Two controlled trials conducted by myself and colleagues in Dundee (Worral *et al*, 1979) are representative and discussed the issues at that time. One of those trials, in which tryptophan was compared with lithium and tryptophan in combination, is regularly quoted in the subsequent combined studies and used as evidence for lithium having an augmenting effect on other antidepressants. We specifically stated that the design of that trial could not possibly prove that. The simplest explanation in that trial was that lithium was acting alone and, in truth, tryptophan was only used because we did not obtain ethical permission for a placebo comparison.

What seems to have happened is that influential figures twenty years ago stated that lithium did not have important acute antidepressant effects and, with minor modifications, that statement has been authoritatively repeated in nearly all reviews since. What the combined studies have shown is that when a first-line treatment has proved ineffective and the clinician has then tried lithium, he has not believed his own eyes when he has seen a major acute antidepressant response, and some form of potentiation or augmentation of the first drug by lithium is instead suggested.

Why expose patients simultaneously to two drugs without first trying each separately, especially if, as is likely, continuation treatment is going to be needed? It may be that in a few patients lithium does need to be used along with another antidepressant, or a neuroleptic, and it would be surprising if on occasion two antidepressants from a different class did not have more effect than one. A controlled trial to prove that would be feasible, but before going to that trouble an open-minded reappraisal of the effects of lithium alone might make such a trial less necessary.

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References

STOKES, P. E., STOLL, P. M., SHAMOIAN, C. A. & PATTON, M. J. (1971) Efficacy of lithium as acute treatment of manic-depressive illness. *Lancet*, *i*, 1319–1325.

WORRAL, E. P., MOODY, J. P., PEET, M., DICK, P., SMITH, A. CHAMBERS, C. A., ADAMS, M. & NAYLOR, G. J. (1979) Controlled studies of the acute antidepressant effects of lithium. *British Journal of Psychiatry*, **135**, 255–262.

At Risk Drinking

Sir:I was delighted to read the article by Dr King (*Journal*, May 1986, **148**, 533–540). In this and other articles Dr King has used the CAGE Questionnaire. Unfortunately, he has repeated a failure of other authors to attribute the questionnaire to the correct source. The CAGE questions were developed by me and my colleagues at the University of North Carolina in the late 1960s. The CAGE questions have been used in many different studies by now, and recently (Ewing, 1984) I published a paper in the *Journal of the American Medical Association* describing their origins, clinical use and efficacy. That reference might be the best one for Dr King and others to use in future.

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References

EWING, J. A. (1984) Detecting alcoholism: The CAGE Questionnaire. *Journal of the American Medical Association*, **252**, 1905–1907.

Anhedonic Depression

Sir: Young *et al* (*Journal*, March 1986, **148**, 257–267) have suggested the possible existence of two subtypes of endogenous depression—anhedonic subtype and vegetative sub-type. Earlier, Hibbert *et al* (1984) reported that symptoms of reduced interest and pleasure and of low mood represent features of the state of depression which are related to each other, but are not much related to the more 'biological' symptoms of reduced sleep and appetite.

In a study of 34 RDC major depressives, I classified the patients as anhedonic and non-anhedonic based on high or low scores on the sub-scale anhedonia-asociality of the Scale for the Assessment of Negative symptoms (SANS) (Andreasen, 1981). The anhedonic sub-type had a significantly ($P < 0.001$) longer duration of illness than the non-anhedonic sub-type. Almost 20% of anhedonic depressives had a duration of illness longer than nine months with treatment. The response to tricyclic antidepressants was unsatisfactory in the anhedonic depressives and the need for adjuvant therapy with electroconvulsive shocks and antipsychotics was significantly ($P < 0.01$) more often required for them. Anhedonic depressives also required a much longer duration of treatment than the non-anhedonic depressives (Chaturvedi & Sarmukaddam, 1986). Interestingly, anhedonic depressives had significantly higher total scores on SANS than non-anhedonic depressives.

These findings help further in identifying an anhedonic sub-type of depression. However, in my study, no demographic differences were observed

between the two groups, especially as regards, age, sex, education, occupation or marital status. There is a need for research on biological parameters to further validate the sub-types.

