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Editorial

When the journal *Vitamins and Hormones* was first published, 62 years ago, the only justification for linking these two disparate groups of compounds together was that neither could be measured by the then available chemical techniques; they were normally present in extremely low concentrations and could only be measured by their biological activity, or, in the case of some vitamins, microbiological growth assays. Now, of course, they can be measured with exquisite precision by liquid chromatography and ligand-binding assays; those who have grown up with these sensitive and specific analytical techniques may wonder why vitamins and hormones are still considered together. Three papers in this issue show that the distinction between them is not as clear as was once thought.

Vitamin D was discovered as a result of studies of rickets, which can be prevented or cured by providing the vitamin, but is generally considered to be a hormone. (I recall being castigated some years ago by a colleague who ran an endocrinology module for medical students because I had included a question on vitamin D in the examination paper for the nutrition module!) Evidence has been accumulating for more than a decade that vitamin D has a wide range of activities unrelated to its classical role in Ca homeostasis and bone health, including regulation of the cell cycle, immune system function and insulin secretion. Flores (2005) discusses the growing evidence that it is also involved in insulin resistance and the chronic low-grade inflammation associated with type 2 diabetes, obesity and the metabolic syndrome.

There are few dietary sources of vitamin D, and in general endogenous synthesis as a result of sunlight exposure is more important than dietary intake. The current UK dietary reference value for vitamin D for the elderly (10 μg/d) is based on intakes that will maintain the same plasma concentration of 25-hydroxyvitamin D as is seen in younger people at the end of winter. This is an amount calculated to prevent deficiency rather than ensure optimum health, but even so it is almost certainly not achievable from food. Widespread advocacy of supplements, and enrichment of foods with vitamin D, is probably not desirable – although rickets was eradicated in Britain in the 1950s, as a result of widespread fortification of infant foods with vitamin D; the amounts were later reduced considerably, because of the sensitivity of a small number of infants to hypercalcaemia as a result of excessive intake. The alternative is to recommend increased sunlight exposure to increase vitamin D status (Holick, 2001; Gillie, 2004), a suggestion that leads to conflict with cancer biologists, who warn us to avoid excessive sunlight exposure because of the risks of skin cancer (Cancer Research UK, 2005).

The other two papers in this issue that blur the distinction between nutrition and endocrinology are both concerned with dietary phyto-oestrogens. These are not vitamins, since there is no evidence that they are dietary essentials, but they are (potentially) protective compounds in foods – so-called nutraceuticals. In the last issue, Hall *et al.* (2005) discussed the role of isoflavones in endothelial function. In this issue, Cassidy (2005) discusses the beneficial effects of isoflavones as an alternative to oestrogen replacement therapy to promote health in peri- and post-menopausal women, while Leung *et al.* (2005) discuss the protective effects of isoflavones with respect to breast cancer.

The fourth paper in this issue links the first three. Inflammation and fatty infiltration of the liver, not associated with excessive alcohol consumption (non-alcoholic steatohepatitis), occurs most commonly in patients with insulin resistance, and Shapiro & Bruck (2005) discuss the potential therapeutic use of curcumin, a polyphenol extracted from turmeric that has anti-inflammatory actions. In animal studies it shows liver-protective actions, suggesting that it may be beneficial in preventing the liver damage associated with insulin resistance and the metabolic syndrome.

McClenaghan (2005) also considers insulin resistance, type 2 diabetes, CVD and the metabolic syndrome, in terms of recommendations concerning carbohydrate intake. He notes that the epidemiological evidence suggests that relatively high-carbohydrate diets (but with a low glycaemic index) may be protective, and for weight loss, reducing the carbohydrate content of the diet is less important than reducing fat intake. He also discusses the problems of determining insulin resistance – like the determination of glycaemic index (Brouns *et al.* 2005), this is not as simple as we would like to think.

One approach to determine relationships between diet and health is to identify individual foods or nutrients that may confer protection – see, for example, the papers by Cooper *et al.* (2004) on wine polyphenols, and Tripoli *et al.* (2005) on polyphenols in olive oil, as well as the papers in this issue and the last on isoflavones (Cassidy, 2005; Hall *et al.* 2005; Leung *et al.* 2005). Michels & Schulze (2005) discuss the problems inherent in this type of research, and ask whether analysis of dietary patterns may provide more useful information. They discuss various ways of analysing the data from surveys, by identifying foods that are frequently eaten together, by grouping together people with similar diets or by identifying patterns of food consumption that explain as much as possible of the variation in biomarkers and other indices of health or disease.

Lindsay (2005) explores the concept of hormesis – the apparently beneficial effects of exposure to toxic agents at doses very considerably below the threshold of toxicity. We generally assume that if a compound is toxic then even minute amounts in foods may pose a hazard, but it is possible that this is not so, and chronic exposure to very small amounts may have beneficial effects in terms of

improving responses to various kinds of stress and metabolic insults.

There is an obvious role for the essay reviews that develop new hypotheses or question old ones that this journal normally publishes, but the final paper in this issue (Ishihara & Brayne, 2005) is a welcome innovation – a systematic review of nutritional risk factors in Parkinson's disease. The take-home message of this paper is that, although there are certainly gene–environment interactions in Parkinson's disease, the seven cohort studies and thirty-three case–control studies that the authors considered sufficiently rigorous to be included show no clear associations with any dietary factors. There is, however, evidence of a protective effect of both coffee drinking and alcohol consumption – some good news to offset the adverse effects of over-consumption of alcohol, and to justify the copious amounts of coffee the editor consumes.

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