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Correspondence

Letters to the Editor should not exceed 500 words.

Gastric and Oesophageal Cancer in the Welsh

SIR,—Your leading article (7 June, p. 590) rightly emphasizes the effects of both genetic and environmental factors in the causation of disease, but in my opinion it draws too strong an antithesis between the two types of cause. Unless a disease has been shown to be caused exclusively and invariably by a particular gene or a particular environmental factor, not only must the existence of both types of cause be considered, but also the possibility of close interaction between them. In some cases an understanding of the interaction may be more important than a knowledge of either factor considered by itself.

A recently investigated case in point is that of kuru, a uniformly fatal neuropathy confined to a small group of tribes in New Guinea. For some 20 years the relative merits of an infective and a genetic aetiology were argued. As a recent review article[1] shows, it is due to a specific infective agent, probably a virus, which has been transmitted to primates—but there is strong evidence that, because of a genetic factor, only members of these particular tribes, of all those who have been exposed to it, become infected or develop symptoms (the main type of "exposure" being probably brain cannibalism as an act of respect to the dead).

In the case of Wales and the Welsh, there are undoubtedly marked contrasts, both of environment and heredity, with England and the English. In a series of papers Watkin[2] has shown contrasts in blood group frequency, which he has well brought out by using Welsh surnames and a knowledge of the Welsh language as criteria of ancestry, the Welsh having a markedly higher frequency of group O, and a lower one of group A, than the English. At the same time certain diseases are much commoner in Wales than in most parts of England, and Ashley in particular has demonstrated this in detail in an admirable series of papers, to some of which you refer. As you rightly infer, further research on possible environmental effects is needed.

The possibility that the high frequency of carcinoma of the stomach is related to blood groups is not, however, ruled out as you suggest. It was the high frequencies of both carcinoma of the stomach and blood group O in North Wales which led the late Professor I. Aird[3] to investigate the blood groups of cases of the disease, and to the initially surprising finding of a statistical association between group A and carcinoma of the stomach, an association which has now been confirmed in patients of many different ethnic groups. It is noteworthy, however, that the Icelanders have even higher frequencies both of carcinoma of the stomach[4] and of group O[5] than the Welsh. These observations on the two populations may be totally unrelated, or if, as some suppose, the original Icelanders came mainly from the British Isles it may be that the blood group constitution, and the liability to carcinoma of the stomach, are genetically separate manifestations of ancient British heredity.

If, however, carcinoma of the stomach is due to an interaction between heredity and environment, and since some O people and by no means all A people get the disease, persons of all blood groups must be in varying degrees susceptible. Thus where the environment favours the disease it will tend to eliminate selectively persons of group A. It must be admitted that deaths from carcinoma of the stomach take place mainly after the end of the reproductive period, so that any selective effects are likely to be relatively slight. It is, however, permissible to speculate on whether such selection may be a part of the cause of the high frequency of group O in Wales and Iceland. In a parallel case where selection has been clearly demonstrated, that of the haemoglobin variants and infection with falciparum malaria,[6] the gene which is associated with susceptibility to the disease, namely that for normal adult haemoglobin, is reduced in frequency in areas where the environment favours the infection, while the allelic gene, that for haemoglobin S, which confers protection, is raised in frequency.—I am, etc.,

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Rhesus Isoimmunization and Therapeutic Abortion

SIR,—With the present discussion on the dangers of therapeutic abortion the following case is of interest.

The patient was first seen in June 1968 at the twelfth week of pregnancy, although she was unsure of her dates, the cycle being 28–35 days. On examination the patient was found to be normal apart from the pelvis being of slightly reduced size. The history of an induced abortion by curettage at the twelfth week of pregnancy exactly two years previously was given. There had been no other pregnancies. No blood transfusion had been given at this or any other time. The result of the test for Rh antibody titre was awaited with interest. The patient was found to be group A Rh-negative, and the serum contained anti-D antibodies, the titre being 2 saline and 8 with the Coombs technique. While pregnancy was otherwise normal, antibody titres were reported as follows:

34 weeks	Anti-D titre	1 saline 128 Coombs technique
36 weeks	Anti-D titre	0 saline 512 Coombs technique
37 weeks	Anti-D titre	1 saline 1,000 Coombs technique

The patient's husband was found to be Group B Rh-positive and the probable genotype R.1. R.1.

The patient was admitted on 3 December 1968 and amniocentesis attempted on two occasions but without success. In view of the rising titre of antibodies (the result of 6 December was not available), induction of labour was carried out on 6 December at 7 a.m. and labour started one and a half hours later. Analgesia