et al 1989). A well-informed general practitioner (GP) can perform an interview followed by a physical examination, and if necessary, specific laboratory tests. However, the GP needs a lot of support to be able to do this properly in the chronic psychiatric population.

A more feasible approach therefore for any community mental-health-care worker is to alert oneself to possible signs and symptoms of physical disease whenever a patient is seen. When in doubt, the supervising community psychiatrist or the patient's GP should be consulted. If necessary, a direct contact between the patient and the GP should be established even if this would mean accompanying the patient to the GP's surgery and explaining the problems. This procedure would promote the reintegration of the patient back into the appropriate ambulatory medical service. A close co-operation between the community mental health service and the patient's GP would be more effective, of higher medical quality (clinical assessment, physical examination and specific laboratory tests) and less expensive than the suggested routine checks in the day-hospital setting. This physical screening procedure is satisfactorily in use in our Community Psychiatric Unit.

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Was Hitler a Christian?

SIR: I found myself agreeing with the central anti-racist thrust of Samuel's letter (*Journal*, October 1989, **155**, 568–569) but cannot let pass his extraordinary assertion that "Stalin, Hitler, Mussolini, Franco and Rudolph Hoess were all Christians". The Shorter Oxford Dictionary defines a Christian as a "person believing in, professing or practising the religion of Christ ... a person showing character consistent with Christ's teaching". My nodding aquaintance with 20th century history suggests that none of these figures fit even this wide and non-denominational definition. Stalin was actively anti-Christian while Hitler, Mussolini and Franco saw the Church as a useful institution to be manipulated for political ends, nothing more. I know no details of Herr Hoess' theological views.

Dr Samuel's letter perhaps reveals a common misuse of the word 'Christian' as a synonym for 'Western'. It is inaccurate, misleading and potentially offensive to both Christians and non-Christians.

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Intestinal permeability in schizophrenia

SIR: Lambert *et al* (*Journal*, November 1989, **155**, 619–622) recently reported that the intestinal permeability of schizophrenic patients, determined by urinary excretion of ingested ^{51}Cr EDTA, was not significantly different from that of normal subjects. They concluded, "schizophrenia is, at least in the majority of cases, unrelated to coeliac disease" since the latter shows a highly significant increase in intestinal permeability to ^{51}Cr EDTA.

These authors imply their results are relevant to my hypothesis of the genetic relationship between the two diseases (Dohan, 1988). They overlook the possibility that *some but not all genes* necessary for susceptibility to coeliac disease are also present in those hereditarily susceptible to idiopathic schizophrenia. This possibility was suggested by clinical observations indicating the two diseases occurred in about 2–5% of patients with a primary diagnosis of either disease – at least 10 times as frequently as chance expectancy. As expected from the co-occurrence of the two diseases noted above, two of the 24 schizophrenic patients (8%) studied by these authors exhibited intestinal permeability well within the coeliac disease range. However, no diagnostic studies for coeliac disease were mentioned.

I have hypothesised (Dohan, 1988) that abnormal alleles in both diseases code for enhanced gut-cell receptor activity for the glutamine-rich gluten peptides and that aberrant alleles at two or three loci coding for defective systemic enzymes catabolising gluten peptides are the same in coeliac disease and idiopathic schizophrenia. In addition, I postulate idiopathic schizophrenia also requires a schizophrenia-specific gene. This I suspect causes brain dysfunction because of preferential binding of opioid peptides, exorphins, derived from glutens

(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