Roughly estimated, the incidence of mania in our cohort is 1.7 per 100 000 per year. In Dublin the annual incidence of mania for the age group 18–29 was 12.9 per 100 000. In London the respective incidence rate was 7.6 and in Aarhus 5.0 per 100 000 (Daly *et al*, 1995). The finding suggests that the incidence of mania is very low in northern Finland.

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Seasonality of birth in Western Australia

SIR: McGrath *et al* (1995) examined seasonality of birth of patients with schizophrenia in a southern hemisphere data set. They report that the quarterly birth distribution of patients differed significantly from the estimated general population distribution. Given the paucity of such studies in the southern hemisphere, we report our experience of seasonality of birth in Western Australia.

We confined our analyses to patients born in Western Australia (total general population in 1960 in the vicinity of 700 000) between 1950 and 1960, who were recorded as having an in- or out-patient contact with the comprehensive Western Australia Mental Health Register, attracting an ICD–9 diagnosis of schizophrenia or related disorders (ICD–9 295.0–295.9, $n=1186$).

Following the methodology of McGrath *et al* (1995), patients were grouped into quarters depending on their date of birth: January to March; April to June; July to September; and October to December. The counts for these quarters were 303, 300, 296 and 287 respectively. Using monthly birth numbers for Western Australia, as recorded by the Australian Bureau of Statistics to determine the proportions of births in each quarter (24.09%, 25.08%, 25.27% and 25.56% in quarters one to four respectively), the expected number of schizophrenia births for each of the four quarters was estimated (285.7, 297.5, 299.7 and 303.1 for the first to fourth quarters respectively). A χ^2 analysis did not reveal any significant difference between the schizophrenia births and the general population ($\chi^2=1.97$, d.f.=3, $P=0.58$). Further analyses did not reveal significant differences between female schizophrenia births and the general female population ($\chi^2=2.01$, d.f.=3, $P=0.57$) nor between male schizophrenia births and the general male population ($\chi^2=3.01$, d.f.=3, $P=0{\cdot}39$).

Thus, our results fail to replicate the findings of McGrath *et al* (1995). A possible explanation for the disparity is that we confined ourselves to a single decade (hence the relatively low number of patients). However, restriction to Western Australia births allowed more accurate estimates of expected numbers of cases.

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Presenile dementia in a Down's syndrome adult with an unbalanced 21/21 Robertsonian translocation

SIR: An association between Down's syndrome (DS) and Alzheimer's disease has now been well established (Oliver & Holland, 1986). Virtually all DS subjects reported to date have had trisomy 21 karyotype. Rarely has Alzheimer-type dementia (ATD) been reported in non-trisomy 21 DS individuals. Such rare cases include subjects with mosaicism (Rowe *et al*, 1989), 21/22 translocation (Sylvester, 1986) and 14/21 translocation (Prasher, 1993). I report ATD in a patient with a previously unreported unbalanced Robertsonian 21/21 translocation.

A 56-year-old woman with DS recently died following a five year history of gradual deterioration in her level of functioning. A history of progressive memory loss, mental confusion, abnormalities of speech, change in personality, mood

lability, onset of urinary incontinence and generalised seizures, loss of mobility, loss of self-care skills and behavioural difficulties was evident. Six months prior to her death she was transferred to a nursing home where she was bedridden, required a permanent urinary catheter and needed 24-hour nursing care. She died from bronchopneumonia. Typical clinical features of DS were present. Prior to the onset of deterioration the patient was in good health and functioned at a moderate level of learning disability. Her full blood count, profile and thyroid hormone results were generally normal throughout her illness although macrocytosis was present for the last three years and borderline hypothyroidism was treated with thyroxine replacement two years prior to death. A clinical diagnosis of Alzheimer's disease was made. There was no family history of dementia although one sister required treatment for depression. The patient's karyotype was 46,XX,-21,+t (21q21q).

Genetic aspects of ATD have recently aroused considerable interest with particular reference to apolipoprotein E genotype and location of the gene coding for amyloid precursor protein on chromosome 21 (Goate *et al*, 1989). It is possible that further significant advances could be made by investigating the occurrence of ATD in uncommon forms of DS. Molecular genetic studies of these rare cases is recommended as they could aid determination of genetic loci predisposing to Alzheimer's disease.

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Train of thought

SIR: I am not a train spotter, but given my interest in Asperger's syndrome, I am a train spotter spotter. I note that many of my colleagues are also train spotter spotters, which makes me a train spotter spotter spotter. I have not yet met any other train spotter spotter spotters but I have started a list just in case and will write down their numbers if I can.

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