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Editorial

With world attention now focusing on the Olympics, the question as to the extent to which nutrient intakes can affect athletic performance is highly topical; in this context, the article in this issue by Margaritis & Rousseau[1], dealing with the antioxidant requirements of athletes, is rather timely. Competitive athletes are renowned for their dedicated pursuit of anything that might give them that extra 'edge', and diet and dietary supplements are often an integral component of training schedules, designed to allow the athlete to produce the best performance possible on the day of competition. Antioxidant requirements would be expected to be higher among athletes, given the stresses placed on their bodies; but which of the various antioxidants are needed, and how much at the various different stages in training, and from what source? Margaritis & Rousseau survey the available literature on all the usual suspects, including vitamins C and E, together with the carotenoids, flavonoids, Se, Zn and Cu, and find little convincing evidence supporting beneficial effects of antioxidant supplements in well-trained athletes, unless their nutrient status was already deficient. Furthermore, there are potential risks associated with the taking of high doses of certain antioxidant nutrient supplements, leading these authors to advise that individual biological assessment of status should be used to identify any nutrient deficiencies and hence any possible requirement for supplements, and that, even among athletes, their needs can usually be met by consuming a balanced, nutrient-dense diet.

One of the benefits of physical activity is its positive effect on mood, but, as all chocoholics can affirm, there are also dietary components that can affect mood. Appleton *et al.* [2] here present an investigation into the effects of *n*-3 PUFA on mood and behaviour. Various mechanisms could be involved, including membrane effects and the biosynthesis of eicosanoids; PUFA have also been shown to affect various neurotransmitter pathways and cytokine production, and to modulate ion channel activity. However, results from epidemiological, clinical and intervention studies are generally conflicting and inconclusive, due partly to differences in design and methodology. Further to confound the issue, not only might PUFA influence mood, but (intriguingly) mood might also influence PUFA status via, for example, effects on dietary intake, or on Δ5 and Δ6 desaturase enzyme activities (stress-related). It is also possible that individual differences in PUFA metabolism could explain some of the differences in reported efficacy of *n*-3 PUFA in treating conditions such as depression, attention deficit hyperactivity disorder and schizophrenia. The study of the polymorphisms in the genes coding for the enzymes responsible for PUFA metabolism should provide some interesting insights in the future, and these authors conclude by calling for more research into the possible mechanisms by which *n*-3 PUFA could affect mood and behaviour, as well as for well-conducted intervention trials.

The prospect of cognitive decline is a cause for depression, thus nutritional interventions that might ward this off would be very welcome. According to Young & Kirkland[3], one nutrient implicated here is niacin, which has recently been shown to be converted to cyclic ADP-ribose (cADPR), an intracellular Ca signalling molecule thought to have a role in learning and memory which, in the brain, may be formed via the activity of the enzyme CD38. This review explores the link between niacin and learning behaviour in mouse and rat models using niacin deficiency, or supplementation, or a model in which the gene for CD38 was deleted. Perhaps surprisingly, their findings suggest an inverse relationship between spatial learning ability and dietary niacin intake and cADPR, so the relationship is not straightforward; nonetheless, investigation of this pathway could possibly help identify potential molecular targets for clinical intervention aimed at preventing or even reversing cognitive decline.

Ca is implicated in the protective effect of dairy products against colorectal cancer (CRC), another scourge of 'Westernised' populations. However, dairy products are many and varied in their nutritional composition, so the effect could be due to other components as well as Ca, which itself could exert its effect by a number of different mechanisms. Maria Pufulete[4] here presents the evidence from epidemiological and prospective studies, demonstrating that the most consistent inverse relationship is between milk consumption and CRC risk, whereas the evidence is mixed in the case of hard cheese, which could be responsible for some cancer-promoting effects. Vitamin D probably acts synergistically with Ca, as shown in animal studies; also, studies on genetic variations in the vitamin D receptor in humans have shown a relationship with CRC risk; the latter constitute important evidence since they are not dependent on estimations of dietary intake and sun exposure, which have been omitted from many studies due to methodological difficulties. Of the many other components present in dairy products, conjugated linoleic acid, produced in the rumen from linoleic acid, is a strong candidate for conferring protection against CRC, possibly via an antiproliferative effect; also, probiotic bacteria in fermented dairy products, butyric acid, sphingolipids and certain milk proteins may be protective. Thus, somewhat as in the case of fruit and vegetables, it would appear that it is advisable to consume the foods in order to achieve the benefit, rather than to rely on taking a supplement.

This being so, the study of rumen biology perhaps assumes a greater importance than hitherto, and the review by Mohamed & Chaudhry[5] in this issue addresses the difficulties associated with studying the degradation of feeds in the rumen. In the past, the most effective methods have involved either suspending a porous bag containing the feed into the rumen of a fistulated animal, or extracting rumen fluid via the fistula to use in the laboratory; these techniques have obvious cost implications, and are becoming increasingly unacceptable from the point of view of animal welfare. Thus, alternative

methods are needed, and these authors present the case for a number of *in vitro* techniques that do not require the surgical modification of ruminant animals, and which offer the advantages of speed and simplicity. With adequate standardisation, these methods could provide data enabling ruminant feeds to be balanced appropriately according to the desired outcome (reproduction, milk production, lean tissue deposition, etc), leading to efficiencies of production that will benefit us all.

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