Renal failure after omeprazole

Srtr.—An 86-year-old woman had a 10 year history of oesophagitis and had been treated intermittently with cimetidine or ranitidine. Because of progressive symptoms in 1989, omeprazole 40 mg daily was started. At that time the patient had normal serum creatinine concentration. 2 months later she was admitted with renal failure: maximum serum creatinine 858 pmol/L (normal <120). A renal needle biopsy revealed interstitial inflammation, with plasma cells, lymphocytes, and eosinophils, and patchy tubulitis but no effect on glomeruli, which is characteristic of acute interstitial nephritis. 2 weeks before admission the patient had been treated with erythromycin for suspected pneumonia. Both drugs were withdrawn, and the patient regained normal renal function on treatment with diuretics. Erythromycin was suspected of being the drug causing the renal failure.

In 1992, during cimetidine treatment, the patient had a peptic stricture of the oesophagus and omeprazole was given with surveillance of renal function. Within a week the patient developed high temperature, a rash, eosinophilia, and diminishing renal function. The patient did not receive any other drugs, and omeprazole was withdrawn after 9 days. The renal failure progressed to anuria, and necessitated haemodialysis for a week. The renal function remained severely affected, and after 3 months serum creatinine had declined from 810 to 396 mmol/L.

Our patient had typical acute interstitial nephritis, with the triad of high temperature, rash, and eosinophilia.1 The pathophysiology remains unclear, but cell-mediated immunity is probably important in most cases.2 The allergic nature of our patient’s disease is favoured by the fact that she had previously reacted with rashes to other drugs (amlodipine with hydrochlorothiazide, phenytoin, and penicillin), and in 1972 she had had sarcoidosis (verified by renal biopsy specimen was investigated by H. Starklint.

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Adverse events after triazolam substitution

Srtr.—It is just over a year since the UK licensing authorities suspended the hypnotic triazolam. To examine the effects of this regulatory decision on patients in general practice, since in 1989, we retrospectively surveyed general practitioners who had switched from triazolam to other hypnotics because of adverse reactions. We used the routine computer records of 163 general practitioners to find patients who had, within the first three months of substitute therapy, a history of high temperature, rash, and eosinophilia.1 The pathophysiology remains unclear, but cell-mediated immunity is probably important in most cases.2 The allergic nature of our patient’s disease is favoured by the fact that she had previously reacted with rashes to other drugs (amlodipine with hydrochlorothiazide, phenytoin, and penicillin), and in 1972 she had had sarcoidosis (verified by renal biopsy specimen was investigated by H. Starklint.

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Pathophysiology of obesity

Srtr.—Dr Bradley (Oct 3, p 848) reiterates the once-popular notion that human obesity is caused by excessive consumption of sugar-containing and therefore palatable foods. His argument is based on the faulty premise that obesity was uncommon in traditional societies whose "bland and monotonous" diets provided an average of 50% of their energy from fat. On the contrary, most pre-industrial diets provided a very small portion of energy from fat. Subsistence-level farming chiefly involves tubers and starchy roots.1 Field agriculture has allowed the cultivation of grasses such as maize, oats, barley, and sorghum, including such major food grains as rice and wheat.1 Geographic location also plays a part. Thus manioc is still the basic foodstuff of African countries, maize is the staple food of Central and South America, whereas the Asian diet is based on rice. Most so-called traditional diets are very high in complex carbohydrates, but very low in fats and oils. Until recently, the Japanese diet provided less than 10% of daily energy from fat.2 The few contrary examples of the cattle-rearing Masa of East Africa or the hunting and fishing Arctic Eskimos have long been known to anthropologists and nutritionists. Although these groups incorporate meat and fish into their diets, and so run counter to the norm, their fat intake is surprisingly low compared with that of many western societies.3 Although animal fats were the most sought-after item in pre-industrial diets, their consumption was rare, and was often associated with feasts and ritual sharing.1 It was economic prosperity that brought about increased meat consumption and the selective breeding of animals for the maximum fat content. The French sociologist Claude Fischler2 notes that historians have long index the prosperity of an era, or the membership in a given social class, in terms of per caput meat consumption. Prosperity is...
associated with increased consumption of animal fats and a decline in the consumption of grain products.

Bradley mistakenly equates palatability exclusively with the sweet taste of sugar. In reality, fats have a more decisive role in determining the palatability of the diet.6,7 Fats endow foods with numerous flavors, aromas, and textures, and some of the most palatable foods in the human diet are those that are rich in fat.8,9 Preferences for dietary sources of fat may in fact be a shared feature of human obesity syndromes. Whereas obese men typically listed meat dishes among their favourite foods, women were more likely to express preferences for sweet, fat-rich desserts.10,11 Prosperity is, admittance, also associated with increased sugar consumption, because sucrose and fructose replace dietary starches and grains.12 However, although the typical American diet provides about 11% of energy from added sugars, 38% of the energy comes from fat, and, according to present medical consensus, fat is the most important dietary factor in the aetiology of obesity.

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Genetic susceptibility to multiple sclerosis linked to myelin basic protein gene

SIR,—Dr Tienari and colleagues' report (Oct 24, p 987) implicates the myelin basic protein (MBP) gene or an associated gene in multiple sclerosis in high risk Finnish families. However, although their criteria for multiple sclerosis are clear, those for optic neuritis are not defined. Were these criteria clinical or based on laboratory tests, or both? In the three pedigrees shown there were 82 individuals of whom 53 had optic neuritis or multiple sclerosis. Yet there were 20 forebears in these pedigrees without any demyelinating disease. Surely this is remarkable for a genetic condition? It may be that in some, particularly pedigree 2 in which there were 20 forebears in these pedigrees without any abnormal laboratory findings.

Colover questions the cause of demyelination in multiple sclerosis, which remains unknown and is probably multifactorial and therefore open to speculation. We regarded the following points as relevant to our report. First, MBP as a target antigen in experimental allergic encephalomyelitis produced by bovine protelipid apoprotein: immunological studies in rabbit. Ann Neurol 1983; 13: 305-08.


Authors' reply

SIR,—Dr Colover's comment about the high frequency of optic neuritis in multiple sclerosis families was based on a mistake, which had escaped our notice. Unfortunately, the symbol for optic neuritis in fig 2 was wrongly assigned in the legend of the figure. The correct symbol for males with optic neuritis should be a half-filled square instead of an open square, which indicated unaffected males. The number of subjects with this condition in the pedigrees shown in fig 2 is therefore 16 instead of 18, including 11 men and 5 women. The risk for optic neuritis were both clinical and laboratory investigations: the patients had had one attack of monosymptomatic disease without any abnormal laboratory findings.

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