

2008

## Editorial: Nutrition Research Reviews

Katherine Younger

Technological University Dublin, [katherine.younger@tudublin.ie](mailto:katherine.younger@tudublin.ie)

Follow this and additional works at: <https://arrow.tudublin.ie/scschbioart>



Part of the [Dietetics and Clinical Nutrition Commons](#)

---

### Recommended Citation

Younger, K. (2008). Nutrition Research Reviews: Editorial. *Nutrition Research Reviews*, Access Volume 21, Issue 2, pp. 83-84. doi:10.1017/S0954422408182579

This Article is brought to you for free and open access by the School of Biological Sciences at ARROW@TU Dublin. It has been accepted for inclusion in Articles by an authorized administrator of ARROW@TU Dublin. For more information, please contact [arrow.admin@tudublin.ie](mailto:arrow.admin@tudublin.ie), [aisling.coyne@tudublin.ie](mailto:aisling.coyne@tudublin.ie).



This work is licensed under a [Creative Commons Attribution-Noncommercial-Share Alike 4.0 License](#)

## Editorial

We are all familiar with the dietary guideline exhorting us to eat so many portions of oily fish per week in order to boost our intakes of *n*-3 long-chain PUFA, but it is perhaps not so widely realised that the fish must themselves be provided with dietary *n*-3 long-chain PUFA or, possibly, their precursors (though, as in humans, the ability of carnivorous fish to elongate and desaturate *n*-3 PUFA appears to be limited). In the wild, fish obtain these fatty acids from marine plankton; however, as Miller *et al.*<sup>(1)</sup> here point out, the harvesting of wild fish to meet human requirements is unsustainable. Farmed fish represent a viable alternative, and Miller *et al.* discuss renewable sources of *n*-3 PUFA for use in aquaculture, including other marine creatures such as copepods, or single-cell organisms such as diatoms that could be grown in culture using biotechnological techniques, or, more futuristically, land plants genetically engineered to produce long-chain PUFA (i.e. 20 and 22C as distinct from the 18C precursors), which would, if successful (and acceptable to consumers), be the most sustainable and cheapest source.

Breast milk is the ultimate functional food, containing both the perfect formulation of nutrients required by the infant (even in the face of the mother's nutritional inadequacy) and a wide complement of protective components, these properties usually being considered in that order. McClellan *et al.*<sup>(2)</sup> here invite us to reprioritise these two major functions of breast milk, citing evidence in their fascinating comparative study of five contemporary mammalian species which suggests that the secretion that ultimately became mammalian milk during the course of evolution started out as a coating that protected the (shelled) offspring from desiccation and microbial attack, the nutritional role coming later. Strikingly, the nutrients themselves (unique to the mammary gland) appear to have evolved from existing protective and immune molecules, and are multifunctional, having antimicrobial and other beneficial effects in addition to their nutritive value (for example, in the case of lactose, its promotion of Ca absorption and prebiotic effects). Mammalian milk is itself arguably multifunctional, a range of ingredients being employed in different combinations and concentrations in different species in such a way as to facilitate widely varying litter sizes, rates of growth and development of offspring. Seemingly, the more that is discovered about this extraordinary secretion, the more clear it becomes that mammals should ideally be reared on their own mother's milk.

Additional to the above, breast-feeding is also known to help protect offspring against obesity, which is now understood to be associated with a state of low-grade inflammation. The review by Forsythe *et al.*<sup>(3)</sup> examines the relationship between this obesity-related inflammation and weight loss, concluding that according to the available evidence, this must be at least 10% in order to be of benefit. However, these authors argue that there is a need for

better-conducted, longer-duration studies, in which males and females are considered separately, the age range of the subjects is tightened, both actual and relative body weight changes are reported, and body fat and lean tissue are calculated relative to height (as fat mass index and fat-free mass index, respectively), this being far preferable to merely reporting surrogate measures of adiposity such as BMI or waist circumference, given that the inflammatory markers are associated with adiposity *per se*. Taking these two reviews together, it could be argued that future studies should also report on whether and for how long subjects were breast-fed.

Once weaned, children may have access to sugar-sweetened soft drinks, and controversy has long surrounded the question of whether or not these are responsible for obesity. Sigrid Gibson<sup>(4)</sup> takes a close look at the evidence from observational studies and interventions, and some will be disappointed to learn that she concludes that the evidence is inconclusive. Inadequacies in the design and conduct of many studies render them less than useful; for example, some don't distinguish between 'diet' and regular soft drinks, or fruit juice, when surveying intakes, and many have not considered activity (which might be expected to be associated with the rate of consumption of soft drinks). Even in well-conducted studies, it is difficult to compare results due to differences in study design, definitions used, statistical analysis and interpretation of results, though it is possible to deduce that the largest studies report the smallest effects. Ultimately, in this universe, it is energy balance that determines whether body energy is gained or lost, and soft drinks have a role to play in obesity if they constitute a significant source of energy, and if not, not. Furthermore, there is no good evidence to support the notion that the energy provided from soft drinks is more obesogenic (even though it might be less nutrient-dense) than energy from other sources.

Abdominal obesity is a feature of the metabolic syndrome, and the review by van Meijl *et al.*<sup>(5)</sup> examines whether this and other features of the syndrome could be prevented by consuming dairy products. Historically, these have been somewhat demonised due to their saturated fat content; hence the longstanding recommendation, endorsed by these authors, that reduced-fat versions are preferable. On the other hand the other components in dairy products could be responsible for beneficial effects such as improved blood-lipid profiles and reduced blood pressure in the case of Ca, while the satiating effects of protein improve weight loss, while preserving fat-free mass, also improve blood lipid profiles, and may reduce blood pressure via an effect on angiotensin-1-converting enzyme (ACE) activity (though this last effect has not been shown in humans). While the evidence in favour of dairy products in the prevention and treatment of the metabolic syndrome is not substantial, it

will be interesting to watch whether further research will prove them to be beneficial and, if so, which components are responsible.

