It is necessary to identify the 'new chronic inpatients' if we are to attend to their needs.

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Is schizophrenia a G-I disease?

SIR: I read with interest the paper by Lambert *et al* (*Journal*, November 1989, **155**, 619–622) and would like to offer a rather different interpretation of their findings.

It may be perhaps that the controls in this study were particularly healthy, in that every one appeared to have a similarly low urinary excretion of ^{51}Cr EDTA. The 24 schizophrenic patients, apart from the two with somewhat raised values, were within the same range and were in fact as normal as the controls. I do not find this result surprising, as all but one were medicated. The exception had previously been tested when taking neuroleptics. We are not told how long this patient had been off drugs. They could perhaps still have been exerting their effects.

Neuroleptic drugs are known to stabilise membranes, including the gut membrane. P. S. Guth (pers. comm.) wrote that when he and his colleagues were first studying chlorpromazine it was thought to be inhibiting its own absorption. This drug is presently being used as an anti-secretory agent in the treatment of cholera and other diarrhoeral conditions. It seems very possible that the neuroleptics taken by the patients in this study could have been the reason for their apparently normal gut permeability.

The Schizophrenia Association of Great Britain (SAGB) had previously considered supporting a study similar to this using ^{51}Cr EDTA, but I was worried about the effects of medication on the validity of the results of such an experiment. The experiment was dropped largely as a result of these doubts.

The SAGB has initiated, and is funding, a programme of research in the Department of Biochemistry in the University of Wales, Bangor, under the supervision of Professor J. W. Payne and Dr J. I. Davies. A gut permeability study is underway with schizophrenic patients, their near relatives, and controls. We have also asked the researchers to investigate the effect of neuroleptics on gut permeability.

It would seem important to investigate gut permeability in never-medicated patients before dismissing too readily the idea that schizophrenia may, for at least a sub-group of patients, be related to coeliac disease. Anecdotally, it has seemed to me that there is a reduction in digestive troubles in well-medicated schizophrenic patients. In the absence of non-medicated schizophrenics, further permeability studies on their near relatives might throw light on the genetic lesion in schizophrenia.

There is a high incidence of coeliac disease among the families of SAGB members in which there is also a patient with schizophrenia. Out of 239 returned questionnaires sent to members, there were 10 cases of coeliac disease, three of which were in the patient and seven in a near relative. The incidence of the disease in the general population is said to be between 1/500 and 1/2000. Were permeability studies to be done in families in which there is both schizophrenia and coeliac disease, it might be possible to identify a sub-group of schizophrenia related in its pathology to coeliac disease. Such patients might respond to a dietary treatment. Certainly I knew one schizophrenic, whose first cousin was a coeliac, who improved greatly on a gluten-free diet. I know also of two families in whom of two siblings, one was schizophrenic and the other coeliac. Unhappily, one of each pair (one coeliac and one schizophrenic) committed suicide.

There is much evidence that members of the SAGB suffered a high incidence of digestive problems before the onset of their schizophrenia. It would seem extremely premature to dismiss lightly the long-held view that schizophrenia is primarily a disease of the digestive system in which the brain is only secondarily affected by the disease process. This view was held at the beginning of the 19th century. It is essential in all gut studies investigating this possibility to beware of the direct effect of neuroleptics in stabilising membranes. The gut may be their chief site of therapeutic action. Who knows?

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More on multiple personality disorder

SIR: Simpson (*Journal*, October 1989, **155**, 565) hypothesised that multiple personality disorder (MPD) is an "iatrogenic, largely culture-bound dis-