Nutrition Discussion Forum

Intestinal absorption of β-carotene, lycopene and lutein in men and women following a standard meal

The study of intestinal absorption of β-carotene, lycopene and lutein following a standard meal by O'Neill & Thurnham (1998) was of considerable interest.

The authors comment on a large degree of variation in the absorption of triacylglycerol-rich lipoprotein carotene uptake. Looking at the data, specifically at the basal β-carotene plasma concentrations, and considering absorption rates in the light of those levels, since they may reflect β-carotene repletory 'status', it is possible to see what may be a relationship of absorption to that status, i.e. with the area under the curve for β-carotene and possibly for triacylglycerol secretion (correlation co-efficient for triacylglycerol *v.* basal β-carotene $-0{\cdot}51$; $P = 0{\cdot}056$, for example). It would be interesting to know whether the authors have considered the possibility of these relationships since the numbers in the report were rather small. One wonders if they may now have additional data and be able to comment further on the possibility of such a relationship?

Reference

O'Neill MA & Thurham DI (1998) Intestinal absorption of β-carotene, lycopene and lutein in men and women following a standard meal: response curves in the triacylglycerol-rich lipoprotein fraction. *British Journal of Nutrition* **79**, 149–159.

B. J. Boucher
Academic Medical Unit
The Royal London Hospital
Whitechapel
London E1 1BB
United Kingdom

Intestinal absorption of β-carotene, lycopene and lutein in men and women following a standard meal – reply by O'Neill & Thurnham

We thank Dr Boucher for her comments (Boucher, 1998) and agree that some of the data we reported in our paper (O'Neill & Thurnham, 1998) and other data not yet published do suggest an inverse relationship between the uptake of carotenoid from the diet (area under the curve) and the basal levels of plasma carotenoid at the time of study. In both the non-smokers (*n* 12) and smokers (*n* 11; M. E. O'Neill and D. I. Thurnham, unpublished results) there were inverse relationships for both β-carotene ($r - 0{\cdot}66$, $P = 0{\cdot}05$; $r - 0{\cdot}25$, $P = 0{\cdot}48$) and lycopene ($r - 0{\cdot}71$, $P = 0{\cdot}04$; $r - 0{\cdot}41$, $P = 0{\cdot}24$) respectively. However, for the third carotenoid examined, viz. lutein, positive relationships between absorption and plasma values were obtained for both non-smokers (r 0·5, $P = 0{\cdot}21$) and smokers (r 0·3, $P = 0{\cdot}41$). However, with the exception of β-carotene and lycopene in non-smokers, none of the relationships were significant and the results may be due to chance. We know of no feedback mechanism whereby circulating concentrations of carotenoids could influence carotenoid uptake from the gut during absorption.

References

Boucher BJ (1998) Intestinal absorption of β-carotene, lycopene and lutein in men and women following a standard meal (letter). *British Journal of Nutrition* **80**, 115.

O'Neill ME & Thurnham DI (1998) Intestinal absorption of β-carotene, lycopene and lutein in men and women following a standard meal: response curves in the triacylglycerol-rich lipoprotein fraction. *British Journal of Nutrition* **79**, 149–159.

Maura O'Neill
Omagh Health Centre
Mountjoy Road
Omagh
Co. Tyrone BT79 7BA
United Kingdom

David I. Thurnham
NI Centre for Diet & Health
School of Biomedical Sciences
University of Ulster
Coleraine, BT52 1SA
United Kingdom

Clarification of the link between polyunsaturated fatty acids and Helicobacter pylori*-associated duodenal ulcer disease: a dietary intervention study*

It would have been interesting if Duggan *et al.* (1997), in their report in your journal, had been able to take duodenal biopsies to compare the presence and extent of duodenal gastric metaplasia between the subjects on a high- and on a low-polyunsaturated-fat diet. It would be of interest also to know whether those subjects who had active duodenal ulcers showed any endoscopic improvement as a result of taking a high-polyunsaturated-fat diet.

Duodenal gastric metaplasia is always found in association with duodenal ulceration and is a necessary prerequisite for *Helicobacter pylori* colonization of the duodenum from the antrum. The geographical distribution of duodenal ulceration, with the presence of areas of high and low prevalence, has shown a strong relationship with staple diets (Tovey & Tunstall, 1975; Tovey, 1979, 1992, 1994; Tovey *et al.* 1989). In India our own studies and those of others showed a high prevalence of duodenal ulcer in all of the polished-rice-eating areas, especially in the South and in Bangladesh, and a low prevalence in the unrefined-wheat-eating areas of the North, in particular in the Punjab. The prevalence was also low in isolated areas where certain millets, certain pulses or ragi (*Eleusine coracana*) formed a large part of the staple diet.

Our experiments on animal peptic ulcer models show that the North Indian Punjabi diet is protective against ulceration, whereas the South Indian diet is ulcerogenic (Jayaraj *et al.* 1980, 1986, 1987). This protective activity is present in certain foodstuffs such as unrefined wheat, soya, ragi and some millets and lentils. The lentil Horse Gram (*Dolichos biflorus*) is markedly protective. This protective activity has been shown to lie in the lipid fraction of these foodstuffs. This fraction contains essential fatty acids such as linoleic acid, phospholipids and phytosterols, all of which have been shown experimentally to enhance mucosal cytoprotection (Tarnawski *et al.* 1987; Lichtenberger *et al.* 1990; Romero & Lichtenberger, 1990).

From available evidence, *Helicobacter* infection of the antrum seems to be equally prevalent in the high- and low-duodenal-ulcer areas of India and is not a factor in explaining the differences in prevalence. It is possible that the presence of protective factors in diet may explain these differences. The protective factors may enhance the resistance of the duodenal mucosa, enabling it to withstand acid and pepsin, and consequently the development of gastric metaplasia. To account for changes in duodenal ulcer prevalence it may be that we ought to be looking for the effect of diet on duodenal gastric metaplasia and not at any effect on the extent of *Helicobacter pylori* infection.

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Frank I. Tovey
Michael Hobsley

Department of Surgery
University College London
London W1P 7LD
United Kingdom

Clarification of the link between polyunsaturated fatty acids and Helicobacter pylori*-associated duodenal ulcer disease: a dietary intervention study – reply by Duggan & Spiller*

We agree that the epidemiological data linking diet and duodenal ulcer disease quoted by Tovey & Hobsley (1998) have yet to be made clear. Our experimental studies show that very substantial amounts of PUFA in the diet do not significantly alter *Helicobacter pylori* infection in the antrum. As Tovey and Hobsley point out, this still leaves a possibility that these diets are protective because of their impact on duodenal metaplasia which we did not examine.

Reference

Tovey FI & Hobsley M (1998) Clarification of the link between polyunsaturated fatty acids and *Helicobacter pylori*-associated duodenal ulcer disease: a dietary intervention study (letter). *British Journal of Nutrition* **80**, 116.

Anne Duggan
Robin Spiller
Division of Gastroenterology
University Hospital
Nottingham NG7 2UH
United Kingdom