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# Editorial

The diverse nature of the reviews included in this issue of *Nutrition Research Reviews* highlights a problem that is becoming increasingly acute for journal editors, namely that of finding suitable reviewers (especially for the more esoteric manuscripts) who are prepared to devote some of their time and expertise to this crucial task. Peer-review underpins the whole ethos of scientific journal publishing as we currently know it. Without it, readers have little guarantee that the published material is credible, which in turn casts the authors in a dubious light, and with them the editors and publishers of the journals; the whole publishing endeavour is then in danger of losing objectivity and hence value (both academic and financial). There is of course increasing pressure on researchers to publish (now even more acute in the current economic climate if they are to attract ever-scarcer funding) which has led to increasing article submission rates to the reputable journals, and an increased headache for their editors who find themselves sending out more and more requests to potential reviewers who seem less and less able to say 'yes'. The law of diminishing returns has definitely set in, but it hardly needs to be said that because the reviewers are themselves authors, they will inevitably suffer themselves from the lack of peer-review. Thus would-be authors are advised to contemplate the fact that if they do not feed into the system at both points, that is as authors and reviewers, the scope for publishing in a career-enhancing manner (i.e. in a reputable peer-reviewed journal) will ultimately dry up. Food for thought, perhaps.

The food industry can now manipulate the composition of spreading and cooking fats using enzymic interesterification in order to produce positional isomers of TAG with desired physical properties (melting point and so on) but without generating *trans*-fatty acids. All good, one might think; but the postprandial effects of these fats and their potential atherogenic effects have been little studied, warns Berry[1] in her review here. In contrast, the cardioprotective effects of *n*-3 long-chain PUFA are well known, while the precursor essential fatty acid, α-linolenic, has been somewhat overlooked once it became clear that it is not much converted to the *n*-3 long-chain PUFA in humans. However, the review by Hall[2] suggests that α-linolenic acid may have beneficial effects on blood pressure and vascular function, together with the *n*-6 essential fatty acid, linoleic, while, unsurprisingly, SFA are detrimental, potentially leading over time to endothelial damage and atherosclerosis. This author cautions that further research into the mechanisms by which these dietary fats exert their effects is needed before making dietary recommendations; similarly, taking Berry's point[1], it may also be necessary to consider the position on the TAG molecule of the fatty acids of interest.

Dietary fat composition is implicated in the modulation of blood lipid profiles (one of the major factors determining CVD risk), but research interest has also focused on other dietary factors that could play a role. One of these is garlic, which can be consumed as part of a Mediterranean-style diet, or as a supplement. Reinhart *et al.* [3] have conducted a meta-analysis of twenty-nine randomised, placebo-controlled trials reporting effects on blood lipids, and demonstrated a small but significant lowering effect on total cholesterol and TAG, but no significant effect on LDL or HDL. Small but useful, they conclude, and worthy of further investigation as adjunctive therapy alongside fibrinates or statins. Vitamins have also been implicated as being protective against CVD, in particular folate and the B vitamins, via their plasma homocysteine-lowering effect, as reported here by Sánchez-Moreno *et al.* [4] in the context of stroke. Whether this effect will track through to an actual reduction of stroke risk among patients with hyperhomocysteinaemia will not be clear until the conclusion of several ongoing intervention studies. Even less clear is the evidence supporting a protective role for the antioxidant vitamins E and C, for which results are conflicting, though vitamin E supplements may benefit high-risk individuals with high blood pressure or diabetes. The authors conclude that, while the evidence is not strong enough to recommend routine supplementation with B vitamins (or vitamin E or C) for the prevention of stroke, the diet containing these vitamins certainly can be recommended; after all, it was the high fruit and vegetable diet that highlighted the possible role of these vitamins in the first place.

Fruit and vegetables also contain many phytonutrients that may be beneficial in reducing the risk of other diseases, including osteoporosis; Trzeciakiewicz *et al.* [5] here investigate the complex mechanisms by which polyphenols might affect bone formation. The majority of work has been carried out with osteoblasts (the bone-forming cells), and shows that polyphenols can act at different stages (proliferation, differentiation, mineralisation), although the molecular mechanisms are far from being fully understood. Furthermore, polyphenols could also act on osteoclasts (bone-resorbing cells), perhaps affecting the balance between the activity of both cell types and, therefore, rates of bone formation (or loss).

Vitamin D is traditionally considered one of the major factors determining bone health, though its remit has expanded in recent years to include possible roles in many other diseases, which is of concern when taken alongside the growing realisation that poor vitamin D status could be widespread in the Northern countries; here, many of us don't eat much of the rather few dietary sources (fortified dairy products, oily fish, liver, eggs), and this is compounded by our poor exposure to sunlight in winter

(and summer, if we use sunblock). Teegarden & Donkin[6] describe how vitamin D could improve insulin sensitivity, and so prevent type 2 diabetes (which has been shown to be inversely associated with vitamin D status in epidemiological studies). Various mechanisms have been proposed, one involving the suppression of parathyroid hormone (which has recently been linked to insulin signalling at the adipocyte), another acting via an improvement in body composition in favour of muscle mass; vitamin D could also enhance insulin synthesis and release, or suppress inflammation. Any or all of these mechanisms could improve insulin sensitivity; however, conventional wisdom argues that the more difficult strategy of preventing obesity or perhaps achieving significant weight loss among the obese would be more effective in the prevention of diabetes.

This latter contention is challenged by Harrington *et al.*[7], whose meta-analysis, although it indicates a modest reduction in risk of all-cause mortality among adults who intentionally lost weight but were unhealthy (with diabetes or obesity-related health conditions), especially those who were obese, surprisingly showed a slightly increased risk of death associated with intentional weight loss among healthy adults who were merely overweight. The authors caution that the studies included all relied on reported body weight, which is obviously less accurate than actual measurement, especially since it is possible that this increased mortality was due to losses of lean body mass rather than fat. Thus, in order to clarify the picture, future studies are needed which measure body composition and fat distribution, as well as physical fitness, rather than simply body weight or BMI. These authors conclude with the somewhat heretical statement that, until better data are available, the concentration of public health initiatives upon the correction of obesity should perhaps be reconsidered. Prevention (preferably involving physical activity) is still, it seems, much, much better than the 'cure'.

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## References

1. Berry SEE (2009) Triacylglycerol structure and interesterification of palmitic and stearic acid-rich fats: an overview and implications for cardiovascular disease. *Nutr Res Rev* **22**, 3–17.
2. Hall WL (2009) Dietary saturated and unsaturated fats as determinants of blood pressure and vascular function. *Nutr Res Rev* **22**, 18–38.
3. Reinhart KM, Talati R, White CM, *et al*. (2009) The impact of garlic on lipid parameters: a systematic review and meta-analysis. *Nutr Res Rev* **22**, 39–48.
4. Sánchez-Moreno C, Jiménez-Escrig A & Martín A (2009) Stroke: roles of B vitamins, homocysteine and antioxidants. *Nutr Res Rev* **22**, 49–67.
5. Trzeciakiewicz A, Habauzit V & Horcajada M-N (2009) When nutrition interacts with osteoblast function: molecular mechanisms of polyphenols. *Nutr Res Rev* **22**, 68–81.
6. Teegarden D & Donkin SS (2009) Vitamin D: emerging new roles in insulin sensitivity. *Nutr Res Rev* **22**, 82–92.
7. Harrington M, Gibson S & Cottrell RC (2009) A review and meta-analysis of the effect of weight loss on all-cause mortality risk. *Nutr Res Rev* **22**, 93–108.

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