References

ANDREASEN, N. C. (1981). *Scale for Assessment of Negative Symptoms*. Iowa City: The University of Iowa.

CHATURVEDI, S. K. & SARMUKADDAM, S. (1986) Prediction of outcome in depression by negative symptoms. *Acta Psychiatrica Scandinavica (in press)*.

HIBBERT, G. A., TEASDALE, J. D. & SPENCER, P. (1984). Convariance of depressive symptoms over time. *Psychological Medicine*, 14, 451 455.

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Quetelet's Index and Appetite and Weight Change in the Context of Depression

Sir: A study of 168 mild to moderately depressed out-patients presenting over a period of six years at St Thomas's Hospital, London, and involved in a series of antidepressant trials, showed that factors linked to appetite and weight change were previous appetite and weight history. Patients gaining appetite and weight with depression had a previous history of overeating in response to the normal stresses and strains of everyday life, and of variable weight history (Harris *et al*, 1984*a*). With recovery after six weeks' treatment, these patients tended to *lose* appetite and weight (Harris *et al*, 1984*b*). A possible explanation for this is to be found in the concept of restraint. According to Polivy & Herman (1976) restraint is characterised by preoccupation with the caloric values of food, dieting, and guilt feeling on diet-breaking. It is high in obese and latently obese subjects, and in normal or even under-weight individuals who respond to societal pressure to be slimmer and run below their "set-points" of body weight. They hypothesised that with anxiety and depression such individuals lose restraint, and there is increase in food intake and weight. Obviously, in large populations of depressives, there will be overlap with factors linked to obesity, and the further question arises as to whether or not it is the fatter patients (i.e., fat relative to height, not fat relative to pre-episode weight), who gain appetite and weight with depression and then *lose* appetite and weight with recovery. To examine this, we looked at subjects in terms of Quetelet's Index (QI) Weight/Height^2 (Quetelet, 1869). QI has recently been validated as a convenient formula and reliable indicator of obesity (Garrow & Webster, 1985): 20–24.9 representing desirable weight, 25–30 mild overweight, and over 30 being clinically relevant.

The method of the study has been reported elsewhere (Harrris *et al*, 1984*a*). At presentation, height and weight were measured, and after six weeks' treatment, patients were weighed again. QI was calculated in terms of the patient's stated *normal* weight. Appetite was scored at presentation and at six weeks, relative to patient's own norm. Standard statistical methods were used, including the unpaired t-test and the product-moment correlation coefficient.

Appetite and QI. At presentation, appetite was more often reduced than increased, and the relative appetite was positively correlated with QI ($r=0.15$ $0.05<P<0.1$). After six weeks' treatment this was reversed and there was a negative association between appetite and QI ($r=0.18$ $P<0.05$). The patients were also divided according to appetite into groups with marked increase, moderate change and marked decrease. These groups were delineated by boundaries approximately one standard deviation either side of the mean. If delineation was by initial appetite, the differences between groups were not significant. When groups defined by appetite at six weeks were examined, subjects whose appetite was severely decreased were significantly fatter (QI mean 28.2) in comparison with those who had both marked increase and moderate change ($P<0.01$). When projected back to what they were at presentation, the same group had had relatively high appetite.

Weight and QI. Weight at presentation showed no significant associations with QI. Patients were also divided into three groups: those with marked increase in weight (109% of their normal weight), those with moderate change (95 to 109% of normal), and those with marked decrease in weight (95% of normal weight). These groups were delineated by one standard deviation beyond the mean (102%). There were no significant differences at presentation but at six weeks the group currently less than 95% of customary body weight had a higher mean customary QI of 25.8 ($P<0.05$).

A number of practical implications result from our findings concerning QI. First, there is a tendency for fatter individuals to increase appetite with depression. Therefore, identification and management of depressive mood changes is an important factor in the management of obesity. Secondly, the question arises as to whether or not further weight gain will occur with antidepressant treatment, e.g., due to tricyclic-induced carbohydrate craving. Our findings indicate that in the short term this is not the case, but that loss of appetite occurs, probably due to regain of control. It might be possible, therefore, to use a first line antidepressant drug such as a tricyclic