schizophrenia. However, the finding of more personality disorder in the families of the schizophrenics is one with which we wish to take issue.

Although the risk of schizophrenia in the parents of schizophrenics is usually less than that for siblings, the parental risk from the Stephens data is less than that seen in the general population, and much less than that seen in the siblings. It has been our experience that when both parents and siblings are interviewed their risks for schizophrenia are similar (Tsuang *et al*, 1974). An explanation for the Stephens results may lie in the frequent diagnosis of schizoid and paranoid personality disorders in the parents.

We take particular issue with the classification of the schizoid (ii) subgroup as a 'personality disorder'. The authors characterize people with this disorder as 'rambling, vague, unrealistic, . . . eccentric and solitary in their personal life'. Certainly there are difficulties in diagnosing schizophrenia in the absence of delusions, hallucinations or clear thought disorder; however, a suspicion of schizophrenia would appear justified in relatives with 'schizoid subgroup (ii)' characteristics. To us, it would appear appropriate to use the term 'suspected schizophrenia' for these relatives, and to include them in the analysis as 'schizophrenia'. In the long run, we feel that a rose by this name not only smells sweeter but is more precisely named.

Interestingly enough, even if the schizophrenic group is broadened along the above lines there may yet remain an excess of personality disorder and heavy drinking in the parents and siblings of schizophrenics.

Data from this Department suggest a possible explanation for such an excess. In an analysis of psychiatric illness in the parents and siblings of 260 process schizophrenics (Fowler *et al*, unpublished), psychiatric disorders in siblings—schizophrenia, alcoholism, affective disorder—were viewed according to the psychiatric disorders in the parents—no psychiatric illness, schizophrenia, alcoholism, other psychiatric illness. Only the following statistically significant associations were noted: (1) schizophrenia in siblings with schizophrenia in parents; (2) alcoholism in siblings with alcoholism in parents; (3) affective disorder in siblings with other psychiatric illness in parents (Table). These data suggest that alcoholism and probably affective disorder are transmitted independently of schizophrenia in these families.

In addition, 49 per cent of the parents with schizophrenia have psychiatrically ill spouses, alcoholism being the most common diagnosis. This reinforces a previous finding that schizophrenics frequently marry alcoholic and personality-disordered individuals (Fowler and Tsuang, 1975). Thus our data suggest that alcoholism and some personality disorders in the families of schizophrenics are more a function of the selective mating of the schizophrenic parent(s) than a biological variant of schizophrenia.

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TABLE

Parental illness and schizophrenia, alcoholism and affective disorder in the siblings of schizophrenics

Illness in parents	Illness in siblings: Schizophrenia +	Schizophrenia −	Alcoholism +	Alcoholism −	Affective disorder +	Affective disorder −
No illness . .	6	325	4	327	6	325
Schizophrenia	6**	27	1	32	2	31
Alcoholism . .	0	34	5**	29	2	32
Other psychiat.	4	118	3	119	9*	113
	16†	504	13	507	19	501

* $P < \cdot 05$.
** $P < \cdot 001$.
† Both parents of one sibling with schizophrenia are ill; therefore, the sibling is counted twice.

PERSONALITY AND DEPRESSIVE ILLNESS

DEAR SIR,

I wish to draw attention to a fundamental misconception in the paper by Serra and Pollitt (*Journal*, September 1975, **127**, p 211). It is an error which has underlain and largely negated the value of uncountable previous research reports and which will no doubt persist into the foreseeable future.

In their paper, the authors attempt to show that the psychic symptoms of depressive illness are largely determined by the underlying personality structure;

by contrast they consider that other symptoms are basic to the disease process of depressive illness and are not modified by personality. In the latter group are insomnia, changes in appetite and weight, decrease (or increase) of libido and diurnal mood variation.

