Most rating values will, therefore, be close to the mean but there is a high probability of one or more outlying points occurring. This will cause statistically significant, but spurious, correlations.

Taking all of this into consideration, I agree with the authors' conclusion that "Further research is clearly needed to substantiate these results".

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Prevalence and incidence of schizophrenia in Afro-Caribbeans

SIR: The recent articles by Wessely *et al* (*Journal*, December 1991, **159,** 795–801) and Castle *et al* (*Journal*, December 1991, **159,** 790–794) describing increased incidence of schizophrenia in those of Afro-Caribbean origin, and Eagles' description of the possible alternatives to psychosocial explanations (*Journal*, December 1991, **159,** 783–789) raise a number of issues.

A recent epidemiological study on the Caribbean island of Dominica showed that over a 30-month period the age-corrected prevalence rate for schizophrenia by ICD–9 diagnosis was 11.8/1000 (Kay, 1990). This figure was obtained from a computerised case register of all admissions, out-patient referrals and prison consultations supplemented by the use of key informants in every major village. When DSM–III diagnostic criteria were applied, the prevalence decreased to 7.8/1000. Given the limitations of case finding in this study, the rates found by either diagnostic system are significantly higher than those generally found in the UK.

Incidence rates for schizophrenia in Dominica in 1989, using the same case register were, by ICD–9 14.8/10 000, and using DSM–III criteria 7.3/10 000, which are considerably higher than comparative figures in Camberwell for the total population (Castle *et al*) by a factor of 6 to 8, but closer to the increased risk ratio of schizophrenia if of Afro-Caribbean origin (Wessely *et al*). The smaller proportion of ICD–9 cases meeting DSM–III criteria in Camberwell may reflect possible diagnostic bias and, in particular, cases of affective psychosis may masquerade as paranoid schizophreniform psychoses possibly as a result of culturally determined projective defence mechanisms being more prevalent in Afro-Caribbeans.

If results from one island state can be generalised to the Caribbean region this would suggest that the increased risk of schizophrenia meeting DSM–III criteria in the UK for those of Afro-Caribbean origin must be explained by factors operating both in the UK and the country of origin, and not the potentially racially prejudiced interaction with the UK mental health care system. Possible explanations include those factors discussed by Eagles. With increasing immunisation rates, and obstetric care approaching UK levels in Dominica (McIntyre, 1988), it will be interesting to observe if there is any decrease in the incidence of schizophrenia in the next 15 to 20 years. Increased genetic risk is possible but unlikely given low reported rates of schizophrenia in West Africa (Sikanertey & Eaton, 1984) where most Afro-Caribbeans originate, and theories based on concentration of genetic factors in small gene pools are unlikely given low rates of other congenital diseases.

A major culturally determined factor in my experience is that the consumption of large amounts of cannabis can act as both a precipitant of schizophreniform psychoses and as a factor in causing relapse and maintaining chronicity, in pre-existing schizophrenia, although the published literature is contradictory. This may partially explain the high incidence and prevalence rates found in Dominica if not to the same extent those found in Camberwell, where cannabis consumption may be assumed to be lower. However, this is an area requiring further study.

KAY, R. W. (1990) Prevalence of psychotic mental disorders in the Commonwealth of Dominica (Abstract) *West Indian Medical Journal*, **39** (suppl.1), 30–31.

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Monsieur Pascal's cognitive therapy?

SIR: I was intrigued recently to read the following and to note the similarities that there are with the concepts that underlie 'modern' cognitive therapies.

> "The nature of man is so framed, that not only by often hearing himself a fool, he believes it; but by often calling himself a fool, he enters into the same opinion. Every person holds an inward and secret conversation with his own breast, and such as it highly concerns him well to regulate, because even in this sense, evil communications corrupt good manners. To study silence as much as possible and to converse with God alone, is the true persuasion in respect of ourselves."

PASCAL (transl. 1893) *Thoughts on Religion and Other Subjects*. Translated into English by Basil Kennet, DD. London: George Rutledge and Sons.

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Lowest effective dose of depot neuroleptics

SIR: We would like to support the statement of Marder *et al* (*Journal*, May 1991, **158,** 658–665) that monitoring of plasma levels is helpful to treat patients with the lowest effective dose of a depot neuroleptic such as fluphenazine. We have followed up stable out-patients on depot neuroleptics for between two and four years. Our data support the statement of Marder *et al* that plasma levels of fluphenazine should be between 0.53 and 3.0 ng/ml.

