Invited commentary

The effects of iron and copper status and of dietary carbohydrates on the activity of rat intestinal \( \beta \)-carotene 15,15\(^{\prime} \)-dioxygenase

In much of the world, the primary dietary sources of vitamin A (an essential nutrient for vision, the immune system, reproduction and growth) are coloured fruits and vegetables rich in carotenoids (Underwood & Arthur, 1996; Olson, 1999). A sobering fact in this regard is that millions of young children, pregnant women and lactating mothers do not ingest enough carotenoids and vitamin A to fulfil their physiological needs (Sommer & West, 1996; Underwood & Arthur, 1996; Olson, 1999). As a consequence, a variety of public health strategies are being used to improve the vitamin A statuses of these at risk groups (Sommer & West, 1996; Underwood & Arthur, 1999). Thus, in Cu deficiency, anaemia would be expected, probably associated with a decline in energy production and an accumulation of Fe in internal organs. In rats, but not necessarily in other species, fructose or sucrose, when substituted for maize starch in the diet, seems to enhance the onset of Cu deficiency (Turnlund, 1999).

That preamble brings us to the interesting paper by During et al. (2000) published in this issue of the British Journal of Nutrition. Groups of rats were fed low, medium or high amounts of ferric citrate in combination with a Cu-deficient or Cu-adequate diet. Either starch or fructose was supplied as the carbohydrate source. The question was: ‘What effect do these different dietary regimens have on the carotene cleavage enzyme in the intestine?’ Let us consider first the starch-fed rats.

In Cu-deficient rats, the concentration of Fe in the liver, but rather oddly, not in the intestinal mucosa, expectedly doubled at the higher two levels of Fe intake. The activity of the intestinal cleavage enzyme was also consistently higher in Cu-deficient than in Cu-sufficient rats and was directly proportional to the Fe content of the intestinal mucosa.
a bit surprising, in that one might expect the enzyme to sequester Fe, such that its activity would reach an optimum at medium intake levels of Fe. As the level of dietary Fe also enhanced cleavage activity in Cu-sufficient rats fed on fructose, but not in those fed on starch, however, other factors are clearly at work here. This study confirms and extends recent studies of the same group (During et al. 1999) that Fe is essential for carotenoid cleavage.

The number of factors, therefore, that influence the activity of the intestinal carotenoid cleavage enzyme keeps growing. The intestinal enzyme activity is enhanced by vitamin A deficiency, polyunsaturated fats in the diet, Cu depletion, fructose feeding, and glutathione, and is inhibited by protein deficiency, heavy metals that bind to its essential sulfhydryl groups, and some aromatic phytochemicals. Whether carotenoid cleavage enzymes in the liver and other organs are similarly affected merits attention.

In closing, it makes sense that an enzyme responsible for vision and for the integrity of several key physiological processes for the vast majority of humans worldwide would be influenced both by nutritional status and by diet. Further developments must certainly be in the offing.

References


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