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 colleagues[3 4] noted a high incidence of duodenal ulcer from areas in which milled 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.[5] 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.[6] 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.[7] 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.[8]

In Soweto (population about 2 million) at Baragwanath Hospital (3200 beds, 52 200 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 34 patients were admitted for treatment.[7 9] In 1964–71 Bremner reported 87 black patients among 31 500 surgical admissions to the Johannesburg Hospital (2·8 per 1000 hospital admissions).[10] The traditional diet of rural blacks was (and still is in many parts[11]) 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 fibre 30–40 g or more daily. Nowadays, in urban areas, in contrast to the past,[12] fat intake has risen (supplying 25–30% or more of energy) and sugar intake also (supplying 10% or more of energy), whereas fibre intake has fallen to that of the white population, perhaps even lower, of 10 g or less daily. The fall in linoleic acid intake now compared with the past derived from cereal products is considerable. Not only is the concentration in superrefined maize meal only a quarter of that in unsifted maize meal,[13] 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 subsidised 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.[14] Currently there are only slight rises in non-infective bowel diseases,[15] but, as stated, large increases in the number of patients with duodenal ulcer.

To keep a perspective, it must be remembered that duodenal ulcer is of multifactorial aetiology, with *Helicobacter pylori*, stress, pepsin, 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.'[16]

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Interleukin-2 receptor expression

SIR,—The study of Choy *et al*,[1] 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 our 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 (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 EBM11 and 3C10.

The expression of IL-2R by lamina propria cells in inflammatory bowel disease was confirmed by studies on isolated cells.[2] 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 interferon 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 studies we did not 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 selected areas of 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 monoclonal antibody RFD9.[4 5] Isolated RFD9 positive macrophages have been shown to be activated[6] and these aggregates may go on from granulomas. Epithelioid cells in fully formed granulomas have also been shown to label with antibodies to IL-2R.[7] 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 ulcerative colitis and Crohn's disease but the distribution of these cells between the two may be different.

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