LETTERS TO THE EDITOR

Faecal pH and colon cancer

SIR,—Thornton has proposed that a high colonic pH promotes co-carcinogen formation from bacterially degraded bile acids or cholesterol, a procedure inhibited at a faecal pH lower than 6·5.[1] He cites the low risk of African blacks, who, characteristically, have a low faecal pH and among whom chronic bowel diseases (appendicitis, diverticular disease, colon cancer) are relatively rare.[2-4] Thornton also noted that vegetarian Seventh Day Adventists, at reduced risk of large bowel cancer, have a lower faecal pH than patients with colorectal cancer.[5] He 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 have found that rural black schoolchildren, of closely similar 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, and 6·0, 6·3, 5·9, 6·0.[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 day periods. Each individual's fibre intake was also estimated. For boys and girls combined, mean pH values were – rural and urban blacks, 5·95, and 6·04 respectively and whites, 6·81. In the white group, 70% had pH values of 6·5 or above; in the black groups proportions were far lower at 5·0%. In the latter, mean (SD) intraindividual coefficients of variation were 5·6 (2·5)% and 4·1 (2·1)%, and in the white group 3·9 (1·5)%. 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 ($p>0·05$).

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 (10–15 g). Evidence indicates that maize, their staple food, is malabsorbed.[7] In this context, starch intake may matter as much as fibre intake in terms of protection from cancer. Presumably this malabsorption contributes to the maintenance of blacks' lower faecal pH value, protecting them from colon cancer and other chronic bowel diseases.

In strong contrast is the experience of Japanese immigrants to Hawaii and California.[8] 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 attain almost double the frequency of sigmoid colon cancer and of rectal cancer compared with the rates for their white host neighbours. No data were given on immigrants' faecal pH values.

Just as a reduction of high serum cholesterol contributes to the avoidance of coronary heart disease, so a fall in the faecal pH value could contribute to the avoidance of bowel cancer. In brief, people who are familially prone and whose faecal pH is high should be advised to eat more plant fibre to decrease their faecal pH value. Recent evidence showing regression of adenomatous polyps after a high fibre diet lends support to this view.[9]

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Molecular form of faecal α_1 antitrypsin in patients with Crohn's disease

SIR,—Boege and Fischbach recently reported the results of a qualitative study of faecal α_1 antitrypsin (α_1AT) in healthy control subjects and patients with Crohn's disease.[1] They found, as we did previously,[2] that α_1AT was present in faeces in various molecular forms. However, their results differ dramatically from ours. They showed proteolytic α_1AT fragments (M_r 5 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 α_1AT was present in faeces in two main biochemical forms, M_r 38000 and 51000 respectively.[2] The α_1AT-M_r 51000 was, in our experience, significantly associated with active Crohn's disease (activity index >150).[2-4] Furthermore, we recently showed that the difference between the two forms of α_1AT was related to a different carbohydrate moiety.[5]

In order to understand further the discrepancies between our results and those of Boege, we tried to characterise the proteolytic α_1AT 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 specificity of immunological detection by using the Perini's method.[6] Similarly we never visualised in serum the component with M_r 20000 described by Boege *et al.*[1] Our method detected in both serum and plasma a unique band exhibiting the immunoreactivity of α_1AT (M_r 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 α_1AT fragments might be the result of hindering of proteolysis by α_1AT in faeces, is not a valuable one. Indeed, it is well established that: (i) α_1AT in faeces has lost its antiproteolytic activity[7]; and (ii) numerous proteases not inhibited by α_1AT are widely present in the alimentary tract.[8]

In fact, some of the bands in Figure 1 of Boege's article (lines 6, 8, 10, 13, 15–17 . . . from the top) could actually be α_1AT-M_r 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, α_1AT is present in faeces in two main forms: unglycosylated α_1AT-M_r 38000 in controls and glycosylated α_1AT-M_r 51000 in most patients with active disease.[2-4] We disagree therefore with the statement of Boege *et al* that 'faecal α_1AT can hardly be used as a diagnostic tool,' even if the clinical usefulness of various biochemical forms of α_1AT in faeces remains to be elucidated.

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