admissions for "psychogenic psychoses",[2] presumably including patients who would have been diagnosed earlier as schizophrenic. Since (for example) sugar, coffee, tea, eggs, and fruit were also scarce,[2] similar correlations could be plotted between these foodstuffs and schizophrenia—or, indeed, other psychiatric diagnoses, which also showed wide variations in admission rates during the same period.[2]

Claims for a high incidence of coeliac disease in schizophrenia,[3-11] based mainly on unpublished evidence, have been refuted by controlled studies.[12-14] Moreover, there is no basis for equating behavioural abnormalities in coeliac patients with schizophrenia or autism.[14,15] The only independent study suggesting that gluten is pathogenic in schizophrenia[16] was received with scepticism[17] and was shown to be methodologically and statistically inadequate.[18,19] Other studies were negative.[20-22] The increased incidence of gluten antibodies in sera of schizophrenic patients[7] was not confirmed.[23] An observation by Ashkenazi et al.[24] that about 50% of psychotic patients, irrespective of the diagnosis, had an increased production of leucocyte-migration-inhibition factor after a challenge with certain gluten fractions, is difficult to interpret without data on possible interference by psychotropic medication.

The gluten hypothesis is a modern version of the bygone belief that the cause of schizophrenia lies in the bowel. Diet and purgation have been used intermittently in treatment of schizophrenia for centuries. The "intestinal intoxication" theory of schizophrenia, still popular in the 1920s,[25] has now been rejuvenated as the exorphin hypothesis in order to incorporate the gluten hypothesis into the mainstream of biological psychiatry. Exorphins are exogenous peptides, found in pepsin hydrolysates of wheat gluten and α-casein, with both morphine-agonist and morphine-antagonist properties; "the combination of stimulatory and inhibitory substances present in the peptide fragments of gluten may be responsible for the proposed relationship between wheat gluten and schizophrenia".[26]

The initial enthusiasm for the key-role of endogenous opioids (endorphins) in schizophrenia has been dampened by the neutralising effect of the conflict between two opposing views, seeking the clue to schizophrenia in either an excess or a deficit of endogenous opioids. Are the opioids from bread, milk, and butter to be the next open-sesame to schizophrenia?

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ACQUIRED IMMUNODEFICIENCY IN HAEMOPHILIA

SINCE our editorial of Jan 22, reports of acquired immunodeficiency syndrome have continued to come in, and the world total now exceeds 1200. Advice from the Centers for Disease Control is essentially that sexual contact with known AIDS patients should be avoided; that homosexual males should limit the number of their partners; and that steps should be taken to exclude high-risk subjects from blood or plasmapheresis panels—a precaution already adopted by some of the commercial organisations collecting and processing blood products.[1] In the March issue of *Annals of Internal Medicine* three articles describe further cases of AIDS, and a fourth T-lymphocyte abnormalities, in patients with haemophilia. An accompanying editorial by White and Lesesne[2] suggests that the T-cell population abnormalities commonly seen in haemophilia may be the submerged part of an iceberg of which AIDS (they mention 7 cases altogether) is the clinically obvious "tip".

Longitudinal studies of patients with abnormal T cell ratios would be necessary to test this hypothesis and to determine the risk of subsequent immunodeficiency disease. Recognising that AIDS has developed in only a few out of the many haemophiliacs receiving large-pool factor VIII concentrates, White and Lesesne discuss additional predisposing agents, and cite repeated attacks from hepatitis viruses as a possible precursor. Because of the (limited) evidence that cryoprecipitate is free of the "transmissible agent", and that the greater the exposure to concentrates the greater the risk, these workers have adopted a series of preventive measures in their own haemophilia practice. Elective surgical procedures have been cancelled, doses of factor VIII reduced, and, where possible, patients switched from concentrate to cryoprecipitate therapy. An editorial in the *New England Journal of Medicine*[3] goes further. On the strength of 5 reported haemophilic cases Desforges suggests that it is time to consider giving up home therapy programmes which are reliant on factor VIII concentrates "even though we may not have enough evidence to demand such a radical change".

Two aspects of this surge of interest in an as yet unexplained syndrome must, at this stage, be clearly separated. Firstly, the finding of alterations in lymphocyte populations, although reported as common may not be causally related.[4] Secondly, the recognition of disease in a few haemophiliacs does not necessarily reflect the tip of an iceberg. Of course we can expect to see side-effects from transfusion therapy with plasma collected from many thousands of donors. But if the explanation of AIDS were that easy, even allowing for a transmissible agent introduced in the late seventies and with a long incubation period, the syndrome would surely have affected greater numbers of either American or West German recipients, who have received far more factor VIII concentrate transfusions of United States origin than have haemophiliacs in other developed countries.[5] The links suggested by the American workers must be regarded as not proven. Whilst careful surveillance must continue, the reported cases do not constitute a strong argument for a change in treatment policy.

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