

- HALSBOER, F., LIEBL, R. & HOFCHUSTER, E. (1982) Repeated dexamethasone suppression test during depressive illness: normalisation of test result compared with clinical improvement. *Journal of Affective Disorders*, **4**, 93–101.
- NELSON, J. G. & BYCK, R. (1982). Rapid response to lithium in phenelzine non-responders. *British Journal of Psychiatry*, **141**, 85–6.

SMOKING PROFILES OF PATIENTS ADMITTED FOR NEUROSIS

DEAR SIR,

I would like to reply to the response of Salmons and Sims (*Journal*, July 1982, **141**, 103) to criticism of their methods and conclusions (*Journal*, January 1982, **140**, 103) reported in the article titled "Smoking Profiles of Patients Admitted for Neurosis" (*Journal*, July 1981, 43–6). Smoking is too important a public health problem for associative or etiological factors to be imputed on scant evidence.

Salmons and Sims appear bent on concluding their hypotheses "upheld", despite the absence of results which would warrant rejection of the null hypotheses (i.e. no significant differences between groups). Inappropriate use and interpretation of statistical tests is not unusual; however, it is not common to find authors willing to present such a large number of non-significant differences in support of their argument.

In acknowledging the direction of the only significant difference between groups reported in their Table III, Salmons and Sims claim this supports their original contention of earlier age onset of smoking by neurotics, as ". . . those who are smokers have started in a much younger age group, leaving only a smaller number to start later". In the younger male age group cited, the (non-significant) difference reported was 49.7 per cent in the general population sample vs. 62.9 per cent in the neurotic patient sample. Salmons and Sims argue that this leaves only 8.1 per cent of the neurotic male sample vs 22.5 per cent of the general population sample to start smoking later. These last figures are interesting, as they indicate the authors have noted that roughly the same proportion of the general population male sample and the neurotic male sample reported having started to smoke by age 30 (i.e. 72 per cent and 71 per cent). However, absence of a significant difference between groups in the younger age group would appear to weaken the argument of Salmons and Sims that neurotics start smoking at an earlier age.

To focus on Table III, describing only contrasts regarding 'Age at Starting to Smoke', may be seen as unfair, as this represented only one component of the authors' search for an association between neuroticism and smoking. Salmons and Sims also reported a higher

proportion of smokers in both age and sex groups in the neurotic sample than in the general population sample, that this higher proportion was maintained over all social classes, and that neurotic patients inhaled more deeply. In all, seventeen statistical comparisons were reported in Tables I-IV of Salmons' and Sims' original paper. Of these seventeen contrasts, only four comparisons resulted in χ^2 values which had a probability of occurring by chance .05—findings generally accepted as reason to 'reject the null hypothesis'. Salmons and Sims have apparently chosen to break with this convention. In cases where statistical tests have indicated a significant difference, they view the difference between comparison groups as "especially marked".

If an association is *not* "especially marked", it often does require a relatively large sample size to be demonstrated. Medical researchers are frequently frustrated by this, as samples generally do not come in 'jumbo' sizes in clinical series. Yet, clearly, there is danger in generalizing from a trend in the data obtained from a small and select sample. Salmons and Sims report their findings as contradicting those obtained in earlier research (e.g. Eysenck, 1963; Eastwood and Trevelyan, 1971), and suggest the factor of "neuroticism" must be taken into account in planning community health strategies directed at reducing smoking in the general population. As the earlier studies were conducted on much larger, community samples, perhaps the previous absence of a significant association should be taken more seriously than this more recent interpretation of data from an inpatient sample.

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TARDIVE DYSKINESIA IN A PHANTOM LIMB

DEAR SIR,

In their article on the phantom limb phenomenon (*Journal*, 1982, **141**, 54–58), Shukla, Sahu, Tripathi and Gupta discussed the various theories of the phantom limb, including central and psychological mechanisms. We present here a case of tardive dyskinesia in a phantom limb which would support a central mechanism for the phantom limb phenomenon.

A 42-year-old white single male with a DSMIII diagnosis of schizoaffective disorder, antisocial personality, and alcohol abuse, was referred to us for management of persistent dyskinesia of two years duration. The patient had lost his right foot and three toes on his left foot 11 years prior to admission due to