Unfortunately for their case, the authors selected the Maudsley Personality Inventory as the measure of personality. This questionnaire is a very poor indicator of personality when completed by a sick person, for its component scales are constructed from a mixture of trait and state items, and the manner in which these items are completed is heavily influenced by illness. The almost universal finding of falling N scores and rising E scores on recovery from depressive illness attests to this, as does the finding in the authors' own paper of a high correlation between the MPI scores and the severity of illness as determined by the Beck Depression Inventory. The finding of lower, but still significant, correlations between the N and E scores and the various sub-groups of the Beck Inventory which deal with psychological symptoms merely reflects a coincidence of items. Conversely, the lack of significant correlations between the MPI and the authors' 'functional shift' symptoms is related to the virtual absence of such items in the MPI.

The authors state: 'These findings as a whole suggest that the Beck Depression Inventory may be measuring personality factors reflecting the underlying illness process rather than estimating the illness itself.' With probably greater justification this statement could be inverted to read that the Maudsley Personality Inventory, far from measuring underlying personality, is a measure of the sickness itself. The production of personality trait measures based upon self-rating and unaffected by illness has so far defeated all who have attempted it, possibly because they have failed to consider the methodological problems involved. Spielberger, in his State-Trait Anxiety Inventory, probably comes nearest to a solution to the problem by the technique of repeated rating of much the same set of items to indicate the individual's view of his present state as opposed to his usual (premorbid) state; however, even this refinement will fail to distinguish basic personality from illness in a large proportion of the chronically sick.

Until better instruments are devised, research of the type undertaken by the authors must rely upon techniques for assessing personality other than those based upon self-rating.

Finally, I wish to point out that the authors have misrepresented my own contribution to this field of inquiry (Snaith *et al*, 1971, *Psychological Medicine*, **1**, pp 239–47). In that paper we studied patients who had recovered from depressive illness, and using a battery of self-rating questionnaires, including the MPI, we found no significant correlations between the scores and any of the features of the illness which we were considering.

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A POSSIBLE NEW TREATMENT OF WEIGHT LOSS IN AFFECTIVE DISORDERS AND ANOREXIA NERVOSA

DEAR SIR,

Impressed by the reports of the efficacy of intravenous chloripramine infusions in the treatment of various affective disorders (1, 2, 3,), I began using this method of treatment in 1971 (4). Since then, I have treated some 700 cases in this way, including cases of anorexia nervosa and four cases of 'dumping syndrome' following partial gastrectomy.

Increase in weight as a side effect of tricyclic drugs is well documented, and the beneficial effect in anorexia nervosa of the infusion treatment, both on the illness itself and on the associated loss of weight, was described by Lopez Ibor (5). My experience is in agreement with these observations.

I have tried various combinations of drugs in association with the infusions in an attempt to increase its efficacy, and a noticeable improvement occurred with a combination of glucagon, diazoxide, and adenosine triphosphate (ATP). This improvement was apparent in mood as well as in weight, and glucagon or diazoxide were not used unless the latter effect was required. No controlled study has yet been done of the effect on weight, but the improvement was so apparent that I think it of value to report the method. To increase the blood sugar, glucagon is given intramuscularly in doses of 1 mg daily for 7 days or 10 mg on the 1st and 8th day of treatment, or diazoxide 50 mg orally daily, especially in 'dumping' syndromes. Glucagon, like the catecholamines, stimulates the formation of 3'' 5' cyclic adenosine monophosphate (cyclic AMP) (6). In addition to this, ATP is given daily in divided doses totalling 12 mg orally. It cannot be added to the chloripramine infusion, as it causes an unidentified deposit to appear (7).

This combination of tricyclic antidepressive, glucagon and ATP, has been used in 40 patients with no ill effects. The reason these drugs were chosen was because of the reports that in depressive states they were found to be at subnormal levels (8–12). Glucagon, by its action on the cell-bound enzyme adenylcyclase, has a considerable influence on the homeostasis of ATP (13). Munch (14) has reviewed