Letter

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Reply

EDITOR.—We thank Mr Chapman for his comments regarding our paper and are grateful to the editor for the opportunity to respond to the points raised.

Chapman and co-workers have attracted a lot of interest on this point, he has correctly identified an error on our part. The units quoted throughout our paper for butyrate and glutamine metabolism should have been mmol substrate metabolised/living propionate per factor. We have now corrected the statement in the Discussion. The conclusion and conclusions for all the data remain as stated.

The critical difference between our study and that of Chapman et al is that we have examined butyrate metabolism in the presence of glucose but not glutamine, whereas their study was performed in the presence of all three energy substrates. Our own studies and those of Roediger3 (but not those of Chapman et al) has shown increased metabolism of glutamine in ulcerative colitis. It seems likely that this may be a reflection of hyperplasia, as rapidly dividing cells have increased metabolism of glutamine. In the absence of glucose we have failed to find any defect in metabolism of butyrate, and suspect that the results of Chapman et al may reflect the predilection of the mucosa for glutamine. It has been shown much the same ability to metabolise butyrate. To some extent a similar conclusion can be reached from part of Roediger’s original study. He showed that the rate of oxygen uptake by the pretreatment of butyrate as the sole energy substrate was normal in quiescent ulcerative colitis and only reduced in acute colitis, and although production of carbon dioxide from butyrate was reduced in ulcerative colitis, the most striking change in the contribution of different fuels to total oxygen consumption was the increased metabolism of glucose in quiescent coli.

With regard to the appropriate choice of glutamine concentration there is no clear answer. As Chapman et al themselves point out there is likely to be little or no glutamine available from the colonic lumen because it is so readily utilised by both big and peripheral blood concentrations of glutamine are less than 0.5 mM, so culture in the presence of 5 mM glutamine cannot be considered physiological.

We were interested in the results from ileal mucosal biopsy specimens reported by Chapman et al in their study. We would suspect that the reduced metabolism of butyrate that they have shown in the ileum is a result of patients undergoing colectomy, presumably for severe colitis, might be to some extent a reflection of the severe illness combined with backwash ileitis. For this reason we chose to study ileal biopsy specimens obtained at colonoscopy from patients in clinical remission. The figures for coefficients of variation we published were for all samples, ileal and colonic included (20% for butyrate metabolism and 23% for glutamine metabolism). The coefficients of variation for ileal and colonic butyrate metabolism in ileal biopsy specimens were 20% and for butyrate 18%, not 50% as stated by Chapman et al. There was, if anything, a very slight trend towards increased butyrate metabolism in ileal ulcerative colitis in our study. We have subsequently increased the numbers of ileal biopsy specimens studied to 18 in all and the conclusion remains the same.

There clearly are some interesting differences in mucosal metabolism in ulcerative colitis but our view is that most of these differences are likely to reflect changes occurring secondary to hyperplasia.

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Risk factors for Helicobacter pylori

EDITOR.—The findings of the EUROGAST Study Group on risk factors for H pylori infection (Gut 1993; 34: 1672-6) interested us because of the relation between this study and our previous work. The principal finding was a positive association between butyrate oxidation and low social class. The prevalence of gastritis increased with descending social class, and increased with age in all classes, becoming roughly equal in each class at 50 years and over.

Unlike the EUROGAST study our data suggested a positive correlation also between atrophic gastritis and excessive cigarette smoking, excessive alcohol consumption, and drinking hot tea.

The study by Vincent et al (Gut 1994; 35: 313) showed a high prevalence of H pylori in children cared for in a medical centre for mentally retarded children. The authors thought that factors related to close contact with other children were probably the cause of the high prevalence.

We were puzzled by our findings nearly 30 years ago. It is of interest that Bateson’s4 in his review of H pylori infection mentions its higher prevalence, and at an earlier age, in the developing world. Perhaps, if there is a relation between atrophic gastritis (and H pylori infection) and social factors it is mediated through interacting elements of living conditions probably associated with low grade education: financial hardship, restricted accommodation with people in close proximity and possibly sharing food and cooking utensils, poor hygiene, and low social class.

