Food safety awareness

Sir,—Dr J M Cowden and colleagues conclude that there is a need for public health education to help reduce food poisoning caused by *Salmonella enteritidis* phage type 4.1 Given the continuing media awareness and criticisms of the Department of Health for giving conflicting or delayed advice, it seemed interesting to survey the recipes in women’s magazines. As 15% of the patients reported that their illnesses were children aged under 10 years the magazines chosen were those directed at housewives and mothers.

Twenty three magazines in the women’s section and another five in the style section were sampled. None of the recipes or other stocked newspaper, all of which had a cookery section. Four magazines contained at least one recipe using raw eggs that would not be cooked before being eaten. These were *Family Circle* (October 1989; two recipes), *Bella* (30 September 1989; one recipe), and *Best* (September 1989; one recipe). The number of raw eggs (yolk or white, or both) for each recipe was 3, 3, 4, 2, 6, 2, and 9.

The comments on the recipes in *Family Circle* show particular lack of awareness of the food hygiene message: one was for ice cream (a favourite of children) and one had been prepared specifically for “my tot.” These recipes had been published in 1983, and 1988. Others were reported without riders in the light of new recommendations.

Another that used nine eggs discussed the “recent controversy over egg production in the UK” but said that the reader “should not worry if you buy from a respectable source—like a local supermarket.” At present there is in fact no way of being absolutely certain that eggs from any source are entirely free of salmonella. The promotion of eggs to date being awareness of the problems of food safety to the public.

Perhaps cookery writers and editors should be urged to double check before printing recipes such as these.

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Drug Points

Lofepramine and motor neuropathy

Dr J A Hewitt (Psychogeriatric Department, Barnucose Hospital, Redruth TR15 3TR) and Mr JOHN GILN (Pharmacy Department, Royal Cornwall Hospital, Truro TR1 3LJ) write: An 84 year old woman was admitted with an 18 month history of lethargy and intermittent agitation. Despite a good appetite she had lost 6 kg during this period. Her medical history was unremarkable but her family a strong family history of depressive illness. The only medication she had taken during this time was a few tablets of thioridazine 10 mg. On examination she had a cachetic appearance, pronounced psychomotor retardation, nihilistic and injection and global disorientation. Physical examination showed no other abnormalities. Investigations including full blood count, erythrocyte sedimentation rate, urea and electrolyte concentrations, and liver and thyroid function tests were all normal. The results did display chest radiography and electrocardiography.

Depressive illness was diagnosed and the patient started on lofepramine 70 mg twice daily. After nine days without response a further increase of 10 mg was given every day until a total of 700 mg three times a day. After one month’s treatment she had developed a high stepping gait. Examination showed an inability to dorsiflex her feet. Reflexes were present, and sensory functions, except for sensation, were normal. Investigations were repeated but no abnormalities found. Lofepramine was suspected of being the cause and was stopped. While her mental state did not change, eight weeks after stopping treatment her foot drop improved such that her gait was no longer high stepping.

An on line search failed to identify any reported cases of motor neuropathy with lofepramine. Other tricyclic antidepressants, however, notably imipramine and amitriptyline (the latter being metabolised through desipramine in the same way...
as lofepramine), have caused neuropathies of a predominantly motor type.1,2 Casarino has described a case of reversible motor neuropathy, manifested as bilateral footdrop, after three weeks of amitriptyline therapy.3 The manufacturers of lofepramine (E Merck) are not aware of any cases of motor neuropathy with the drug. This case has been reported to the Committee on Safety of Medicines, which, so far, has received only a few reports of lofepramine causing adverse effects on the peripheral nervous system. Although it is difficult to attribute this woman’s motor neuropathy directly to lofepramine, we draw attention to a previously unreported potential side effect of this drug.


Cyclosporin decreases nifedipine metabolism

Drs J P McCadden, J E Ponton, A V Powles, L FRY, and J R Iole (Departments of Pharmacology and Dermatology, St Mary’s Hospital and Medical School, London W2) write: In common with several other drugs nifedipine is metabolised by a glucocorticoid inducible cytochrome P450 enzyme, P450pcc. This same P450 isoform has recently been shown to be the major cyclosporin metabolising enzyme in human liver.1 In our post-surgery clinic we have observed reactions to nifedipine among patients taking cyclosporin. A 44 year old woman, who developed hypertension while taking cyclosporin for psoriasis (4 mg/kg/day), was started on nifedipine 40 mg/day. At follow up two weeks later she complained of unpleasant “burning” sensations two hours after taking nifedipine. The nifedipine was stopped, with abatement of the symptoms, but on restarting treatment she complained of severe flushing reactions, which again stopped after the drug was withdrawn. Another patient, a 54 year old woman taking cyclosporin (2 mg/kg/day) for psoriasis, developed a rash while taking nifedipine 20 mg twice daily, which cleared after stopping the drug. Data from the Committee on Safety of Medicines reveal that symptoms of paraesthesia, flushing, and rash are well known to occur with cyclosporin therapy. We have suggested that since cyclosporin is metabolized by cytochrome P450pcc, a possible interaction exists between the two drugs. To assess the possible interaction of cyclosporin and nifedipine at a metabolic level we performed a study to assess the effect of cyclosporin on P450pcc enzyme activity.

Eight psoriatic patients (three men, five women; mean age 49 years [range 32-64] years) were tested for P450pcc enzyme activity while taking cyclosporin (3 mg/kg in seven subjects, 4 mg/kg in one) and one week after cessation of the drug. All were given an oral 5 mg dose of nifedipine (Adalat, Bayer) after an overnight fast and voiding of the bladder. All urine collected in the eight hours after the dose was bulked and an aliquot stored at -20°C. Analysis of the urine capillary electrophoresis (ME) and the MII was by automated capillary gas chromatography.2 P450pcc activity was assessed by measuring the excretion of the ME, expressed as percentage recovery (MII excreted over eight hours divided by the theoretical MII possible by 100).

The mean percentage recovery while patients were not taking cyclosporin was 51.75 (SD 13.59)% compared with a mean of 34.58 (10.14)% while taking cyclosporin (P=0.05). Thus cyclosporin in vivo decreased P450pcc availability, the metabolism of nifedipine presumably being reduced through direct competition with cyclosporin.

Nifedipine is an effective, relatively safe anti-hypertensive agent that has a good record in the treatment of nephroprotective in subjects treated with cyclo-