erroneously states that I appear to concur with the view that the effect of vulnerability factors can only be demonstrated with data about life events and difficulties. The quote Cooke uses to support his statement about my view is in fact a quote from Brown (Roy, 1978). My studies were carried out totally independently of Brown, or his critics, and my first hypothesis was in fact that these factors would not be found in depressed psychiatric patients.

It may be a bonus if these risk factors, as I prefer now to call them (Roy, 1979), can be studied with information about events and difficulties though I believe they and other risk factors can be studied alone.

Therefore, the conclusions that early parental loss, unemployment and poor marriage before onset of depression are risk factors for 'neurotic' depression in both female and male depressed working class patients do follow from my data (Roy, 1978, 1981). Their specificity for depression is the focus of current work (Roy, 1981).

ALEC ROY

Clarke Institute of Psychiatry,
250 College Street,
Toronto, Canada

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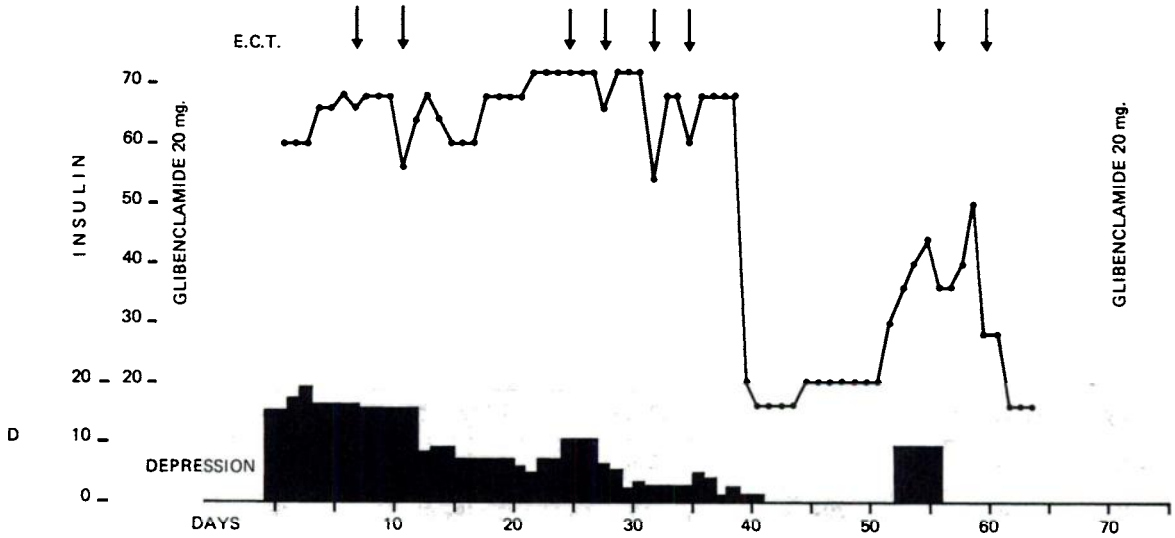
PSYCHIATRIC ASPECTS OF DIABETES MELLITUS: DIABETES AND DEPRESSION

DEAR SIR,

There is a need to look out for a biochemical relationship between diabetes mellitus and depressive illness, although so far nothing of this kind has been described in the clinical literature. We recently had as a patient a woman in her 50's, who shows such a link. She has both diabetes and affective disorder in her family history. Her first serious depression, successfully treated with ECT as an in-patient, was when she was 31, and she had several further depressive attacks in the following 15 years. Eventually, the manic-depressive condition became cyclical, repeating about every 20 weeks. Lithium treatment diminished the severity but did not abolish the cycle.

When she was 50, onset of vulval irritation drew attention to her maturity onset diabetes, which was well controlled for a time with an oral anti-diabetic agent. However, when she was admitted to our metabolic ward in a particularly severe retarded depression, her diabetes was found to be out of control. Off all drugs, and on a firmly enforced 1,000-calorie diet, she required about 60–70 units soluble insulin per day to keep her urine (tested four-hourly) fairly free of glucose. Her depression did not resolve spontaneously, and she was given ECT (see vertical arrows in Figure).



