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alternatives from certain foods, the most suitable of which are spreads and cooking or salad oils. This is not a surreptitious attempt to drive up PUFA intakes by recommending the exclusive use of high-PUFA-containing products. The practical problem, however, is to maintain PUFA levels. To do this by the increased consumption of fish, poultry, cereals, and vegetables alone would require more than a doubling of consumption of these foods. Most of these foods are bulky and have a low fat content, even if it is one rich in PUFAs.

Many dietary recommendations include a slight increase in dietary PUFA above the current level. Marr and Morris are right to emphasise the "modest" extent of their 7 g per day increase to no more than 6·7% of total energy. This increase, it is suggested, could come from half an ounce (14 g) of margarine and a smaller amount of PUFA rich vegetable oil per day, which is well within the existing range of distribution of consumption of such foods.

A diet with 30% total energy from fat with no more than 10% from saturated fatty acids has been advocated widely for nearly twenty years. It is based on acceptable diets and not a theoretical optimum for blood lipids. Marr and Morris's rejection of this diet on grounds of unacceptability is a subjective judgment which is difficult to support. While their faith in the likelihood of action by Government and industry is encouraging, it is important to recognise that, in the past, industry has tended to defend existing market patterns, and we have already witnessed the opposition from the agriculture and food industries to any suggestion of even a slow adjustment to the fat content of liquid milk available in the U.K. Any recommendation for reducing fat consumption will be resisted, especially with the strong fat producing interest in the U.K. agricultural and food industries.

The dietary recommendations are widely supported and the health professions in the U.K. should seek their implementation. Industry should be encouraged to identify and exploit the many food-marketing opportunities which are in line with the dietary goals—for example, low fat dairy and meat products, fruit and vegetables, cereals. A healthy diet for Britain and a secure food industry are not incompatible goals.

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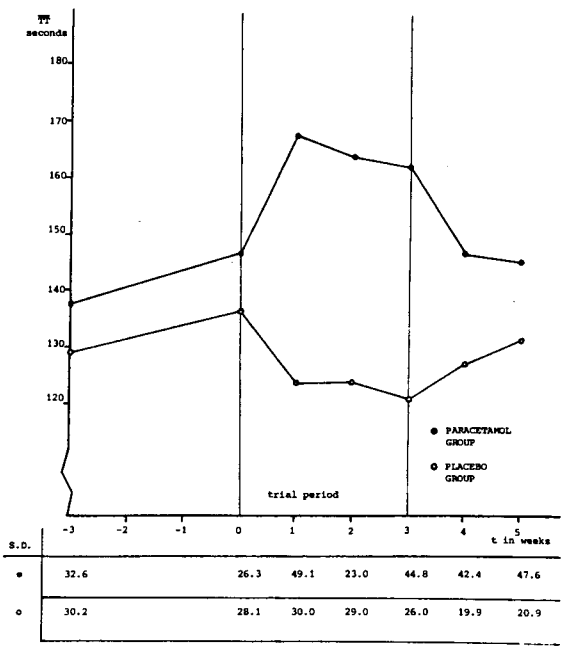
INTERACTION BETWEEN PARACETAMOL AND COUMARIN ANTICOAGULANTS

SIR,—Investigations of the effects of paracetamol (acetaminophen) on anticoagulation with coumarin derivatives have yielded differing results. Antlitz et al.[1] studied fifty patients on long-term medication with warfarin, dicoumarol, and phenprocoumon who took 2600 mg paracetamol daily for 2 weeks. The PTT (prothrombin time) rose slightly but significantly compared with values in a placebo group. By contrast, Udall[2] showed that PTT values did not change in ten patients on warfarin medication who took 3250 mg paracetamol daily for 14 days. Antlitz and Awalt[3] demonstrated that two doses of 650 mg given for one day did not influence the PTT. Because of these inconclusive results and because of the enormous consumption of paracetamol with and without prescription we mounted a double-blind investigation.

Twenty volunteer patients on coumarin therapy were selected from patients controlled by the Amsterdam Thrombosis Service. They were allocated at random to paracetamol (ten patients) or placebo (ten patients). The patients took four tablets daily at 0900, 1300, 1800, and 2300 hours containing 500 mg paracetamol or placebo for 3 weeks. Their 'Thrombotest' times[4,5] and anticoagulant dosages were recorded once a week for 3 weeks before the trial, during the trial, and for 2 weeks afterwards. Coumarin medication was reduced for 2 days in patients whose thrombotest values exceeded 200 s.



S.D.							
●	32.6		26.3	49.1	23.0	44.8	42.4	47.6
○	30.2		28.1	30.0	29.0	26.0	19.9	20.9

Effect of paracetamol and placebo on thrombotest times (TT).

Standard deviations shown below figure.

Patients taking paracetamol showed a significant ($p<0·05$, t test) increase in thrombotest times (figure). Dosage of anticoagulant had to be reduced in five patients in the paracetamol group and in one of the controls.

The mechanism underlying this interaction is not clear, but interference with hepatic synthesis of factors II, VII, IX, and X is possible. This study shows that high daily doses of paracetamol significantly prolong thrombotest times in patients on coumarin therapy.

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MODE OF ACTION OF SULPHASALAZINE: AN ALTERNATIVE VIEW

SIR,—Recent *Lancet* correspondence[1] has suggested that sulphasalazine (SPZ) is effective in ulcerative colitis by altering prostaglandin turnover. However, whilst prostaglandins can mimic several facets of the inflammatory response it seems unlikely that cyclo-oxygenase products of arachidonic acid metabolism mediate all aspects of the inflammatory processes in ulcerative colitis. Potent inhibitors of PG synthesis have not been effective as therapeutic agents in this disease.[2] Does SPZ affect other aspects of the inflammatory response? In ulcerative colitis, as in many forms of chronic inflammation, there is lymphocytic involvement, with persistent infiltration of the colonic mucosa by lymphocytes, of which a large proportion are in an activated phase.[3] Drugs used in

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