Of the 252 tumours, 37 were Bloom grade I and also node negative. In this small subgroup those patients with oestrogen-receptor-positive grade I tumours appear to have a more favourable prognosis than those with oestrogen-receptor-negative grade I tumours. Life table analysis shows that in the group of 22 patients who were lymph-node negative and oestrogen receptor-positive, 100% were disease free at 60 months, whereas in the 15 who were node negative and receptor negative only 55% were disease free. (Survival figures are not given since too few events have occurred to allow meaningful assessment.)

While we accept that tumour grade is a useful prognostic factor, histological grading is inevitably subjective; and this may well explain the discrepancies that exist between the results of different centres. By contrast, oestrogen-receptor analysis is a more objective measurement, and in our hands is independent of tumour grade based on the criteria described by Bloom and Richardson. This finding has perhaps the advantage that both tumour grade and oestrogen-receptor status can be combined to provide a more accurate prediction of prognosis than either factor used on its own.

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Changes in glycosylated haemoglobin after oral glucose load

Sir,—We read with interest the paper by Dr N J Scofield and others (3 October, p 877) describing changes in glycosylated haemoglobin (HbA1c) up to 30 days after an oral glucose tolerance test. While HbA1c values remained unchanged during the test the authors noted a significant increase of HbA1c 10-30 days after the test. We would like to report our own observations during and after continuous glucose infusion. After an overnight fast we administered 10 g/h glucose per kg body weight to 10 healthy volunteers for six hours. Blood glucose and the total fraction

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Diagnosis and treatment of lactose intolerance

Sir,—I have read Dr Anne Ferguson’s leading article (23 October, p 1463) on this subject with interest. May I, however, register a mild protest on one small point? Dr Ferguson apparently attributes the persistence of lactose intolerance in Europe and North America and its cessation after early childhood in many Asians and Negroes to genetic differences. How is the gene mutation responsible for persistence to be visualised? Did it take place in a large number of members simultaneously or in a single remote ancestor? In the latter case the ancestor in question could not have been very far removed from Adam and Eve.

Indications are that the change to lactose tolerance in adults is of multicentric origin. Man has colonised many inhospitable habitats with little if any vegetation suitable for human consumption. Under such conditions he had to become either a hunter or a herdsman, in the latter case usually living on both the meat and the milk of his animals. Such conditions arose in such widely separated parts of the world as parts of Africa and Antarctica. In all such cases tribesmen differ as the Khirgis, the Masai, and the Lapps must have acquired the ability to digest milk in adult life. There is a strangely similar change in adult lactose tolerance: it is acquired when needed, abandoned when not needed.

Apart from deserts, semi-deserts, and similar habitats, lactose tolerance probably has a negative survival value. Why should it not be maintained throughout life when, under natural conditions, they are needed only in infancy? Apart from that, many of us suspect that milk is atherogenic. In a recent epidemiological survey I have found strong correlation between the consumption of unfermented milk proteins and mortality from coronary disease. For instance, the highest known consumption rate of such proteins, in Finland, is associated with the highest known male mortality from heart disease. In Germany, Yugoslavia, and Japan, where the consumption of unfermented milk proteins is approximately a half, a quarter, and a tenth of that in Finland, male coronary mortality is also approximately a half, a quarter, and a tenth of the Finnish rate. To give another example, negative correlation between lactose intolerance and coronary mortality was reported by Segal. The only example with which this paper is concerned, is that of people with the milk of their ancestors, who may have acquired the ability to digest milk in adult life. It is important to note that while the above-mentioned examples of lactose intolerance is associated with the ability of digesting milk in adult life. It is important to note that while the above-mentioned examples of lactose intolerance seems to be an acquired trait, there is evidence of a genetic factor involved as well.