Case report: A 44-year-old man of Polish descent first presented to the psychiatric services in 1982 with recurrent unipolar depressions. His subsequent treatment was complicated by his suffering significant side-effects on most first-line antidepressants. Those that he was able to tolerate (e.g. low-dose clomipramine) tended to induce hypomania after a few weeks of treatment. It was in some desperation, therefore, that I put him on L-tryptophan in 1985. Such was his apparent sensitivity to psychotropic medication that he became hypomanic on 4.5 g of L-tryptophan per day. During the next few months he became very stable on 1·5 g of L-tryptophan per day and he was discharged from follow-up in July 1985.

He was referred back by his general practitioner this August. It seems that he had remained entirely well over the five years previous to this, apart from a bout of depression with biological features every three months or so. At these points he took about 1·5 g of L-tryptophan over two days and found that this quickly restored his well being. When he was referred back to me he had been constantly depressed for three months with anergia, early-morning wakening, loss of appetite and anhedonia.

Another patient (a medical practitioner) who had suffered sleep disturbance on withdrawing from the L-tryptophan on which she had been maintained, had researched the topic and established that pumpkin seeds were the most cost-effective means of administering natural L-tryptophan: a typical 250 g bag contains 1300 mg and costs about £1.00.

Within 24 hours of ingesting about 200 grams of pumpkin seeds (i.e. about 1 gram of L-tryptophan), the patient felt quite transformed. He was no longer anergic or depressed and happily returned to work the following day.

Clearly this patient is atypical in that his mood state appears to be remarkably sensitive to L-tryptophan. However, for patients who were successfully maintained on L-tryptophan or for whom one wishes to instigate L-tryptophan, pumpkin seeds may represent the best alternative at present. Perhaps, as in this case, the biggest hurdle is overcoming the scepticism of one's patient!

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Down's syndrome, dementia and myoclonic jerks

SIR: The recent publicity surrounding bovine spongiform encephalopathy (BSE) has intensified the search for cases of the human concomitant, Creuzfeldt Jakob disease. The combination of a dementing process and myoclonic jerks in a person under the age of 70 years raises suspicions of such a diagnosis. I urge for caution before reaching this conclusion.

There are two accounts in the literature of a combination of Down's syndrome, dementia and myoclonus (Blumbergs *et al*, 1981; Good & Howard, 1982). Both were originally presumed to be Creuzfeldt Jakob disease but post-mortem findings indicated Alzheimer's disease which commonly occurs prematurely in people with Down's syndrome. Faden & Townsend (1976) draw attention to the confusion arising when myoclonus occurs in association with dementia, and assert that this should not be considered pathognomonic of Creuzfeldt Jakob disease and that a diagnosis of Alzheimer's disease should be considered. This difficult diagnostic problem is illustrated by the following case.

Case report: A is a 48-year-old single lady with Down's syndrome. She developed *grand mal* epilepsy three years ago and an electroencephalogram (EEG) at that time showed no evidence of a space-occupying lesion. She was successfully treated with phenytoin and seizures are now infrequent.

For the last 18 months there has been a steady deterioration in her memory and she is now unable to recognise close family members. Her speech has deteriorated and she spends much of her time muttering to herself. Her self-care has deteriorated and she has become doubly incontinent, necessitating full nursing care. Her motivation is poor and her mood is labile. One year ago she developed myoclonic jerks. These occur only 1–2 hours after waking and affect her head, arms, shoulders, body and legs. They are abrupt, brief, irregular, asymmetrical and can be so severe as to catapult her out of her wheelchair.

A repeat EEG shows generalised irregular 3–7 Hz slow activity, with no change from her EEG of 1987. In particular, there is no evidence of periodic biphasic or triphasic complexes characteristic of Creuzfeldt Jakob disease.

In the light of the experience of other authors, the time course of the clinical findings and the lack of specific abnormalities on the EEG of this patient, the diagnosis should be Alzheimer's disease until proven otherwise.

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How informed and binding is informed consent?

SIR: We report a case in which the patient's consent for an interview with him to be broadcast, given while he was well, had an affect on his mental state.