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

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References

RUIZ-RIPOLL, I., GARCIA RIBERA, C., HORTAL, M., *et al* (1986) Existe el nuevo paciente de larga estancia? *Sant Pau*, **7**, 126–129.
—, —, —, *et al* (1987) The new long-stay psychiatric patient. Abstract 187. *IV International Congress of Psychiatric Epidemiology*. Madrid: International Federation of Psychiatric Epidemiology.

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-

order, with some resemblances to folie à deux . . ." which is created by clinicians, the media, and the health care system through "selective reinforcement of symptoms". Simpson is but one of a number of clinicians recently who have professed to have seen either none or at the most one patient with MPD, yet find themselves qualified to comment on the diagnosis, aetiology, and treatment of MPD (Levitt, 1988; Fahy, 1988). What has become of the scientific method? Conclusions about such matters should only be made after thoroughly reviewing the literature or after accumulating a robust series of cases.

The history of MPD dates back to antiquity (Ross, 1989). Since 1860 over 700 scientific articles, chapters, books, etc. have appeared on MPD (Coons, 1986–89). Half of this literature has appeared since 1980, when MPD was first recognised as a diagnosis in the DSM–III.

Although there are a few 'media multiples' who derive much secondary gain from their illness as Simpson suggests, this pattern is not common. Most patients with MPD are deeply troubled by their illness and therapy can be very arduous. I've found few clinicians who make a spectacle out of demonstrating their "talented offspring" and showing their "latest cute trick".

Professor Simpson is incorrect when he states that "a very small number of clinicians report the great majority of case reports". The 211 case reports in the English literature have been reported by 180 different clinicians. The International Society for the Study of Multiple Personality and Dissociation has a membership of over 1300 clinicians, predominately in the United States and Canada, and most have seen patients with MPD. I have personally corresponded with numerous clinicians from throughout North America, several Latin American nations, numerous European countries, and Australia who have encountered MPD. Analogues of MPD exist in Africa and Asia. The few clinicians who have reported large series of cases have amassed these over 10–15 years and have large referral areas encompassing several states or provinces.

A recent issue of *Dissociation* (Kluft, 1989) was devoted to the iatrogenic aspects of the diagnosis and treatment of MPD. It is clear that dissociative symptomatology may be worsened by improper forms of treatment. In occasional instances new personalities may form over the course of treatment secondary to new traumatisation. At this time, however, the most common iatrogenic problem is not making a diagnosis of MPD and instituting proper treatment.

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References

COONS, P. M. (1986–1989) Current books and articles. *Newsletter of the International Society for the Study of Multiple Personality and Dissociation*.
FAHY, T. A. (1988) The diagnosis of multiple personality disorder: a critical review. *British Journal of Psychiatry*, **153**, 597–606.
KLUFT, R. P. (ed.) (1989) *Dissociation*, **2**, 59–115.
LEVITT, E. (1988) Questions about multiple personality. *The Harvard Medical School Mental Health Letter*, **4**, 8.
ROSS, C. A. (1989) *Multiple Personality Disorder: Diagnosis, Clinical Features, and Treatment*. New York: Wiley.

SIR: Simpson (*Journal*, October 1989, **155**, 565) has stated that multiple personality disorder is an iatrogenic artifact, and that it does not occur as a natural condition. Neither he nor Fahy (*Journal*, November 1988, **153**, 597–606), upon whom Simpson comments, refer to the substantial evidence accumulated in North America that MPD is a relatively common post-traumatic disorder of dissociative type, linked to severe chronic child abuse.

Rather than disagreeing with Professor Simpson on an ideological level, I would like to point out that these two competing hypotheses about MPD could be tested using the Dissociative Experiences Scale (Bernstein & Putnam, 1986) and the Dissociative Disorders Interview Schedule (Ross, 1989; Ross *et al*, 1989).

If all individuals admitted to an acute care adult psychiatric in-patient unit in Britain or South Africa were screened with the Dissociative Experiences Scale, I predict that about 15% would score above 30. Of these individuals, one-third would meet DSM–III–R criteria for MPD on the Dissociative Disorders Interview Schedule, and one-third would have another dissociative disorder.

Review of the past histories of these individuals with MPD would reveal that they had been in the mental health system for an average of between five and ten years. Only part of their complex symptom clusters would have been documented during this period. The dissociative disorders would not have been diagnosed because of failure to enquire systematically about dissociative symptoms.

In some countries the prevalence of dissociative disorders would vary from that in North America, perhaps due to chronic childhood trauma in the form of war, famine, and natural disaster, or a different prevalence of child abuse. Although an actual calculation would be difficult to make, conceptually the correction factor for the prevalence of dissociative disorders in other parts of the world compared with that in North America would be the ratio of trauma in those countries compared with that in North America. MPD would occur in response to chronic