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Reply

EDITOR.—Based on their findings, Edwards and Coghill suggest that social factors implied in the spread of H pylori infection could explain the epidemiological pattern of atrophic gastritis.

Crowding and promiscuity are probably the ultimate risk factors for H pylori infection. These conditions are mediated by a low social economic class, which is a good risk marker but not a causal factor of infection. Our study showed that when they have been living in the same conditions for years, cohabiting children from different ethnic and social origins attain the same level of risk for H pylori infection. Furthermore, the high seroprevalence known in gastrosendoscopists5 shows that infection can be acquired even in hygienic conditions. This is not in disagreement with the social distribution of atrophic gastritis. H pylori, which is generally acquired in childhood, induces a chronic inflammatory reaction for years, but epithelial changes of atrophy and metaplasia are not constant and could vary with the host and circumstances. Atrophic lesions are uncommon in infected children, and it has been shown in adults that the mucosal colonisation area of H pylori can be larger than the area of chronic active gastritis. Moreover, an aggravation of mucosal inflammatory activity by H pylori associated gastritis can be found after absorption of non-steroidal anti-inflammatory drugs (NSAIDs). An atrophic gastritis found in adults might be a multifactorial condition resulting from H pylori infection (as a necessary but not sufficient factor) associated with consumption of gastric toxic agents, such as NSAIDs, alcohol, tobacco, spices, food, etc.
Consequently, growing older, educational standard, and social habits could participate in atrophic gastritis but not in the same way as for the acquisition of H pylori.

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**BOOK REVIEW**


The evolving relationship between a gastroenterologist who has acquired a new textbook can be compared with a car owner and his new car. Both objects look bright and new at the beginning. It is a different and interesting experience to get into them but, quite soon, the novelty wears off. Occasionally, when they are particularly needed, either can let you down.

With textbooks, it is perhaps a little unfair to come to a hurried judgment on a first edition. Indeed, there may be a case for sparing detailed critical attention until the second edition has come along. If the textbook is not up to scratch, it will not actually reach a second edition but, by the time a revised version does hit the market, authors and editors will have had an opportunity to rectify the product.

I had a copy of the first edition of this book and, judging by its rather osteoporoic spine, I seem to have used it quite a lot. It was certainly unique on my bookshelf in being the only substantially textbook of gastroenterology that could be easily held and read in the hand. Its length and breadth were barely larger than the original size of this journal (deceased 1989). The second edition has grown in length and breadth, and I suspect the dimensions of the modern Gut - 1990 onwards) and has slightly contracted in width. How good is this new model?

It is very much a textbook of clinical gastroenterology, as its title would imply. Furthermore, it is emphatically in a single volume, and I have to say it is my strong personal view that it is very much a frustration of modern life to have to read multi-author chapters in multi-editor texts that come in multiple volumes. The growth of knowledge is inexorable, but it is good to see this matched by editorial excellence, which makes the book both readable and undaunting. For example, the whole of inflammatory bowel disease is covered in 90 pages with lucid and Crohn’s disease, each having three chapters on their clinical features, medical management, and surgery respectively. I have not seen the clinical aspects of inflammatory bowel disease covered so well given this sort of space constraint, but there is necessarily some sacrifice of detail, particularly the pathogenesis (especially immunology) of these conditions. One accepts that a single volume will inevitably concentrate on the most clinically relevant areas but, since its first edition, there has been really a substantial expansion in the number of references for each chapter with almost all increasing by at least half, and in many cases, doubling.

The publisher has served the authors and editors really well. The various hierarchies of headings and subheadings are very easy on the eye. The figures and tables are beautifully produced and the x rays are compellingly clear. As usual, black and white histology is disappointing and it seems curious that there are so many photographs of extremely thin, naked people. Sadly, gastrointestinal disease may cause patients to become thin and I am not sure how this is gained from reminding us of this so visually.

