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Editorial

Although this is the first issue of *Nutrition Research Reviews* for which I am responsible as Editor-in-Chief, the contents reflect material that I inherited from my predecessor, Professor Mike Forbes. During his editorship he established a healthy list of reviews in progress, and I am reaping the benefit. Mike was also involved in the decision to change the format of the journal, to match that of the other Nutrition Society journals; the change in cover colour was because a number of members of the editorial board thought the brown and orange was depressing and uninspiring on the shelf. I hope that the new colours are more inspiring.

It may or may not bode well for my editorship that the first paper in this issue has the word 'optimistic' in its title. Miles & Scaife (2003) consider the problem of optimistic bias, the observation that people believe that they are less likely to experience negative events than are others. If individuals see themselves as less at risk than others, they may not be influenced by health promotion messages, believing them to be aimed at others. They consider two aspects of optimistic bias in relation to food: the likelihood of suffering 'specific health effects' associated with diet and the likelihood of experiencing negative effects associated with 'potential food hazards'.

We often say that nutrition encompasses everything from the farmyard, via the supermarket to the table and into the body; it is rare to find discussion of farm production and health effects in a single paper, but this is what Lyons *et al*. (2003) have achieved. They review the evidence linking Se deficiency with health risks, and discuss the benefits of increased intakes (in the light of evidence that in most parts of the world intake is falling). They then discuss ways in which Se intake can be increased, by use of Se-containing fertilisers and/or selective breeding of strains of wheat that accumulate Se more effectively. They conclude with the caveat that before we embark on any programmes to increase Se intake we should await the results of trials of Se supplementation in progress concerning cancer, asthma and HIV and AIDS.

Three papers in this issue have a genetic flavour, showing the impact of molecular genetics and decoding of the human genome (and others) on nutrition as well as other sciences. Swallow (2003) reviews the genetics of carbohydrate digestion from a nutritional perspective, covering a variety of conditions, with special emphasis on the phenomenon of lactase persistence in north European populations and nomadic pastoralists. In these populations that traditionally consume fresh milk there is evidence of selective pressure to retain lactase after adolescence; she suggests that in northern Europe the pressure was the importance of milk as a source of Ca and vitamin D, while among desert nomads the water content of milk may have been the important factor. She also discusses sucrase–isomaltase deficiency, which affects about 10 % of Inuit, whose traditional diet was devoid of sucrose, and the dire consequences for infants of the introduction of sucrose in the mid-twentieth century.

Wells & Bennik (2003) review the impact of modern genomics on food microbiology. Food-borne illness remains a serious problem worldwide, and bacterial evolution is rapid, so that as one hazard is brought under control, another appears. Comparative genomics is providing new insights into bacterial evolution and, perhaps more importantly from a practical point of view, there are now DNA micro-arrays that permit rapid identification of pathogenic organisms and individual genes associated with virulence. Perhaps this new information will permit us to get more than one step ahead of the bacteria.

The third genetic paper reviews the links between genomic instability and adverse health outcomes. Fenech (2003) suggests that, in the absence of exposure to gene-damaging toxins, genetic instability, as detected by micronucleus assay, is a sensitive indicator of nutritional deficiency. He raises the hope of setting vitamin and mineral requirements to maximise genetic stability and hence promote good health, a far more desirable aim than the present estimates of requirements to prevent deficiency.

The review by Champ *et al*. (2003) has a more traditional theme, the continuing debate as to what we mean by dietary fibre: should the definition include resistant starch? Is 'NSP', which can at least be characterised chemically on the basis of constituent monosaccharides, a more useful measurement than 'dietary fibre', which includes non-carbohydrate materials? They conclude that none of the current answers are wholly satisfactory, either scientifically or with respect to food labelling and health claims.

Dardevet *et al.* (2003) review the evidence that leucine is the key signal for increased protein synthesis in the post-prandial state, acting (independently of insulin) to activate protein kinases, as well as reducing protein catabolism. They then demonstrate that the response is blunted in aging rats, which require higher than normal concentrations of leucine to stimulate protein synthesis. This leads on to speculation that leucine supplements may be beneficial in preventing the loss of muscle tissue (sarcopenia) associated with aging, and may serve to maintain a higher total body protein content in the elderly, with possible benefits for immune function. It remains to be seen, of course, what effect leucine supplementation might have on the metabolism of tryptophan and other large neutral amino acids (Bender, 1983).

Since its discovery, less than a decade ago, leptin has grown from a hormone that was believed to control long-term appetite by signalling the size of adipose tissue reserves, into a hormone that has a major role in lipid metabolism, regulation of energy balance, immune function, growth and development and the control of fertility. Houseknecht & Spurlock (2003) review the role of leptin in

regulating lipid metabolism and the effects of diet on leptin function. Dietary fat intake affects the expression of the leptin gene, and its receptor, as well as later events in the response to leptin signalling. Our old villain saturated fat has a new role; saturated fatty acids seem to cause leptin resistance.

Continuing with the theme of fat metabolism, Mittendorfer & Klein (2003) review the factors that control the use of fat and carbohydrate reserves during endurance exercise. They note that while the intensity of exercise determines the increased requirement for energy-yielding metabolism, it is the individual's maximum aerobic capacity that, together with the intensity of the exercise, determines the relative amounts of plasma glucose and fatty acids, muscle glycogen and intramuscular triacylglycerol that will be used. Diet and body composition also affect fuel utilisation in exercise, as does gender, and for women the menstrual cycle also affects fuel selection. Fatty acids (from adipose tissue) provide 40–80 % of the fuel for moderate exercise, but as the intensity of exercise increases, proportionally more carbohydrate is consumed, suggesting that prolonged moderate exercise is more beneficial than high-intensity exercise for those who exercise to lose excessive adipose tissue.

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