UNSATURATED FATTY ACIDS IN NEW ZEALAND MARGARINES - AN APPROPRIATE COMBINATION?

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Epidemiological evidence from the large US Nurses Study (Willett et al. 1993) indicated a correlation between high dietary intake of *trans* fatty acids in 1980 and subsequent coronary heart disease risk. Case control studies have also shown a higher *trans* fatty acid content in adipose tissue (reflecting dietary intake) in people dying of CHD (Thomas and Winter 1987) and of dietary *trans* fatty acids in cases of first myocardial infarction (Ascherio et al. 1994). The mechanisms which might underly any such association are unclear, but *trans* fatty acids have been shown to increase LDL-cholesterol, reduce HDL-cholesterol (Mensink & Katan I 1990) and to increase Lp(a) (Nestel et al 1992a). All these effects would be expected to be potentially atherogenic. Other possible effects may result from the competitive interactions of *trans* fatty acids in essential fatty acid (EFA) metabolism and eicosanoid production. The latter have mainly been studied in animals but the detrimental effects are porported to result from the creation of a relative deficiency of EFA. This occurs by inhibition of the conversion of linoleic acid (cis C18:2) to arachidonic acid, and may be more relevant if the linoleic acid supply is low. Concentrations of linoleic acid in adipose tissue triglycerides (an indicator of long term intake) are often low in 'western' populations. Low linoleic acid intake associated with a moderate intake of *trans* fatty acid might thus be deleterious.

The main dietary sources of *trans* fatty acids are hydrogenated oils and margarines. The hard margarines used in the US at the time of the Nurses Study comprised up to 35% - 40% *trans* fatty acids and relatively little linoleic acid.

The current study investigated the fatty acid composition of NZ margarines. The percentage of the total fatty acids as both *cis* and *trans* C18:1 and C18:2 was determined by lipid extraction and gas chromatography using a BPX 70 polar capillary column. The *trans* C18:1 and C18:2 ranged from 5.4 to 11.2 and 19 to 41.7% respectively, with only tiny amounts of *trans* C18:2. The ratio of *trans* C18:1 to *cis* C18:2 was 1:5 to 1:2.4 for margarines, except for the oleic- acid-rich spread (1:2.5), and 1:5 to 1:2.4 for fat-reduced margarines/blends. By contrast, the ratio for butter was 3:1.

Most margarines available on supermarket shelves in New Zealand contain quite modest amounts of *trans* fatty acids, little saturated fatty acids and high amounts of linoleic or oleic acid. Nestel et al. (1992b) showed a manufactured fat with a high linoleic acid content reduced the hypercholesterolaemic effects of the *trans* C18:1 fatty acids. Thus it may be expected that the 30-40% fatty acids as linoleic acid in most NZ margarines could minimise or counteract any adverse effect of the *trans* fatty acids on lipoprotein levels. Further work is however needed to assess this and any effects on platelets and/or eicosanoid production, and to document the total amount of *trans* fatty acids in the New Zealand diet by obtaining accurate analysis on a wide range of other foods.

WILLETT, W. et al. (1993). Lancet 341: 581.
MENSINK, R. and KATAN, M. (1990). N. Eng. J. Med. 323: 349.
THOMAS, L. and WINTER, T. (1987). Hum. Nutr. Clin. Nutr.41F: 153.
ASCHERIO, A. et al. (1994). Circulation 89: 94.
NESTEL, P., NOAKES, M. and BELLING, B. (1992a). J. Lipid Res. 22: 1029.
NESTEL, P., NOAKES, M., BELLING, G., McARTHUR. R., CLIFTON. P. and ABBEY.M. (1992b). Am. J. Clin. Nutr. 55: 46.

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