Our patient group consisted of 103 stable out-patients (mean age 42 years, range 18–72, mean weight 76 kg, mean height 1.75 m), receiving depot medication for at least six months (mean interval 13 days; range 7–28) and having no change in medication for three months.

All patients had schizophrenia or schizoaffective disorder as a primary diagnosis. The types of medication were: fluphenazine (FPZ) $n=27$; flupenthixol (FPX) $n=57$; and haloperidol (HAL) $n=19$.

Repeated measurements of plasma levels, one hour before injection, using a high pressure liquid chromatography or gas liquid chromatography method, were performed. No metabolites were determined. The coefficient of variation was less than 5% for FPZ, FPX and HAL, quantitation limit FPZ 0.2 ng/ml; FPX 0.5 ng/ml; HAL 0.5 ng/ml.

An estimated linear regression for all data was performed for each medication group. The 'kinetic' model used was the anticipated relationship between the administered dose at the previous visit divided by the number of weeks since the previous visit (i.e. interval) and the plasma concentration. The usual way to estimate the assumed linear relationship (with respect to time) is by use of a linear regression model with no respect to different variances at different dose levels.

Plots of estimated line, confidence limits and observations were made, 'B' representing the slope of the regression line in the case of one variable. The confidence interval of 95% is the interval of the regression line. The relationships between plasma level and dosage are shown below.

For fluphenazine: B = 0.04, 95% confidence interval = 0.027–0.0053. 52/58 observations (89.7%) of the plasma levels were within the 0.5–3.0 ng/ml range.

For flupenthixol: B = 0.05, 95% confidence interval = 0.038–0.06. 114/140 observations (81.4%) of the plasma levels were within the 0.5–3.0 ng/ml range.

For haloperidol: B = 0.05, 95% confidence interval = 0.02–0.07. 42–47 observations (89.3%) of the plasma levels were within the 0.5–4.0 ng/ml range.

The results show that in this stable group the plasma levels are within a definite range. The variation of haloperidol is higher than for flupenthixol and fluphenazine.

As Marder *et al* (1987) and Johnson *et al* (1987) have shown for fluphenzine and flupenthixol, lower dosage may be as effective as higher dosage in preventing relapse in maintenance therapy with depot neuroleptics.

However, both report that a 'too low' dosage leads in the long term (more than one year) to a statistically greater chance of relapse. Marder *et al* (1990) showed that lower plasma levels of fluphenazine (<1.0 ng/ml) do indeed significantly correlate with a relapse after six or nine months. They measured the plasma level at the day of injection using a RIA-method and report a range of 0.5–3 ng/ml.

Norman *et al* (1987) found in a comparable group of stable out-patients on long-term fluphenazine decanoate an average plasma level for males ($n=9$) of 1.3 µg/l and for females ($n=8$) 1.0 µg/l. They found no relationship between BPRS-scores and plasma levels over time.

In a preliminary analysis we found no influence of anxiolytics, anticholinergics, or sex on plasma level. We realise that our study is methodologically weak, but the results correlate quite well with the results of the study that Marder *et al* report in this journal. Furthermore, we think that our data do support the view that in future research on relapse prevention in maintenance therapy, more attention should be given to plasma levels, since patients receiving 20 mg or 40 mg of flupenthixol or fluphenazine per week may have the same plasma level. Individual, unique, metabolic factors may play a role in these differences. Other factors such as age, race, or co-medication might also influence the plasma levels.

We propose for future studies concerning relapse prevention in maintenance therapy that plasma levels of flupenthixol of 0.5–3.0 ng/ml, fluphenazine 0.5–3.0 ng/ml and haloperidol 1.0–4.0 ng/ml are studied.

JOHNSON, D. A. W., LUDLOW, J. M., STREET, K. *et al* (1987) Double-blind comparison of half-dose and standard-dose flupenthixol decanoate in the maintenance treatment of stabilised out-patients with schizophrenia. *British Journal of Psychiatry*, **151,** 634–638.

MARDER, S. R., VAN PUTTEN, T., MINTZ, J., *et al* (1987) Low-and-conventional-dose maintenance therapy with fluphenazine decanoate. *Archives of General Psychiatry*, **44,** 518–521.