Primary biliary cirrhosis and primary sclerosing cholangitis are of infectious origin!

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Primary biliary cirrhosis and primary sclerosing cholangitis are of infectious origin!

EDITOR,—Haydon and Neuberger (Gut 2000;47:586–8) elegantly summarised the possibility that specific bacterial and viral pathogens may trigger early bile duct damage in the pathogenesis of primary biliary cirrhosis (PBC). It was concluded that a chronic infection, probably of viral origin, was driving an immune response with antimitochondrial and other autoantibodies enhancing the tissue damage in later stages of PBC. Besides detection of antibodies specific for mycobacterial antigens, such as the 55 kDa and 65–75 kDa antigens of M. gordoniae, in PBC the authors emphasised, based on studies by Mason and colleagues, that immune responses detected by immunoblotting to retroviral proteins with homology to HIV p24 and other retroviral antigens, such as HLA, are common in PBC, in primary sclerosing cholangitis (PSC), and in Sjögren’s disease.

Recent studies propose a possible role for Helicobacter pylori in Sjögren’s disease. A Con- tradictory data have appeared more recently based on a possible high relevance of anti-Helicobacter antibodies in saliva and sera of these patients. Most recently, Fox et al found that patients with chronic cholangitis in Chile were commonly infected by bile tolerant novel Helicobacter species, previously only detected in chronic liver disease in mice and other rodents, such as H. hepaticus and H. bilis. Nils- on et al first reported on bile and liver samples positive for Helicobacter DNA by polymerase chain reaction (PCR) in nearly half of 24 patients with PBC and PSC, and later immuno- blot analyses of patients with these and other chronic liver diseases. Conflicting negative PCR results on bile were reported by Tanka and colleagues while studies in Taiwan and Korea regularly seem to detect Helicobacter in human bile in chronic cholestasis bile tract disease. Recently, Bulajic et al reported on a strong correlation between bile duct malignancies and the presence of H. pylori DNA in bile. Since bile acids, intestinal cells, and highly charged mucin components are strong inhibitors of the PCR reaction, all of these studies have to be interpreted with caution until methods to safely remove or neutralise the effect of these inhibitors in bile, bile tract, and liver biopsies have been developed. We recently reported that PCR analyses of formalin fixed, paraffin embedded liver, pancreas, and bile tree samples may be a safe way to produce reproducible PCR analyses of Helicobacter and other potential bacterial invaders of the human bile tract. Interestingly, preliminary findings in our study on experimentally infected laboratory animals with various Helicobacter strains suggest that these may be translocated from the stomach and the intesti- ne to the liver, and we speculate that this may involve uptake and intracellular survival in macrophages and other professional phago- cytes activated in the stomach during most Helicobacter infections (T Wadström et al, unpublished observations). The pathogenesis may then be similar to infection of the bile tree they will not respond to antibiotic therapy for Helicobacter and other pathogens when these patients develop clinical disease. However, development of sensitive immunodiagnostic tests may serve as a screening tool and permit early diagnosis of Helicobacter associated bile tree and liver diseases in human patients as well as in laboratory animals.

We would certainly like to add PBC and PSC to the list of infectious diseases, and it seems likely that in a certain proportion of patients, H pylori and other Helicobacter species may play a role in the pathogenesis.

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Our own studies were supported by the Swedish Medical Research Council (16X-04723).

differentiating between trimeric (bioactive) TNF-α and proteolytic split products of TNF-α. Both forms are thought to be measured by the Medgenix assay. They go on to propose that this allows a measure of recently released bioactive TNF-α as opposed to an estimate of release over past hours. The authors do not include any data to substantiate such a claim.

Following discussion with R&D systems, we can confirm that they do not claim that their high sensitivity kit measures only trimeric (bioactive) TNF-α and that there are no data comparing this kit with a bioassay as they measure different things, namely immunoreactivity (mass) versus bioactivity. The apparent twofold greater level of TNF-α found using the Medgenix kit may simply reflect a calibration difference between the two kits.

