(Zioudrou *et al*, 1979), to certain brain receptors which normally modulate dopaminergic and cholinergic activity.

Coeliac disease requires abnormal alleles at another locus. This probably codes for increased immunological response to gluten antigens and is in linkage disequilibrium with certain human leucocyte antigen (HLA) loci. These loci occur in most coeliac patients but are as low in frequency in schizophrenics as they are in the general population. Thus most patients with idiopathic schizophrenia do not have coeliac disease, but a small per cent do because they have all the genes for both diseases.

A previous article (Dohan, 1988) presents the evidence for and against my hypotheses. In addition to dietary trials it includes: the extreme rarity of schizophrenia in populations consuming little or no grain; the exceptionally high correlation between rates of wheat and rye consumption and first admissions to mental hospitals of women with schizophrenia ($r = 0.98$, $P < 0.001$); the increased excretion of small peptides, some with opioid activity, by schizophrenic patients (Reichelt *et al*, 1985); the stereotyped behaviours and patterned seizures in rats, hours after intracranial injections of gluten peptides; the changes in schizophrenia-relevant neurotransmitters in cats' brains produced by chronic high-gluten ingestion (Thibault *et al*, 1988).

My article suggests many ways of testing my hypotheses. For example, Bruce *et al* (1985) reported increased transglutaminase activity in gut biopsies of active and remitted coeliac patients which might be important in gliadin binding to gut tissues. This, I postulated, might increase the transcellular passage of glutamine-rich peptides, including exorphin precursors, across the gut barrier. What is the activity of this enzyme/receptor in schizophrenic patients?

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ZIOUDROU, C., STREATY, R. A. & KLEE, W. A. (1979) Opioid peptides from food proteins: the exorphins. *Journal of Biological Chemistry*, **254**, 2446–2449.

SIR: Abnormal intestinal absorption has been suggested to be an aetiological factor in schizophrenia. In an earlier study, the cellobiose/mannitol test, which is a reliable index of intestinal permeability, was carried out on long-stay psychiatric in-patients (Wood *et al*, 1987). Each patient was investigated by duodenal biopsy for the presence of abnormalities of the mucosal morphology of the small intestine. The study showed that 34% of the patients had abnormal absorption which could not be attributed to established bowel disease. Patients who were receiving neuroleptic medication but not anticholinergic drugs were those who most frequently showed abnormal intestinal permeability. These findings were considered to suggest that a proportion of patients with schizophrenia have abnormal gut permeability which might result in increased absorption of molecules which induce psychosis, but which at the same time, protect against the development of Parkinsonian side effects of treatment.

Following these earlier findings, we set out to test the hypothesis that within an otherwise unselected group of chronic schizophrenic patients there are two populations, one taking neuroleptic medication without anticholinergic medication and showing abnormal intestinal permeability, and another taking both neuroleptic and anticholinergic medication but showing normal intestinal permeability. The cellobiose/mannitol test was used in two groups of in-patients with schizophrenia (Research Diagnostic Criteria (RDC); Feighner *et al*, 1972), both of which were receiving neuroleptic drugs but only one of which required anticholinergic drugs. It was difficult to find in-patients fulfilling RDC criteria for schizophrenia who were not taking anticholinergic medication and who could give informed consent. Consequently, only 25 patients were recruited, 16 receiving anticholinergic drugs and 9 not receiving them: we had hoped for larger, more balanced groups. Eleven patients (44%) had abnormal intestinal permeability, but they were evenly divided between the groups. This rejected our hypothesis, albeit in a very small sample.

Five patients with normal tests in Wood *et al*'s (1987) study were re-tested and three had abnormal results. We can offer no satisfactory explanation for this finding. The cellobiose/mannitol test is not significantly affected by extraneous factors such as

gastrointestinal transit times and renal function, and the results cannot be explained by changes in the patients' medication. The fact that three patients with chronic schizophrenia developed abnormal intestinal permeability over time argues powerfully against the latter's role as an aetiological factor. The questions remain as to what the mechanism of abnormal intestinal permeability is, and what influence it has on both the disease process and the efficacy of medication.

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'New long-stay' patients and social deprivation

SIR: Abbott (*Journal*, January 1990, **156**, 133) makes the point that a detailed analysis of the socio-demographic characteristics of catchment areas may reveal associations between accumulation rates of 'new long-stay' patients (admission duration of more than one year) and indices of social deprivation. At present, the Team for the Assessment of Psychiatric Services (TAPS) is evaluating the reprovision of services for patients leaving Friern and Claybury Hospitals. Furthermore, such an analysis has already been performed (Jones & Margolius, 1989) in which the Spearman rank correlation coefficient between the annual rate of accumulation of new long-stay patients and the Jarman-8 index (Jarman, 1983) of social deprivation for their nine districts of residence was 0.82 ($P < 0.01$).

Although this association has not been previously reported, it is not surprising. The association between residential area and incidence of schizophrenia was described in Chicago over 50 years ago by Faris & Dunham (1939) and has been replicated more recently in Bristol and Nottingham (Ineichen *et al*, 1984; Giggs & Cooper, 1987). The Royal College study (Hirsch, 1988) found a correlation of 0.76 between district psychiatric admission rates and Jarman-8 indices in North West Thames. Indeed, a number of other combined deprivation scores such as ACORN, Unit 9, and the Department of the Environment Social Index (but not Standard Mortality Rate (SMR)) correlate equally highly with admission rates (Thornicroft, 1989). The strength of these associations support Hirsch's proposals that the service norms used in planning psychiatric services should be weighted to take explicit account of the extent of local social and economic deprivation.

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Platelet MAO and 5-HT uptake in agoraphobics

SIR: The hypothesised association between neurotransmitter abnormalities and the anxiety-states panic disorder and agoraphobia with and without panic attacks has been the focus of renewed attention since the introduction of the concept with DSM–III (American Psychiatric Association, 1978). The recent report by Flaskos *et al* (*Journal*, November 1989, **155**, 680–685) describes studies of platelet monoamine oxidase activity (MAO) and serotonin (5-HT) uptake in patients with agoraphobia and neurotic depression compared with a control group. Both measurements are relevant to the neurotransmitter hypotheses of these disorders. The findings of elevated MAO activity in agoraphobia are supported by some