Inevitably, any reviewer will find ‘highs’ and ‘lows’. I have already mentioned my liking of the section on inflammatory bowel disease, but I also enjoyed the chapter on nutrition. The section entitled ‘Colon Polyps’ was a little disappointing. This is surely an area of keen interest for gastroenterologists. The table of polytosis syndromes made no mention of hereditary non-polyostosis colon cancer or Lynch syndrome. Perhaps a detailed discussion of molecular genetics was outside the scope of the book, but I think this most exciting area of gastrointestinal research could have merited a few lines. By contrast, the immunogenetics of coeliac disease is discussed but, in this otherwise very readable chapter, it is surprising to find no discussion of the widening spectrum of the condition.

This is an excellent book. It is written by practising clinicians for practising clinicians and, as such, is not surpassed.

IAN FORGACS

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**NOTES**

Sir Francis Avery Jones BSG Research Award 1995

Applications are invited by the Education Committee of the British Society of Gastroenterology who will recommend to Council the recipient of the 1995 Award. Applications (fifteen copies) should include:

1. A manuscript (2 A4 pages only) describing the work conducted.
2. A bibliography of relevant personal publications.
3. An outline of the proposed content of the lecture, including title.
4. A written statement confirming that all or a substantial part of the work has been personally conducted in the United Kingdom or Eire.

Entrants must be 40 years or less on 31 December 1995 but need not be a member of the BSG. The recipient will deliver a 40 minute lecture at the Spring meeting of the Society in 1995. Applications (fifteen copies) should be made to: The Honorary Secretary, BSG, 3 St Andrews Place, London NWI 4LB by 1 December 1994.

Gastroenterology and liver disease

The 20th anniversary of Annual Topics in Gastroenterology and Liver Disease will be held on 5-7 October 1994 in Baltimore, Maryland, USA. Further information from: Program Coordinator, Johns Hopkins Medical Institutions, Office of Continuing Education, Box 368, University of Virginia Health Sciences Center, Charlottesville, Virginia 22908, USA. Tel: 410 955-2959.

Gastrointestinal motility

The Eighth Biennial Meeting of the American Motility Society will be held on 20-23 October 1994 at Wintergreen Resort, Wintergreen, Virginia, USA. For further information contact the Office of Continuing Medical Education, Box 368, University of Virginia Health Sciences Center, Charlottesville, Virginia 22908, USA. Tel: 804 924 5310.

Liver diseases

The American Association for the Study of Liver Diseases will hold a series of meetings during November 1994 in Chicago, USA. Further information on these meetings is available from: AASLD, Registration Manager, 6000 Grove Road, Thorofare, NJ 08086, USA. Tel: 609 848 1000, extn 213.

Pancreatic diseases

The Combined Meeting of the International Association of Pancreatology and American Pancreatic Association will be held on 2-4 November 1994 in Chicago, USA. Further information from: Linda Bakken, GI Unit, 2-424, Mayo Clinic, 200 1st Street SW, Rochester, MN 55905, USA. Tel: 507 255 4303; fax: 507 255 6318.

Therapeutic endoscopy

The Chinese University of Hong Kong and the Hong Kong Society of Digestive Endoscopy will hold the Ninth International Workshop on Therapeutic Endoscopy on 6-8 December 1994 in Hong Kong. Further information from: Dr Sydney Chung, Endoscopy Centre, The Chinese University of Hong Kong, Prince of Wales Hospital, Shatin, NT, Hong Kong. Tel: 852 636 2233; fax: 852 635 0075.

Correction

An authors’ error occurred in the paper by Dr A Finnie et al (Gut 1993; 34: 1552-8). The units of metabolism for butyrate and for glutamine throughout the paper should have been nmol substrate metabolised/h/mg protein and not, nmol/h/mg protein as stated. The ratios for butyrate, transaminase, pyruvate and glutaminase analysis, and the conclusions remain unchanged.