The ability of dietary factors to reduce blood pressure and other CVD risk factors *in vivo* is also examined in relation to grape products by Pérez-Jiménez & Saura-Caulixo<sup>(6)</sup>, in both human and animal studies. Grape products contain polyphenols and many other compounds, including alcohol in the case of wine, and it is important to distinguish between the effects of these by, for example, using grape juice or dealcoholised wine in studies. Reassuringly for those of us who like a drink, alcohol has positive effects on blood pressure and blood lipids separate from those of the polyphenols, tannins etc, which are also anti-thrombotic, anti-atherosclerotic and, of course, antioxidant. Lack of consistency in reporting of the composition of the various preparations makes it difficult to compare across studies, but future work may prove grape products also to be anti-inflammatory and of benefit in the management of type 2 diabetes. Cheers!

Cancer is another main scourge of human populations, and we here present two reviews relating to the effects of diet in cancer prevention. Thompson *et al.*<sup>(7)</sup> investigate the role of *trans*-fatty acids, hitherto linked with adverse effects on blood lipids but also, perhaps, with cancers of the breast, colon/rectum and prostate, among others; while Vossenaar *et al.*<sup>(8)</sup> discuss how concordance with World Cancer Research Fund and American Institute of Cancer Research (WCRF/AICR) dietary and lifestyle recommendations can be evaluated. Both reviews grapple with problems associated with methodologies; in the case of *trans*-fatty acids, assessment of dietary intake is made difficult by the continuous reformulation of margarines and cooking fats by manufacturers, and the fact that it is not possible to calculate dietary intake from the proportions of *trans*-fatty acids measured in tissues; meaning that establishing a link between *trans* fatty acid intakes and cancer risk is not straightforward thus finding that a relationship with cancer risk is not straightforward. On the whole, the evidence for an association appears weak; the strongest association reported (from the Nurses' Health Study, for non-Hodgkin's lymphoma) relies on dietary intakes assessed in 1980, which clearly need to be verified by more up-to-date dietary intake measurements in this population. Very interesting is the association between *trans*-fatty acid intakes and increased risk of prostate cancer among those with a particular genotype (35% of the population), which these authors recommend should certainly be further investigated. They also anticipate that ongoing prospective studies in which dietary fat composition is being measured as accurately as possible will shed further light on the dietary fat and cancer story. Vossenaar and colleagues take up the vexed question of how one can evaluate whether and to what extent populations or individuals comply with dietary and lifestyle recommendations, in this case the ones set by the WCRF/AICR for cancer prevention. These authors argue that selection of appropriate evaluation criteria and

assessment methods is key in order to be able usefully to relate such compliance (or the lack of it) with health indices, to help in the identification of population-specific health education targets and to evaluate health interventions.

The study of the regulation of energy intake and expenditure over the last decade has identified a plethora of signals involved in stimulating, sensing and discontinuing nutrient intake, as well as monitoring the metabolic status of the body. This explosion of data is overwhelming, making it difficult for non-experts to see the overall picture. In this context, the review by Roche *et al.*<sup>(9)</sup> is very welcome; it lucidly describes the origins and actions of these signals, providing an integrated picture of how food and nutrient intake is regulated in single-stomached and ruminant animals. This review beautifully illustrates how research into two disparate areas, both of great importance to humans, that is, human obesity and the optimisation of ruminant animal production, can be of mutual benefit.

K. M. Younger

Editor-in-Chief

School of Biological Sciences  
Dublin Institute of Technology

Kevin Street

Dublin 8

Republic of Ireland

Katherine.Younger@dit.ie

## References

- 1 Miller MR, Nichols PD & Carter CG (2008) *n*-3 Oil sources for use in aquaculture – alternatives to the unsustainable harvest of wild fish. *Nutr Res Rev* **21**, 85–96.
- 2 McClellan HL, Miller SJ & Hartmann PE (2008) Evolution of lactation: nutrition *v.* protection with special reference to five mammalian species. *Nutr Res Rev* **21**, 97–116.
- 3 Forsythe LK, Wallace JMW & Livingstone MBE (2008) Obesity and inflammation: the effects of weight loss. *Nutr Res Rev* **21**, 117–133.
- 4 Gibson S (2008) Sugar-sweetened soft drinks and obesity: a systematic review of the evidence from observational studies and interventions. *Nutr Res Rev* **21**, 134–147.
- 5 van Meijl LEC, Vrolix R & Mensink RP (2008) Dairy product consumption and the metabolic syndrome. *Nutr Res Rev* **21**, 148–157.
- 6 Pérez-Jiménez J & Saura-Calixto F (2008) Grape products and cardiovascular disease risk factors. *Nutr Res Rev* **21**, 158–173.
- 7 Thompson AK, Shaw DI, Minihane AM, *et al.* (2008) *Trans*-fatty acids and cancer: the evidence reviewed. *Nutr Res Rev* **21**, 174–188.
- 8 Vossenaar M, Solomons NW, Valdéz-Ramos R, *et al.* (2008) Evaluating concordance with the 1997 World Cancer Research Fund/American Institute of Cancer Research cancer prevention guidelines: challenges for the research community. *Nutr Res Rev* **21**, 189–206.
- 9 Roche JR, Blache D, Kay JK, *et al.* (2008) Neuroendocrine and physiological regulation of intake with particular reference to domesticated ruminant animals. *Nutr Res Rev* **21**, 207–234.