In strong contrast is the experience of Japanese immigrants to Hawaii and California. With changes in lifestyle, including a rise in fat and a fall in fibre intake, these people soon experience tremendous increases in the occurrence of polyps and colon cancer. Indeed, first generation immigrants attest almost double the frequency of sigmoid colon cancer and of rectal cancer compared with the rates for their white intake (10-15%).

We postulated that it may be possible to eat a high fat (and a low fibre) diet with impunity provided one's colon is sufficiently acidified. What factors regulate faecal pH? How important is diet? How influential is the constitutional factor? What avoiding action can be taken? In South Africa, even among traditionally living rural blacks on a low fat, relatively high fibre diet, there are wide ranges in numerous metabolic parameters that are only partially explicable by individual dietary differences. We found that the black schoolchildren of similarly structured socioeconomic level and village environment, have unexpectedly wide ranges in blood glucose, serum protein, albumin, cholesterol, and haemoglobin concentrations, and also in bowel behaviour and faecal pH. Serial studies have confirmed that despite day to day and weekend changes in food intake and composition, alterations in a black individual's faecal pH were slight. Typical sequences were 5.6, 5.7, 5.5, 5.7-5.6, and 6.6, 5.9, 5.6. This behaviour suggests that there is an important constitutional determinant in faecal pH and other metabolic parameters.

To further understanding, faecal pH studies were undertaken on series of 20 rural and urban black children and white children. Faeces were collected for three interrupted sequences of three periods. Each individual's fibre intake was also estimated. For boys and girls combined, mean pH values were – rural and urban blacks, 5.3-5.5, and 5.4-5.0 respectively and whites, 6.81. In the white group, 70% had pH values of 5.5-5.0 and 5.4% were more than 5.5. In the latter, mean (SD) intradintrial coefficients of variation were 5.6-2.5% and 1-2.1%, and in the white group 3.9-1.3%. Interindividual coefficients were larger, 6.8% and 7.8% and 6.7%, respectively. While there were inverse associations between faecal pH levels and dietary fibre intake, values did not reach significance (ρ<0.05).

Locally, there is a puzzling situation in that frequencies of chronic bowel diseases in urban blacks are still low, as are their faecal pH values, despite their now relatively low daily fibre intake. This findings indicate that maybe their sugar intake contributes to the maintenance of blacks' lower faecal pH value, protecting them from colon cancer and other chronic bowel diseases.

Molecular form of faecal α1-antitrypsin in patients with Crohn's disease.

Sir,—Boege and Fischbach recently reported the results of a qualitative study of faecal α1-antitrypsin (α1AT) in healthy control subjects and patients with Crohn’s disease. They found, as we did previously, that α1AT was present in faeces in a qualitatively anomalous form. However, their results differ dramatically from ours. They showed proteolytic α1AT fragments (M, 5, 3 to 42000) as well as polymers in faeces, with a similar distribution in controls and patients with Crohn’s disease. We have shown conversely that α1AT was present in faeces in two main biochemical forms, M, 38000 and 50000 respectively. The α1AT-M, 50000 was, in our experience, significantly associated with active Crohn’s disease (activity index >150). Furthermore, we recently showed that the difference between the two forms of α1AT was related to a different carbohydrate moieties.

In order to understand further the discrepancies between our results and those of Boege, we tried to characterize α1AT fragments in the stools of patients with Crohn’s disease. We were able to find these fragments occasionally, and only as traces. Furthermore, these traces disappeared by improving the specific immunological and biochemical detection by using the Perini’s method. Similarly we never visualised in serum the component with M, 20000 described by Boege et al. Our method detected in both serum and plasma a unique band exhibiting the immunoreactivity of α1AT (M, 54000). Differences in methodology might explain these discrepancies between our results and those of Boege et al.

Moreover, we think that the hypothesis of Boege et al. of claiming that the different α1AT fragments might be the result of hindering of proteolysis by α1AT in faeces, is not a valuable one. Indeed, it is well established that α1AT in faeces has lost its antiproteolytic activity; this suggests that other proteases, including α1AT are widely present in the alimentary tract.

In fact, some of the bands in Figure 1 of Boege et al. (article lines 6, 7, 10, 12, and 13) from the top) could actually be α1AT-M, 51000 that has not been identified correctly. Unfortunately the authors did not report on the Crohn’s disease activity indices of the patients corresponding to these samples, thus making interpretation difficult.

In conclusion, α1AT is present in faeces in two main forms: unglycosylated α1AT-M, 38000 and in glycosylated α1AT-M, 51000. At present we do not know the significance of these.

We disagree therefore with the statement of Boege et al. that ‘faecal α1AT can hardly be used as a diagnostic tool,’ even if the clinical usefulness of various biochemical forms of α1AT in faeces remains to be elucidated.

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