

in an inversely proportional manner according to the severity of the disease'. In fact, the decrease in polyunsaturated fatty acids as the disease activity increases occurred for both series, although it was more noticeable for n6 polyunsaturated fatty acids. As a consequence our hypothesis was that in inflammatory bowel disease an increased polyunsaturated fatty acid biosynthesis coexists with an increased polyunsaturated fatty acid consumption, the second being associated with the disease activity. The increased polyunsaturated fatty acid biosynthesis would be more noticeable in the n3 series as these fatty acids have the highest affinity for desaturases. Hyperconsumption, although occurring in both polyunsaturated fatty acids series, is more evident in n6 products probably because of an increased arachidonate derived eicosanoid production. This hypothesis seems to be further supported by data from recent studies in patients with non-active inflammatory bowel disease.

Of course, it will be interesting to confirm these findings in different plasma lipid fractions, as well as in colonic mucosa. We are analysing these data, which hopefully will be reported soon. Nevertheless, as far as plasma long chain polyunsaturated fatty acids (mainly n3) are concerned, their concentration in total lipids mainly reflects that found in phospholipids, as most of them are bound to this fraction.<sup>2,3</sup>

The dietary habits of the patients in our study were similar to that of healthy controls (standard Western diet). This type of diet contains negligible amounts of n3 polyunsaturated fatty acids (less than 1% of the total fat).<sup>4</sup> In addition, most of the patients included were in hospital because of moderate to severe attacks of inflammatory bowel disease. Most of them were anorectic and tended to decrease their food intake rather than change to different types of food. No patient had been on artificial nutritional support before plasma sampling was performed.

Although a 14 hour overnight fast might theoretically be a source of error, it is generally assumed that this condition provides an easily reproducible state of metabolic equilibrium. In fact, approximately 15 hours after the last meal there is a progressive decrease in carbohydrate oxidation and a rise in fat oxidation. Increased lipolysis in adipose tissue and fatty acid mobilisation are seen whereas, in the liver, fatty acid synthesis is progressively replaced by fatty acid oxidation and ketone body production.<sup>5</sup> Therefore, longer fasting would lead to misleading results.

We look forward to seeing the findings reported by Belluzzi *et al*<sup>6</sup> published in full as it is difficult to draw real conclusions from an abstract. Data on the location and extent of the disease, bowel resections, nutritional state, and other factors liable to lead to fat malabsorption would be of utmost interest as in the patients described there is a decrease in essential precursors – that is, linoleic acid, which may account for the deficiency seen in long chain polyunsaturated fatty acids. In fact, similar data were also reported by Färkkilä *et al*<sup>7</sup> in plasma lipids from a series including many Crohn's disease patients with bowel resection. So, the findings by Belluzzi *et al*, by contrast with our results, closely resemble the pattern of essential fatty acid deficiency.

Certainly, our results may be interpreted in different ways. On the one hand, as n3 polyunsaturated fatty acids are increased, it would not be necessary to supply them in increased dietary amounts. Conversely, a plasma long chain n3 polyunsaturated fatty acid increase might be seen as an unsuccessful

attempt to prevent an excessive production of arachidonate derived eicosanoids. In such cases the slight clinical response seen when fish oil is given, despite modifying eicosanoid production, could suggest that the amount of n3 polyunsaturated fatty acids given should be increased. All these data, however, provide an attractive insight into the pathogenesis of inflammatory bowel disease, in which the true role of fatty acid treatment also has to be investigated further.

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#### Open access gastroscopy

EDITOR, — Dr Bramble and colleagues describe a most efficient and well run open access endoscopy service (*Gut* 1993; 34: 422–7). Even they, however, only achieve a mean waiting time of 17 days for open access endoscopy. Most of us would fair far worse and therein lies one important problem of open access endoscopy. Do we deprive out symptomatic patients of effective ulcer healing treatment for 17 days so as to obtain a 'pure' endoscopic diagnosis but subject patients to 17 days of unnecessary symptoms and risk of complications, or do we treat patients in the knowledge that if an ulcer is present it may well have healed by the time the patient has an endoscopy, especially – as is increasingly so – the general practitioner has prescribed a proton pump inhibitor.

A short outpatient visit in a general clinic or a specialist dyspepsia clinic would seem a better solution. General practitioners can be encouraged to treat patients as they see fit and the patients seen on treatment. If still symptomatic and, if it is appropriate, they can have an endoscopy immediately. If asymptomatic they can be asked to complete their treatment and arrangements made for the patient to book themselves in for endoscopy by telephone if and when their symptoms next occur before starting treatment. Additional investigations can be arranged and the patient counselled as to the likely result of the endoscopy; normal endoscopy still being the single commonest finding. In our experience with this system<sup>1</sup> 35% of patients will be spared endoscopy.

We found 68% of patients that had an endoscopy preferred this system to open access endoscopy despite the need for two hospital visits and if those spared endoscopy are taken into account 81% preferred the clinic appointment first.

Pressure from general practitioners to set up open access endoscopy is considerable. We feel a clinic appointment first, however, is a more logical solution and the one favoured by the patient. The endoscopies saved may also make it more cost effective.

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#### Reply

EDITOR, — We are grateful for the opportunity to respond to the comments of Drs Trewby and Saunders. There is a basic misunderstanding in their letter. Our provision of an efficient open access gastroscopy service does not in any way deprive patients of effective treatment. We have not made any attempt to discourage general practitioners from treating patients with dyspepsia on clinical grounds. Our guidelines point out that it is appropriate to refer patients for investigation if it is felt that a diagnosis is important before treatment is given or if this has happened and there has been a failure to respond after a reasonable time. The general practitioners in our district have a range of options for patients with dyspepsia and almost one in three of dyspepsia referrals are still by standard letter to a targeted consultant. The type of service described by Drs Trewby and Saunders is still available in Middlesbrough alongside the open access system. (In the case of open access endoscopy, the general practitioner has a further choice of asking for 'report only' or giving the endoscopist discretion about treatment and further management. 'Report only' is specified on about 50% of open access endoscopy referral forms.) We are currently setting up a pilot scheme with a number of GP practices, which will allow them to gain direct access to our open access endoscopy computer scheduling system. This will mean that the general practitioner can give our system the details and obtain the booking and instruction sheet while the patient is still present. It is quite likely that this will bring the time from request to 'investigation' down to about 12 days.

There is no basis to an assumption that screening patients through a specialist hospital clinic will have much effect on the results of endoscopy. The proportion of different abnormalities found on our open access endoscopy lists is very similar to those lists generated from patients seen in clinic and to those published figures in other series. We are developing the software to permit us to send regular feedback to our general practitioners about their use of the open access endoscopy service. This will be similar to the information they receive regularly about their prescribing practice. It will be possible for them to see how they compare with their peers in terms of number of referrals, age, sex, and findings. General practitioners already deal with most cases of dyspepsia that they see.