements or β-carotene enriched wafers to breast feeding women with low haemoglobin levels in Indonesia. Only 3% of the study population were actually vitamin A deficient. In the vegetable group there were no significant changes in serum retinol, β-carotene or other serum carotenoids and breast milk retinol. However, in the enriched wafer group there was a mean increase in serum retinol of 0.32 μmol/l from a baseline level of 0.84, serum β-carotene 0.73 from 0.19, and breast milk retinol of 0.59 from 0.84. The authors concluded that the approach to combating vitamin A deficiency by increasing the consumption of provitamin A containing vegetables should be re-examined. However, this study may indicate that the factors affecting the absorption of food source carotenoids mentioned above and in particular the matrix effect may in part be responsible for the lack of response to the vegetable supplement.

Whilst the processing and/or cooking of vegetables may cause all-*trans*(all-E)/cis(Z) isomerisation of carotenoids, the *cis* isomer having lower vitamin A activity, studies have suggested that the absorption of carotenoids from raw vegetables is probably less than 10% and up to 50% or more from 'cooked' materials. This and the factors mentioned above will undoubtedly have an effect on the potential amount of the ingested carotenoid converted to vitamin A. Thus, assuming 5% absorption and 50% conversion, the factor would be 1:40 and, at 75% absorption, 1:2.7.

A fundamental, yet often ignored aspect, of 'feeding' studies is the accurate determination of the carotenoid content of the food eaten. The calculation of the apparent carotenoid content and retinol equivalence of foods calculated from food composition tables can lead to inaccuracies. These may be compounded in the longer term feeding studies where there may be day to day variation in the food lots and cooking conditions. Unlike the situation with the carotenoid fed in capsule form or a singe batch of a pre-prepared foods with a long shelf life where a single analysis of the carotenoid content may be sufficient, where foods are prepared daily they will require monitoring on a daily basis. It could be argued that the unknown factors associated with absorption and conversion may be far greater than any inaccuracies associated with the calculation or measurement of the actual carotenoid content of the food. However, in order to measure absorption or availability of the carotenoid for conversion, it is a prequisite that the amount ingested is measured accurately.

Whilst studies with foods are important in establishing their effects on vitamin status, other studies with 'pure carotenoids' are essential in helping to understand the mechanisms of carotenoid absorption and metabolism.

In a novel approach (Faulks, Hart, Wilson, Scott & Southon, 1997), the apparent absorption of a physiological dose of 10 mg of β-carotene was assessed by the measurement of the unabsorbed fraction of the vitamin in the ileostomy effluent of ileostomy subjects. Average absorption was 90% (74–97%). In a similar study (Faulks, personal communication), when subjects were fed whole-leaf or chopped spinach 'meals' containing

15.5 mg lutein and 10.7 mg β-carotene, the absorption of both was between 20 and 30%. Whilst there was no discernible increase in plasma carotenoid concentrations, there was a marked increase in the carotenoid concentrations in the plasma chylomicron fraction between 4 and 8 h after consumption of the meal. This type of experimental approach indicates that the lack of a plasma response cannot be interpreted as a lack of absorption, and disproves the contention that there is little or no absorption of β-carotene from green leafy vegetables.

In another recent study (Borel et al., 1998), a prepared meal containing a pharmacological dose of 120 mg of β-carotene in oil was fed to healthy volunteers. In summary, the authors concluded that the efficiency of intestinal absorption of β-carotene seems highly variable in healthy humans, but is apparently a constant characteristic of individuals. The variability of the chylomicron β-carotene response appears to be due mainly to differences between individuals in the efficiency of intestinal absorption and chylomicron secretion and clearance. The inter-individual variability of the apparent vitamin A activity of β-carotene ingested is also high, although the higher the amount of β-carotene absorbed, the higher the amount of retinol palmitate is secreted into the chylomicrons. Thus it can be concluded that the dietary equivalence of β-carotene and retinol varies greatly among individuals.

Current, albeit limited, research using modern approaches to assess the absorption of carotenoids from various foods may in the future enable a more accurate estimate of potential retinol equivalence to be made. However, the numerous combinations of food- and host-related factors that ultimately determine carotenoid bioavailability in individuals or populations, which include large variations in dietary preferences, processing and methods of food preparation, would mean that any data on absorption of individual foods is bound to have a limited practical application.

The authors would endorse suggestions that until more definitive data on the potential absorption of carotenoids from various foods is known, any calculated value of the retinol equivalent of the provitamin must be treated with caution. However, large variations in the potential and actual absorption of β-carotene from foods highlight the fact that a single conversion factor is inappropriate.

We would, therefore, further suggest that the use of current data on retinol equivalents should be abandoned, and tables of food composition should quote only retinol and the individual carotenoids as amount per 100 g edible portion.

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