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 attain almost double the frequency of sigmoid colon cancer and of rectal cancer compared with the rates for their white intake. The differences 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 faecally 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.

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Molecular form of faecal α, anti-trypsin in patients with Crohn's disease

SIR,—Boege and Fischbach recently reported the results of a qualitative study of faecal α, anti-trypsin (α,AT) in healthy control subjects and patients with Crohn's disease. They found, as we did previously, that α,AT was present in faecal extracts of patients and control subjects. However, their results differ dramatically from ours. They showed proteolytic α,AT fragments (M, 5 to 42000) as well as polymers in faeces, with an similarity in controls and patients with Crohn's disease. We have shown conversely that α,AT was present in faeces in two main biochemical forms, M, 38000 and 51000 respectively. The α,AT-M, 51000 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 α,AT was related to a different carbohydrate moieties.

In order to understand further the discrepancies between our results and those of Boege and Fischbach, we tried to characterize the α,AT 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 radioimmunoassay 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 α,AT (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 α,AT fragments might be the result of hindering of proteolysis by α,AT in faeces, is not a valuable one. Indeed, it is well established that (α,AT in faeces has lost its antiproteolytic activity; it seems that these fragments are not more degraded by α,AT are widely present in the alimentary tract.

In fact, some of the bands in Figure 1 of Boege et al's article (lines 6, 7, and 8 from the top) could actually be α,AT-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, α,AT is present in faeces in two main forms: unglycosylated α,AT-M, 38000 and in glycylated α,AT-M, 51000 in most patients with Crohn's disease. We disagree therefore with the statement of Boege et al that 'faecal α,AT can hardly be used as a diagnostic tool', even if the clinical usefulness of various biochemical forms of α,AT in faeces remains to be elucidated.

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