LETTERS TO THE EDITOR

Dietary factors associated with duodenal ulcer

Sir,—The results of two recent studies showed that duodenal ulcer was associated with low dietary linoleic acid and high refined sugar intake.1 2 In India and Bangladesh Tovey and colleagues3 noted a high incidence of duodenal ulcer from areas in which milk or polished rice, sorghum, or yams are staples, and a low incidence where unrefined wheat, certain millets, or pulses are staples. The protective factor could be either a lipid or a liposoluble substance.4 It has been hypothesised that in Western populations the falling incidence of duodenal ulceration may be related to the increased consumption of essential fatty acids, particularly linoleic and arachidonic acids.5 In Africa the frequency of duodenal ulcer is rising in urban populations with their transition in diet and other aspects of lifestyle. Rises in frequency occur in the 'Western' type of duodenal ulcer, with haemorrhage and perforation being the major complications.6 This picture contrasts with the complication of gastric outlet obstruction resulting from the 'stenosing' type of ulcer, which occurs in rural, non-Westernised areas of Africa.7

In Soweto (population about 2 million) at Baragwanath (3200 beds, 200 inpatients annually) in 1983 there were 236 patients with duodenal ulcer (4.5 per 1000 hospital admissions), a considerable increase, even allowing for population growth, from 1956 when only 16 patients were admitted for treatment.8 In 1964–71 Bremer reported 87 black patients among 31 500 surgical admissions to the Johannesburg Hospital (2.8 per 1000 hospital admissions).9 The traditional dietary meal was (and still is in parts) high in cereals, beans, and 'spinaches', with meat and dairy products eaten infrequently. It was a diet low in fat (supplying 10–15% energy) and sugar (5–8% energy) and high in dietary fibre only a quarter of that in an unrefined maize meal.10 But consumption of maize meal has fallen. Although bread consumption has increased, most of the black population favour white bread, despite it being dearer than the more subsided brown bread. Bean consumption has halved compared with the past. There is no likelihood of a reversion to the past; the black population are avid for more meat, more fat, and less carbohydrate in foods. There is likely to be little restraint in increases of diseases of prosperity.11 Currently there are only slight rises in non-infective bowel diseases,12 but, as stated, large increases in the mortality from patients with duodenal ulcer. To keep a perspective, it must be remembered that duodenal ulcer is of multifactorial aetiology, with Helicobacter pylori, stress, peptic, acid, and other factors sharing responsibility. Spiro urged physicians to recognise 'that an ulcer is not a disease or an entity ... but a manifestation of many different processes.'13

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3 Tovey F, Peptic ulcer in India and Bangladesh. Gut 1964: 5: 399–405.


5 Jayaraj A, Tovey FJ, Clark CG. Possible dietary protective factors in relation to the distribution of duodenal ulcer in India and Bangladesh. Gut 1980; 21: 1068–76.


15 Segal I, Walker AR. Low fat intake with falling fibre intake was associated with rarity of non-infective bowel diseases in blacks in Soweto, Johannesburg, South Africa. Nur Canc 1986; 8: 185–91.


Interleukin-2 receptor expression

Sir,—The study of Choy et al1 in which they have shown interleukin-2 receptor (IL-2R) expression by lamina propria T cells and macrophages in inflammatory bowel disease, is largely in agreement with the previously published study.2 In our study IL-2R positive cells in the lamina propria had morphological appearances of lymphocytes and macrophages and in many sections were most prominent in the superficial lamina propria, especially in the submucosal (subepithelial) region. In normal colonic tissue only occasional weakly positive cells were seen with macrophages usually in the superficial lamina propria. We have also reported IL-2R positive cells in and around the germinal centre and dome regions of Peyer's patches.3 Interestingly, cells with dendritic morphology in the interfollicular region which are strongly HLA-DR, HLA-DQ, and RFD1 positive were not labelled by antibodies to IL-2R nor by macrophage-specific antibodies EB11 and 3C10.4 The expression of IL-2R by lamina propria cells in inflammatory bowel disease was confirmed by studies on isolated cells.5 Many of these cells were positively identified as macrophages by their capacity to phagocytose opsonised zymosan (no other mononuclear cell would be expected to do so). We also showed that the IL-2R positive macrophages were activated as shown by their capacity to release oxygen radicals. It was therefore incorrect of Choy et al to state that we did not establish the phenotype of the IL-2R positive cells in our study. In vitro stimulation with interleukin γ and lipopolysaccharide of mononuclear cells isolated from normal colonic mucosa did not increase the proportion of IL-2R positive cells. This led us to suggest that these cells (in inflammatory bowel disease mucosa) were derived mainly from the peripheral circulation. In our study we did find significant differences between ulcerative colitis and Crohn's disease in the cell populations that were IL-2R positive. The differences described by Choy et al could be accounted for by the fact that they were only able to study 'intrinsic' host cells such as the lamina propria—where the IL-2R positive cells were scattered. Clusters of macrophages have been shown in Crohn's disease which are labelled by the mononuclear antibody RE.4,5 A Re-positive RFD1 positive macrophages have been shown to be found in subepithelial aggregates may go on from granulomas. Epithelioid cells in fully formed granulomas have also been shown to label with antibodies to IL-2R.6 It is possible, therefore, that in the study by Choy et al the IL-2R positive macrophages in the Crohn's disease patients were largely aggregated. Thus there may be no overall significant difference in the proportions of IL-2R positive lymphocytes and macrophages in the mucosa in the studies by Choy et al and our study of Crohn's disease but the distribution of these cells between the two may be different.

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