(Dalen, 1968, 1974; Ødegård, 1974; Hare et al, 1974). It therefore seems reasonable to examine the recently reported data specifically for cyclic trends. Edwards' method (Edwards, 1961), probably the most commonly used, allows the estimation of cyclic trends and also the calculation of the time of year when birthrate of schizophrenics (in this case) is maximal. More recently, Roger (1977) has noted difficulties in applying Edwards' method to small numbers of observations and has devised a more sensitive alternative.

For each of the three schizophrenic populations reported in the original study, total monthly births were divided by the mean monthly seasonal indices of total live births for the years 1939–1971 (OPCS, 1971) (ideally, mean indices should have been calculated for the period 1936–1960, which covered the years of birth of 75 per cent of the total schizophrenic sample, but data were not available before 1939). This step corrects the original data for seasonal bias in monthly birthrate in the population as a whole, i.e. had the original schizophrenic groups constituted representative samples of the normal population of England and Wales in terms of season of birth, seasonal variations ought not to have been detected in the corrected data.

TABLE

Variation in season of birth of schizophrenics of different genetic risk. The SCZ group had first or second degree relatives with schizophrenia, the APD group had similar relatives with any psychiatric diagnosis

Group	N	Seasonal variation in birthrate*	Peak month of birth†
Low genetic risk	598	0.025 > p > 0.01	October
High genetic risk APD SCZ	377 92	0.025 > p > 0.01 0.3 > p > 0.2	May April-May

^{*} The probability that the observed trends in corrected monthly birthrates could have arisen by chance, assessed according to Roger (1977).

† Calculated according to Edwards (1961).

The results (Table) show that this is not the case. All three groups of schizophrenics display seasonal variations in birthrate although for the SCZ group (with first or second degree relatives with schizophrenia), this trend fell short of significance. Particularly noteworthy is the fact that the peak month of birth of this group is very similar to that of the other 'high genetic risk' group and differed by six months from the peak month of birth of the 'low genetic risk group'.

This highlights the potential differences between high and low genetic risk groups, reinforcing the need for their separate analysis. The results as presented here are not necessarily incompatible with the original analysis, which was based on a different model for seasonal variation not assuming cyclic variation. However, they serve also to illustrate the applicability of appropriate tests for cyclic variation which tend to have been underused in psychiatry as elsewhere in epidemiology.

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PSYCHIATRIC MORBIDITY AND CIRCADIAN RHYTHMS

DEAR SIR,

In their elegant assessment of psychiatric morbidity and time zone changes, Jauhar and Weller (*Journal*, March 1982, 140, 231–35) show that most hypomanic cases (10/15) had flown eastwards, and all depressive cases (8) had flown westwards.

In addition to earlier known facts, with regard to the effects of drugs on affective disorders, and the relation between affective disorders and circadian rhythms (Pflug, 1978), this makes it possible to add the effects of time zone changes on mood.

Flying east reduces the day below the ordinary 24 hours (when departure is in the evening and arrival in the morning, the real dark period is usually less than an hour, so that the night often becomes totally omitted). Flying east also means a phase advance in the sleep-wake cycle. Elevated mood (hypomania) was the result in 10 patients (*ibid.*). A phase advance in the

sleep-wake cycle has also been shown to have some antidepressant effect, thus also elevating mood from depression to normal (Wehr et al, 1979).

It is now well known that sleep deprivation elevates mood (Pflug, 1978). A deprivation of a night's sleep may result in a slight increase in mood, as is often experienced by doctors after an entire night on duty. Whether such sleep deprivation is causative or is only the effect of mania ("decreased need for sleep" as indicated in DSM III) is still an open question.

Tricyclic antidepressants (TCA) are not so far known to affect the circadian rhythm, but increase mood in depression. It has also been said that they might induce mania (Bunney, 1978), but this has recently been contradicted (Lewis, 1982).

Flying west prolongs the 24 hour day, and delays the circadian cycle. In Jauher and Weller's material, all eight of the depressed patients who had flown along circles of latitude had flown westwards. There is no published evidence that flying west might reduce mania, although one of my patients indicated such an effect. One manic patient, with mania resistant to pharmacotherapy (neuroleptics and lithium) recovered after a journey westwards.

No reports have shown that sleep induction or increased sleep may reduce mania. Another of my patients with recurrent mania or hypomanic attacks after not sleeping for 3-7 days cut his attacks by drinking himself to sleep after a period of 10-15 days. The possibility that sleep might affect mania should be investigated further.

Lithium is well known to reduce mania. It also affects the circadian rhythm (increasing the time cycle) both in plants and in man (Engelmann 1973; Johnsson et al, 1980).

TABLE

Variable	Night duration	Mood
Fly east	_	+
Sleep deprivation	_	+
TCA	?	+
Fly west	+	
Sleep	+	?
Lithium	+	_

Mood changes (+ = elevation, i.e. development of mania or recovery from depression — = lowering) related to changes in night duration (+ = increase; — = decrease) after east/west flights, sleep deprivation sleep, and drugs used in affective disorders (Tricyclic antidepressants/lithium).

The information available today is compiled in Table I. Rather congruent findings indicate that the affective disorders involve a disturbance in the

circadian rhythm system. It has earlier been suggested that abnormalities of sleep patterns in some types of depression are due to abnormal internal phase relationships of circadian rhythms (Wehr et al, 1979). It is fair to suppose that this also includes manic states. In the future, this might also have further implications for the treatment of affective disorders.

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TREATMENT OF COMPULSIVE GAMBLING DEAR SIR.

The article by Drs Greenberg and Rankin (Journal, April 1982, 140, 364-66) prompts me to question whether the cautious and limited use of apomorphine as an aversive agent might not reasonably be reconsidered.

Several years ago I used a short course of apomorphine injections with apparent success in the treatment of a young man whose addiction to fruit machines had landed him in trouble with the law and threatened the breakdown of his marriage. With the co-operation of a local licensee, the patient was taken twice a week to a nearby public-house during afternoon closing hours, given an injection of apomor-