FIG—Course of depression and diabetes mellitus: upperline, soluble insulin daily in units; black areas, depression rating in arbitrary units from daily nurse ratings on a Phipps Behaviour Chart. Arrows, ECT.

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After the sixth ECT her depression—rated daily on a Phipps Behaviour Chart—had disappeared, and her insulin requirement declined dramatically, this being signalled by an unexpected hypoglycaemic attack. After 10 days her depression began to return, heavy glycosuria reappeared, and insulin had to be resumed. Two further ECT produced fresh recovery and she was able to be discharged on oral glibenclamide.

Plasma cortisol and urinary free cortisol, and also urinary vanillyl mandelic acid (VMA) derived from adrenalin, were examined on two occasions during her depression, and all values were within the normal ranges, making it unlikely that change in cortisol or circulating adrenalin could explain the increased insulin requirement. During her manic phases, in contrast, no change in diabetic treatment was required.

There is a great need to identify further cases where insulin requirement rises during depression in spite of good control of diet, and to explore whether growth hormone, or some other factor plays any part in it.

It is interesting that last year Fakhri *et al* reported from Baghdad in the *Lancet* (1980, *ii*, 775–77) that ECT was a successful treatment of maturity onset diabetes. We are grateful for Dr R. Donmal's help.

JOHN CRAMMER
CHIRSTY GILLIES

Institute of Psychiatry,
De Crespigny Park,
Denmark Hill, London SE5

DEAR SIR,

In his review article on this subject (*Journal*, January 1981, **138**, 1–9), Dr Wilkinson alluded to a possible relationship between diabetes mellitus and affective disorder, but found little evidence in the literature to support this conclusion. Recent developments in the psychoendocrinology of the affective disorders have shown lowered glucose utilization rate and insulin resistance in patients with endogenous depression (Mueller *et al*, 1969; Carroll, 1969; Wright *et al*, 1978). Although the diabetogenic effects of such metabolic abnormalities are of doubtful clinical significance in the depressed patient with no medical problems, the occasional diabetic patient who also suffers an episode of depression will often find his diabetes more difficult to control during the course of his depressive illness.

We recently treated a 66-year-old man with a long history of insulin-dependent diabetes mellitus for a recurrent unipolar endogenous depression. During his stay in hospital, his serum glucose concentration was monitored at 8.00 a.m. and 4.00 p.m. daily, and his Hamilton depression rating scale (Hamilton, 1967) was monitored weekly. His insulin requirements were adjusted according to the serum glucose concentrations, with the goal of keeping his serum glucose level in the 100–200 mg/100 ml range. Although there were fluctuations in the glucose concentrations and insulin requirements from day to day, there was a trend, as shown in Table I, for higher serum glucose concentrations and higher insulin requirements during the depressive episode (Hamilton score 16) than following recovery (Hamilton score 6). Diet and physical activity remained constant, and thus could not account for the changes in glucose concentrations and insulin requirements. The dose of imipramine was increased, but imipramine is not known to have an anti-diabetic effect.

During a severe episode of depression, the insulin antagonists growth hormone (Mueller *et al*, 1969), cortisol (Carroll *et al*, 1976) and epinephrine (Wyatt *et al*, 1971) are often secreted in excessive amounts. This is most likely the basis for the instability of diabetic patients who also suffer from depression. Further studies among larger samples of diabetic patients are needed to enhance our understanding of the relation between diabetes mellitus and depression.

ZIAD KRONFOL
JOHN GREDEN
BERNARD CARROLL

Clinical Studies Unit,
Department of Psychiatry,
University of Michigan,
Ann Arbor, Michigan 48109

TABLE I

Day	Hamilton*	Serum glucose, mg/100 ml			Insulin, u/24 hrs	Imipramine, dose
		8.00 a.m.	4.00 p.m.	8.00 a.m.**		
20	16	223	286	193	48	150
35	6	204	88	163	30	225

* Hamilton depression rating scale.
** Serum glucose concentration at 8.00 a.m. of the following day.

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