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Correspondence

GLUCOSE TOLERANCE IN DEPRESSION

DEAR SIR,

Herzberg, Coppen and Marks have recently declared that the glucose tolerance in depressive patients does not deviate from the norm (*Journal*, May, 1968, p. 627). However, to my mind it would be premature to conclude from this that carbohydrate metabolism is unimpaired in depression. Van Praag and Leijnse (1, 2) made a longitudinal examination in depressive patients for the arterio-venous difference in glucose ($\triangle$ glucose) and non-esterified fatty acids ($\triangle$ NEFA) concentration: criteria of the extrahepatic uptake of glucose and NEFA respectively. It appeared that within a group of patients suffering from endogenous depression there is a subcategory with abnormally low $\triangle$ glucose values. Besides this, they found indications that the subnormal extrahepatic uptake of glucose is attended by an increased consumption of NEFA. It is not possible as yet to distinguish clinically between endogenous depressive patients with and without disordered energy metabolism.

The group of the endogenous depressions is fairly homogeneous as far as the psychic symptoms are concerned. Similarity in symptomatology does not, however, mean similarity in origin. Pathophysiological mechanisms of a different nature can draw forth the same clinical syndrome: of this somatic medicine supplies several examples. At any rate the above-mentioned observations make it probable that, in the field of depression, distinctions of a metabolic nature are possible that do not concur with the usual psychopathological distinctions. Averages can be deceptive in biological psychiatry.

It seems to me that this view is also, and indeed particularly, significant for clinical psychopharmacology (3, 4).

H. M. VAN PRAAG.

Department of Biological Psychiatry,
Psychiatric University Clinic,
Oostersingel 59,
Groningen,
The Netherlands.

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DEAR SIR,

I was interested to read the paper by Drs. Herzberg, Coppen and Marks (*Journal*, May, 1968, pp. 627–630). After investigating 14 patients, only eight of whom were given intravenous glucose-tolerance tests, they conclude that "carbohydrate metabolism . . . is unimpaired in depression if the effects of previous undernutrition and inactivity are adequately controlled". They also state that: "No abnormality in glucose tolerance attributable to depression was observed. . . ."

I feel that these conclusions are an over-simplification of a complex problem and are not supported by the authors' data. Firstly, in my experience, the effects of undernutrition cannot be reversed as easily as the authors suggest. Reviewing data from three investigations of glucose utilization in depressions (Pryce, 1960) I found 11 cases (out of a total of 43) with an initial glucose utilization rate (K) of less than 1·30 per cent. per minute and an increase in K of 0·25 per cent. per minute or more on re-testing. This group had a low mean initial body-weight (53·4 kg.), a significant mean gain in weight (3·1 kg.) after treatment, and included the only episodes of illness (5 in 4 subjects) in which feeding difficulties were encountered before (initial) tests, which suggested that previous carbohydrate deficiency might have been responsible for the observed changes in glucose utilization. However, this association of marked weight-gain with a marked increase in K was observed not only in the five instances with feeding difficulties, but also in four others in whom an adequate hospital diet supplemented with 150 g. glucose daily was taken for four days before testing. Moreover, in four instances the increase in K was due not to expected low initial values but to abnormally high re-test values, suggesting that a phase of