Nutrition Research Reviews (2005), **18**, 1–2
DOI: 10.1079/NRR2005105

Editorial

Anyone who lives near a pig farm knows the unpleasant odour of the excreta from intensively farmed animals, and when the farm is in or near a centre of population this can create a considerable nuisance. Anything that can be done to reduce the odour is obviously desirable. Phung *et al.* (2005) discuss the various odiferous compounds and how diet affects their production by intestinal bacteria: sulfur compounds, phenols and amines, volatile fatty acids, ammonia and amines. Their conclusion is that what is important is the balance between dietary protein and fermentable carbohydrate (i.e. carbohydrates that are not digested in the small intestine, but provide a substrate for bacterial fermentation in the large intestine). When intestinal bacteria are given a plentiful source of fermentable carbohydrate they produce large volumes of gas, but utilise most or all of the available amino acids for protein synthesis and increase in biomass. By contrast, when they have little fermentable carbohydrate, they have to utilise amino acids as metabolic fuels; bacterial metabolism of tryptophan leads to foul smelling indole and skatole, and the sulfur amino acids yield a variety of equally unpleasant volatile compounds. Well-fed intestinal bacteria are obviously as important for the quality of life of those around animals as for the intestinal health of the animals themselves, and we can assume that the same arguments apply to the metabolism of human intestinal bacteria as well.

Those who prepare diets for laboratory rodents frequently include choline, although in human nutrition we consider choline to be a marginal compound that is (almost certainly) not required in the diet, since it can readily be synthesised by successive methylation of phosphatidylethanolamine. However, this is at the expense of adenosylmethionine, and hence dependent on adequate methionine cycle activity and especially adequate folate status. Choline is metabolised by oxidation to betaine (trimethylglycine), which can act as a methyl donor in a variety of reactions, and betaine is commercially available in large amounts as a by-product of sugar refining. Eklund *et al.* (2005) explore the potential of betaine as a feed additive, both as a source of methyl groups (hence sparing methionine and choline) and also to protect the intestinal tract against osmotic stress occurring during diarrhoea in pigs and coccidiosis in poultry. The effects on intestinal health are especially important in view of the restriction of antibiotics in animal feedstuff.

Biochemists traditionally regard urea as a non-toxic endproduct of N metabolism that is simply excreted in the urine; physiologists know it is important in maintaining the osmotic gradient for water resorption in the kidney. Ruminant nutritionists have long known that bacteria can utilise urea as a source of N for amino acid synthesis, providing protein that is available to the animal host. Regoeczi *et al.* (1965) demonstrated that there is considerable enterohepatic circulation of urea in non-ruminants, with bacterial hydrolysis to yield ammonia, which may be utilised by the bacteria themselves, or may be absorbed into the hepatic portal circulation and used for amino acid synthesis in the liver. Jackson (1995) discussed the potential relevance of this salvage of urea N for human protein requirements a decade ago. Stewart & Smith (2005) discuss the importance of urea N salvage in both ruminants (which secrete much urea in their saliva, as well as having intestinal urea transporters) and non-ruminants, which also have functional intestinal urea transporters. They cite a number of studies showing that urea salvage makes a significant contribution to human N balance, and can compensate, to a limited extent, for inadequate protein intake.

Benton (2005) asks whether artificial sweeteners help to control body weight and prevent obesity. He reviews the mechanisms involved in regulating food intake, hunger and satiety, and suggests that the consumption of low-energy-density foods leads to a later compensatory increase in energy intake. He also notes that fat, rather than sugar, is the important determinant of energy intake. He concludes that while low-energy-density foods may be helpful in weight reduction, when individuals show restraint in eating, there is no evidence that they are beneficial in long-term weight maintenance. My (cynical) observation of overweight Americans (and others) guzzling large volumes of sugar-free 'diet' drinks supports his view.

We are all subject to multiple stresses, and stress-related disorders are estimated to affect 450 million individuals worldwide. Hamer *et al.* (2005) review the ways in which the endocrine and sympathetic nervous systems can be targeted to reduce the effects of psychological stress, and the various nutrients and non-nutrients that may be effective, and could be included in functional foods for stress relief. They conclude that while there are promising candidates, there is, as yet, little evidence, and there is a need for more psychobiological research.

