Preference is given to letters commenting on contributions published recently in the *JRSM*. They should not exceed 300 words and should be typed double spaced

Coronary heart disease in Africa

In their article about coronary heart disease (CHD) in Africa (January 1997 *JRSM*, pp 23–7) Dr Walker and Dr Sareli indicate that the fat intake of Africans (57 g in men and 42 g in women contributing 23.8% and 26.1% of total energy intake) is substantially lower than that of Western populations (typically more than 100 g fat, contributing about 35% of calories). These figures are similar to observations in Indian patients with CHD that revealed 50 g fat ingestion in patients and 50 g in healthy controls (24.3% of total calories in patients and 24% in controls)[1]. It is perplexing that a high and rising incidence of CHD that exceeds the rate in Western populations (13.9% in individuals between the ages of 25 to 65, that is *26.4% higher* than the British) was observed in Indians even at low levels of fat ingestion[2]. Exact reasons for this phenomenon are unknown. Possibilities are insulin resistance and accelerated atherosclerosis, abdominal obesity (increased waist to hip ratio) and genetic susceptibility including angiotensin converting enzyme gene mutations, antioxidant status and stress associated with lifestyle changes[3,4]. Despite the low fat intake, Africa is likely to move in the same direction as exemplified by the high rates of CHD in Afro-Americans and the high prevalence of risk factors in native Africans. This scenario is quite akin to the observed increase in CHD in Indians, temporally associated with progressive adoption of Western lifestyle by a predominantly rural farming populace.

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Foodborne triggers for epileptic attacks?

I wonder whether others have noticed a connection between pesticides in food and triggering of epileptic attacks. My son, born December 1957, was well until 9 years of age when he developed *petit mal* attacks, followed a few months later by *grand mal* convulsions. Over subsequent years he was seen by several neurologists and had electroencephalograms and brain scans by computed axial tomography and nuclear magnetic resonance, but no localizing cause could be found. He was treated at various times with phenobarbitone, phenytoin, sodium valproate, carbamazepine, lamotrogine, and vigabatrin. Doses were scrupulously regulated with checks on blood levels where appropriate. Nevertheless, complete control of fits could not be established and as a result he suffered many falls and fractured the mandible on three occasions. In September 1994, he had a partial cerebral callosotomy which greatly reduced the number of falls, but did not stop the other epileptic attacks.

By 1982 we had noted an effect of diet, but the precise cause was difficult to establish because the fits occurred some 18–24 h after the offending food intake. Wine, oranges, lemons, apples and most other fruits were epileptic triggers, and we suspected that organic acids, in combination with metals, might be responsible[1]—perhaps by stimulating absorption of aluminium and other metals (as is known to occur). Cutting out these foods helped but did not solve the problem, and so we had to look further afield. Next, we suspected that bread was also a culprit, and found that switching to bread made with organic flour was a further help. Therefore we also changed to organic breakfast cereal, organic vegetables whenever possible, and organic meat.

We began to suspect that pesticides might be responsible, and this has been strongly supported by three further pieces of evidence. First, he developed fits while on holiday in Spain for no obvious reason until we realized that the active ingredient of the anti-mosquito machine that he was using in the bedroom was a pesticide vapourized by gentle heat. When this was removed the fits stopped. Second, we noticed that farmed salmon was a cause of fits, whereas wild salmon was not. Farmed salmon have to be treated with high doses of pesticides to prevent infection by water lice. Third, about 18 months ago we were forced into considering whether the drinking water supply might contain an epileptic trigger. An analytical report from our water company showed that the water contained some three times the permitted levels of a number of pesticides and herbicides because of lack of equipment to remove them. We therefore installed a domestic reverse-osmosis system for drinking and cooking water. The water company analysed the exit water from this system and found all herbicides and pesticides to be undetectable. This seems to have been the final step in removing the triggers from the diet. There was an immediate reduction of the fits and improvement in the general mental state. In the last year or so we have been able very gradually to reduce the dose of vigabatrin, with benefit. Despite this reduction, the lessening of fits has been maintained. He still has them occasionally, but only if he has eaten the wrong food (in restaurants etc) or if he has forgotten to take the medicaments.

The idea that food can trigger epilepsy is not a new one, having first been proposed in 1904[2]. 46 of 122 epileptics[3] showed skin sensitivity to various food proteins and some of these responded to restriction of foods concerned. A study[4] of 100 consecutive epileptics did not confirm the skin allergy tests but did show that certain foods induced fits in some patients. Cheese was found to be a cause of fits in one epileptic[5]. However, these allergy-induced fits occurred quite quickly after the particular food intake, whereas in the case described here there was a considerable delay. Moreover, the previous data preceded the use of pesticides, so that the mechanism may well be different. Pesticide poisoning in man can have a delayed effect in slowing nerve conduction velocity and reducing vibrotactile sensitivity[6]. In rats, lindane and endosultran lower the threshold of convulsions[7]; and, in man, application of lindane to the skin can cause convulsions[8,9]. It would therefore not be surprising if they had effects in some individuals, even in low doses.

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