the disorder (cumulative evidence from the literature indicates winter and early spring as the period when most schizophrenic births occur).

This study, comprising 187 patients (117 males and 70 females under 40) who fully met the DSM–III–R criteria for schizophrenic disorder (American Psychiatric Association, 1987), showed considerable differences in familial predisposition to schizophrenia in the two seasonal groups. Five of the 84 patients born in the winter and early spring (6%), and 17 of the 103 patients born in the other months (16.5%) had a family history of schizophrenia ($\chi^2 = 4.9$, $P = 0.025$). Furthermore, morbidity risk data confirmed the role of family history. Five of the 241 age-adjusted relatives of the probands born between December and April (age adjustment according to Weinberg's abridged method) and 18 of the 323 age-adjusted relatives of the probands born during the remainder of the year turned out to be schizophrenics ($\chi^2 = 4$, $P = 0.043$), so that the latter group had a familial morbidity risk (5.3%) 2.65 times greater than that of the former (2%).

Consequently, our findings are apparently compatible with previous hypotheses of a lowered familial loading of schizophrenia among patients born in the winter and early spring, but manifestly differ from those of Drs Baron & Gruen. Undoubtedly, the lack of complete comparability of the protocols may have accounted for some of the differences in the results: for example, we used the end of April instead of the end of May as one of the benchmarks for separating the season of birth, and the Weinberg's abridged method instead of the more precise age correction based on continuous distribution of the ages at the disease onset. But we should not neglect the possibility that the samples selected in one or another or both of the studies are not completely representative of the phenomena under analysis. In fact, the figures for the secondary cases in our sample are somewhat inferior to those to be expected for relatives of Italian schizophrenics (Macciardi *et al*, 1987) and, on the contrary, the greater risk found by Drs Baron & Gruen (among the relatives of the patients born in winter and spring) apparently seems largely sustained by a few families with many cases of schizophrenia, i.e. by high-risk families. If this is true, then some of the discrepancies could be reconciled, given that systematic diathesis–stress interactions along a continuum of susceptibility are the bases for the seasonality phenomenon. Within this framework, we cannot only expect that the harmful effect of seasonal environmental factors is maximised when, as was apparently the case in our sample, the weight of familial predisposition to schizophrenia is in some way minimised, but also that the former further increases the likelihood of the schizophrenic disorder when subjects at high risk are predominantly considered.

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Antidepressant toxicity

SIR: I read with interest the review by Beaumont (*Journal*, April 1989, **154**, 454–458), and was surprised to note that there was no mention of the relatively new antidepressant fluvoxamine, which is reputed to be very safe when taken in overdose. Fluvoxamine inhibits the neuronal uptake of 5-hydroxytryptamine with little or no effect on the catecholamine system. Minor reported side-effects include: nausea, vomiting, dizziness, dyspepsia, headache, anxiety, palpitations, diarrhoea, and a rash (Roos, 1983; Classen *et al*, 1977).

There have been 42 cases of self-poisoning with fluvoxamine (Banerjee, 1988). One patient died, but necropsy showed the fluvoxamine tablets to be intact in the stomach, and the death was attributable to an overdose of propanolol. In another reported case, the patient was unconscious for five days, but recovered fully from her coma (Banerjee, 1988). The prolonged cerebral depression was thought to be due to a possible interaction between fluvoxamine and

benzodiazepine metabolites. Thus it appears from the available data that fluvoxamine is relatively safe in overdose compared with the tricyclic antidepressants.

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Attitudes to anxiety

SIR: We are grateful to the late Dr Kraupl Taylor (*Journal*, May 1989, **154**, 697–704) for his timely reminder that anxiety disorders are true psychiatric disorders rather than perjorative labels. The very first patient we saw after reading his article gave such a clear example of this prejudice that we felt moved to write.

Case Report: The patient, a 34-year-old married white male labourer, presented to our anxiety disorders clinic with a five-year history of panic attacks and generalised anxiety. The severity of his illness may be judged from his opening statement: "If you can't help me then I'll have to jump off the [Clifton Suspension] bridge". The onset of this disorder was in 1984, with a sudden, spontaneous, severe panic attack. He immediately took a taxi home, but during the journey diverted it to the local casualty department. The principal symptoms he described were bilateral chest pain and paraesthesiae. He was examined and was "reassured" that his symptoms were only due to anxiety.

He remained disabled, despite relaxation training, for 2 years, and was reassessed in 1986 by a cardiologist for continued episodes of faintness and chest tightness. This consultant's view was that there was no evidence of heart disease, and he stated that "the right policy was to reassure him strongly in the hope that they [the panic attacks] would go away". He was subsequently prescribed diazepam by his GP. However, because he was frightened of becoming addicted, he did not take the drug. Following this he was prescribed propranolol, which he found ineffective. He has continued to suffer, and his continued employment has been jeopardised by frequent absences from work caused by panic attacks.

This case serves to emphasise how underestimation of the personal suffering caused by anxiety may lead to inadequate treatment, and potential damage to family life and employment. In the light of this and the many other examples we have seen, we strongly support Dr Kraupl Taylor's point of view. We suggest that the present negative attitudes towards the illness concept of anxiety and the consequent anti-benzodiazepine climate of opinion causes much extra suffering for those people least able to tolerate it.

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The epileptic arsonist

SIR: The recent article by Carpenter & King (*Journal*, April 1989, **154**, 554–556), makes compelling reading. The association between epilepsy and arson is a fascinating one, and we would like to describe a further case seen recently in our unit.

Case Report: Ms B, a 26-year-old woman, was referred to our unit following an incident in which she set fire to a shop in her locality. This was one of a series of fires set by this lady, and she had received a diagnosis of personality disorder at another psychiatric institution. The patient was initially maintained on chlorpromazine therapy, and during her stay she set another fire on the ward. Careful reassessment of her case revealed that her fire-setting behaviour occurred in response to "a male voice", which she heard intermittently. EEG studies were performed, and revealed gross abnormalities consistent with an epileptic focus in the temporal lobe. The patient was started on carbamazepine therapy, and her auditory hallucinations have ceased. She remains much more settled on anticonvulsants alone, and there has been no recurrence of her fire-setting behaviour.

This is another case of arson associated with epileptic activity. The excellent response to anticonvulsants has led to a change in the diagnosis in this patient. Unlike Drs Carpenter & King's case there was no evidence of alcohol use or brain injury in this patient's history. Her progress has been most encouraging. Her legal status is currently under review, and the diagnosis of epilepsy should have considerable bearing on the outcome of her case.

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