Letters to the Editors

The influence of mild cold on human energy expenditure: is there a sex difference in the response?

We read with great interest the account by Warwick & Busby (1990) of their finding that the metabolic response to mild cold is not inhibited by voluntary increases in clothing insulation. Mild-cold-induced thermogenesis has been demonstrated in healthy female subjects whose clothing has been standardized (Dauncey, 1981; Lean & Murgatroyd, 1987). The corresponding results for men are, however, unconvincing, the conclusions of De Boer *et al.* (1988) being drawn from the comparison, on a weight-normalized basis, of two separate groups of men measured some years apart.

We have for some time suspected that the thermogenic response to mild cold in male subjects may not be so pronounced as in females, and have some unpublished data to support this conclusion.

We were pleased to find that Warwick & Busby (1990) had presented their data for individual subjects, giving us the opportunity to re-analyse their data for the men and women separately. By paired *t*-test we conclude that the women in the data of Warwick & Busby (1990) showed a significant response to mild cold overnight and over 24 h but that the men showed no significant response. Furthermore, a *t*-test comparison of the responses of the men and women showed that they were significantly different.

We feel that there may well be an indication of sex differences in the response to mild cold and that this merits further investigation in a rather larger group than that of Warwick & Busby (1990) and with standardization of clothing.

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Dietary trans *fatty acids and reproduction*

The observation of Hanis *et al.* (1989) of reduced growth and reproductive performance of rats fed on diets containing butter (BF) and hydrogenated vegetable oil (HO) may confirm earlier observations of the antagonistic effects of *trans* fatty acids, especially *trans,trans*-18:2, under conditions of essential fatty acid (EFA) deficiency. However, their claim to have demonstrated adverse effects of *trans*-fatty acids on reproductive performance of rats given diets not deficient in EFA does not stand up to scrutiny.

Their diets contained only 50 g fat/kg. Diets with BF and HO as the only source of fat had 2·90 and 4·13% w/w of total fatty acids as linoleic acid (*cis,cis*-18:2) equivalent to 0·38 and 0·54 energy % (en%) respectively. The authors state that the linoleic acid supply was

at the lower margin of rat requirement as determined by Pudelkewicz *et al.* (1968). The latter determined the minimum requirement of growing female rats to reduce the triene:tetraene ratio to 0·4 as 0·5 en% but the corresponding value for young male rats was 1·3 en%. These values were determined with fat-free diets supplemented with linoleic acid. Under these conditions there is no competition of other fatty acids for Δ6 desaturase activity. Addition of only 50 g fat rich in oleic acid/kg diet enhances the need for linoleic acid and requirement is generally held to be in the range of 1–2 en% from linoleic acid. Requirements for pregnancy are not known, but Menon *et al.* (1981) reared female rats for 4 months on a diet with 50 g fat/kg containing 3·5% linoleic acid (0·43 en%) until mating, and then transferred them to a diet with 50 g fat/kg containing 2.0% linoleic acid (0·25 en%) for 21 d of pregnancy. Both dams and fetuses were markedly EFA-deficient, more so than those of Hanis *et al.* (1989). By comparison the probability must be that the diets of Hanis *et al.* were suboptimal in supply of *cis,cis*-18:2, and supporting evidence is seen in the triene:tetraene ratios of adipose tissue of 5·3 and 13·3 for BF and HO respectively. The greater effect of HO may be due to the greater dietary intakes of *cis*-9-18:1 (45·2% of fatty acids *v.* 23·2%), *trans*-18:1 (19·5 *v.* 3·1%), 18:1 (*cis* plus *trans* 64·7 *v.* 26·2%) or *trans,trans*-18:2. The contents of *trans,trans*-18:2 at 0·98 and 3·19% w/w of fatty acids in diets BF and HO respectively are also surprisingly high. Commercial hydrogenated vegetable oils normally contain less than 1%, while major brands are quality controlled to contain less than 0·5% *trans,trans*-18:2. A dietary situation in which the supply of *trans,trans*-18:2 is 77% of a limiting level of *cis,cis*-18:2 is of little relevance to practical nutrition.

The presence of 2 en% *cis,cis*-18:2 has been shown to be sufficient to prevent undesirable effects of high amounts of dietary C_{18} *trans* fatty acids on mitochondrial function in rats (Zevenbergen *et al.* 1988). The study of Hanis *et al.* (1989) should be repeated with diets containing at least 2 en% *cis,cis*-18:2 and with controls with a comparable amount of *cis*-18:1 as *trans*-18:1 before any conclusion can be made that hydrogenated fat adversely affects reproduction in a situation where the diet is not EFA-deficient. The danger of misinterpretation of their findings is illustrated by the unqualified statement, citing this work, in a review article by Chong (1990) that *trans* fatty acids adversely affect the reproductive performance of animals.

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Reply from Hanis

We based our conclusions (Hanis *et al.* 1989) on the observation that no overt signs of essential fatty acid (EFA) deficiency were observed in Wistar rats fed on the butterfat diet containing 0·38 energy % (en%) of linoleic acid (*cis,cis*-18:2), and that the hydrogenated vegetable oil diet contained 0·54 en% of linoleic acid. Pudelkewicz *et al.* (1968) found the minimum requirement for linoleic acid to be 0·4 en% in female and 1·3 en% in male

Sprague–Dawley rats. It is also supposed that the requirement for linoleic acid is higher in the presence of high concentrations of other fatty acids, i.e. *cis*-18:1 and its isomers, and isomers of 18:2. Our observations seem to be in line with those of Zevenbergen *et al.* (1988), who did not find any signs of EFA deficiency in Wistar rats fed on diets containing only 0·4 en% as linoleic acid, even in the presence of high concentrations of *trans* fatty acid isomers (20 en%). That is why we tend to describe our diets as marginal in linoleic acid content rather than deficient. The signs of EFA deficiency occurring in rats on the hydrogenated vegetable oil diet could have been caused by high concentrations of isomeric fatty acids, but we could not exclude the effects of the presence of a high concentration of *cis*-18:1. Future experiments should be designed to answer the question: 'What is the minimal requirement of linoleic acid in the presence of different levels of isomeric acids and *cis*-18:1?', rather than to investigate the effects of isomeric fatty acids in diets containing optimal concentrations of linoleic acid. There are no indications that in the case of optimal supplementation with EFA there should be any adverse effects of isomeric fatty acids on animal physiology.

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