Kidney diseases are a major cause of morbidity, and cardiovascular mortality is higher in kidney patients than in the general population – an observation that is not explained by traditional cardiovascular risk factors. Caimi *et al.* (2005) discuss the nutritional factors that affect patients in renal failure – it is a chronic inflammatory state, and there is accelerated atherosclerosis, exacerbated by malnutrition. They conclude that improved nutrition would improve the prognosis considerably.

Nutritionists are sometimes seen by the public as being negative, drawing attention to the hazards of (excessive) consumption of most foods that are pleasurable. Certainly I can remember sitting through Nutrition Society meetings and coming to the conclusion that there is little, if anything, that it is safe to eat, having heard evidence of the adverse effects of fat, sugar, vitamins, minerals and protein. Perhaps nutrition is entering a more positive phase. In recent issues of *Nutrition Research Reviews* we have published papers showing beneficial health effects of wine (Buemann *et al.*

2002; Cooper *et al.* 2004), wholegrain cereals (Slavin, 2004), probiotics (Rafter, 2004) and *n*-3 PUFA (Baracos *et al.* 2004). Three papers in this issue are similarly optimistic. Tripoli *et al.* (2005) discuss the polyphenols in olive oil. Like the polyphenols in wine (Cooper *et al.* 2004), those in olive oil are potent antioxidants with potential health benefits. The good news for gourmets is that these compounds are found in extra-virgin olive oil, to which they give the characteristic flavour, but are mainly removed during refining.

Ruxton *et al.* (2005) review the beneficial effects of *n*-3 PUFA. There is good evidence of their efficacy in the prevention of CVD, stimulation of immune function and alleviation of rheumatoid arthritis. They note that for other inflammatory conditions, psychiatric illness and cognitive impairment there is little evidence of benefit. Similarly, there is little evidence of beneficial effects for the fetus of long-chain *n*-3 PUFA supplementation during pregnancy (as opposed to the adverse effects of inadequate intake).

At one time hormone replacement therapy was seen as the key to improved quality of life and improved health for peri- and postmenopausal women. However, recent studies have shown an increased risk of breast cancer and CVD with long-term use of oestrogens. Hall *et al.* (2005) discuss the health benefits of isoflavones and other phyto-oestrogens in foods, and their effects on endothelial function. Perhaps predictably, they conclude that there is a need for further research, not only at a molecular level, but also in rigorous intervention studies to clarify equivocal findings and establish appropriate levels of intake.

If I were challenged to explain the 'low glycaemic index' flash on a food package in my local supermarket, I am confident that I could do so very succinctly; indeed, I do so for my first year students, and they seem to understand the concept. As with so many apparently simple concepts in nutrition, the reality of how you measure glycaemic index is more complex (see, for example, the discussion of the problems of determining the amino acid content of foods and amino acid availability by Moughan (2003)). Brouns *et al.* (2005) need twenty pages of this journal to discuss the problems in determining glycaemic index. They range from discussion of how many subjects need to be tested to achieve reliable results, through how many times each subject should be tested with each food, to, more importantly, asking what the reference carbohydrate should be. Many individuals find it unpleasant to drink a solution containing 50 g glucose (and if glucose solution is the reference carbohydrate, how rapidly or slowly should it be drunk?), so some investigators use boiled rice, or white bread (but should it be steam-baked white sliced bread or baguette? how fresh should it be?). How long should the subjects fast before the test? Can they be allowed unsweetened tea or coffee beforehand? Can they be allowed to smoke? (Caffeine and nicotine have an acute insulin antagonistic action.) One surprise to me was the note that it does not matter whether the subjects have impaired glucose tolerance, or are even frankly diabetic – but since the test compares an individual's glycaemic response to test foods and the reference carbohydrate, I should not have been surprised, since glycaemic index reflects intestinal handling of foods, and each subject is his/her own control.

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https://doi.org/10.1079/NRR2005105 Published online by Cambridge University Press