Beta carotene - Statement on the effects of excess Vitamin A on maternal health

In this guide

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44 Allen and Heskell (2002) found no reports of high-carotene intakes from foods ever having caused vitamin A toxicity. It had been assumed that about

one-third of a dose of dietary carotenoids was absorbed and half that amount was converted to retinol, resulting in a bioconversion factor of 6:1 for b-carotene to retinol. This bioconversion factor had been used in most food composition tables to convert carotenoids to retinol equivalents. However, in the early 1990's it become apparent that absorption of carotene from plant sources, especially from vegetables, was substantially less than one-third of that absorbed from a dose given in oil. More recent estimates of b-carotene absorption from a diet consisting mainly of vegetables showed that absorption was about one half of what was previously assumed. Based on such studies, the Institute of Medicine estimated that 1 µg RA equivalent was equal to 12 mg of b-carotene instead of the 6 mg of b-carotene estimate used previously.

Acute and chronic toxicity

45 Acute clinical features of vitamin A toxicity in age groups other than infants are lethargy, pain in the joints, dry skin, headache and nausea and vomiting, although these vary in severity. More severe signs that are diagnostic of hypervitaminosis A clinically include alopecia, drowsiness, liver and bone damage and visual problems (Loughrill, 2016; SCF, 2002). In infants, the major sign of toxicity is bulging fontanelles.

46 Signs of chronic toxicity include dry thickening of the skin, cracking of lips, conjunctivitis, erythematous eruption, alopecia, reduced bone mineral density, bone joint pain, chronic headache, intracranial hypertension and hepatotoxicity. Some adverse effects, for example hepatotoxicity, are regarded as reversible with withdrawal of the vitamin but others, such as deficits in the eyes and bone, are not. Kamm, 1982).

47 Penniston and Tamuihardjo (2006) found that few human studies had looked at the acute effects of a large dose of vitamin A on circulating vitamin A concentrations. Evidence suggested that intermediate effects without clinical signs of toxicity may be a growing concern, because intake from preformed sources of vitamin A often exceeded the recommended dietary allowances (RDA) for adults, especially in developed countries. Osteoporosis and hip fracture have been associated with preformed vitamin A intakes of only twice the current RDA. Assessing vitamin A status in cases of intermediate effects or overt toxicity is complicated because serum retinol concentrations are non-sensitive indicators in this range of liver vitamin A reserves.