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Reply
EDITOR,—Several years ago we compared different tumour necrosis factor (TNF) assays.1 We observed a strong correlation between the bioactive TNF trimer and the R&D assay (r=0.88) whereas the correlation was poor (<0.3) between both assays and the Medgenix ELISA. Moreover, the kinetic studies after in vitro lipopolysaccharide stimulation showed that bioactive TNF is produced for a few hours only whereas the Medgenix TNF assay (in contrast with the R&D assay) detects TNF even 24 hours after stimulation.1 In addition, monoclonal antibodies recognising and neutralising TNF interact with the R&D assay but not with the Medgenix assay. Hence the differences between the assays are not simply due to the differences in calibration. If sera that contained TNF immunoreactivity were fractionated into fractions less than or greater than 40 kDa (the trimers has about 51 kDa) it was observed that the Medgenix but not the R&D assay recognised low molecular weight as well high molecular weight fractions (unpublished data). In summary, there is strong evidence that the Medgenix assay recognises a strongly inactive TNF split product in addition to the bioactive TNF trimmer but the R&D assay does not.

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Chromosome 3p and inflammatory bowel disease
EDITOR,—We were particularly interested to read the paper of Hampe et al (Gut 2001;48:191–7) which provides some supportive evidence for the presence of a gene involved in susceptibility to inflammatory bowel disease on chromosome 3p. As Hampe et al describe, this region of interest was initially identified in a study of 186 affected sibling pairs, all resident in and indigenous to the UK. Since that initial observation, subsequent genome wide scans in European and North American populations have produced inconsistent data for the chromosome 3p region. There are a number of possible explanations for the inconsistent data, all well summarised by Hampe et al.

We suggest that the issue of heterogeneity between populations may be pertinent to the study of the chromosome 3 locus. We noted with interest that the dataset of 353 sibling pairs studied by Hampe et al includes a high proportion (48%) of UK sibling pairs, together with sibling pairs from Germany (46%) and the Netherlands (6%). In view of the fact that there appears to be heterogeneity between different continental populations in Europe concerning the chromosome 16 and 12 loci,1-3 we would be particularly interested to know whether the data implicating chromosome 3p in Hampe’s study is in fact stronger in the subset of families from the UK than those from Germany and the Netherlands. It would be of benefit in future studies to ascertain whether the chromosome 3p region does have a relatively stronger effect in the UK population than in other populations.

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Reply
EDITOR,—The point raised in the letter by Satsangi and Vermeire is very valid. There has been significant variation in the presence of linkage in different populations.1-3 From a preliminary re-analysis of the data presented in our paper (Gut 2001;48:191–7), the hypothesis raised through Satsangi and Vermeire appears to be true: there is a stronger contribution of linkage in the UK families than in the German families to the chromosome 3p linkage with a peak multipoint LOD score of 1.0 in the UK and 0.8 in the German families (not all markers analysed). It should be noted that each subsample is somewhat subcritical for a proper linkage analysis and thus these data have to be viewed with caution.

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Indecision and irritable bowel
EDITOR,—The Guidelines for the management of the irritable bowel syndrome supplement was a commendable venture, but with physicians tending to depend more and more on sources other than reading for their continuing education, actual guidance is also necessary, especially from what physicians such as Almy1 and Kirsner2 have found does and does not work. Sadly they were not quoted. In addition, indecision and “fence sitting” has been found to be the most common stressful life situation presented at the time of onset or relapse of IBS.3 Therefore, any indecisiveness on the part of doctors about choice of treatment of IBS is likely to be picked up by their patients.

The guidelines rightly pointed out the limitations of “end organ” treatments compared with centrally directed therapies, such as hypnosis and relaxation methods. However, these are also rarely effective when a patient has a nagging personal problem at the back of his or her mind. Uncovering such doubts requires open ended questions as recommended by Almy.4 Patients may be encouraged to make such decisions by the likelihood of remission of symptoms if they do.

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Reply
EDITOR,—The importance of stressful life events is of course well recognised and new evidence is constantly accumulating. In addition to the earlier papers quoted by Dr Paulley, more recent publications in Gut indicate that resolution of chronic life stresses are important predictors of clinical outcome in irritable bowel syndrome supporting the earlier publications quoted by Dr Paulley.

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BOOK REVIEWS


A booklet little larger than the size of a two column (10 cm x17 cm) may seem unimportant. But this is an exception. This publication is for patients with ulcerative colitis and such sources of information should be the concern of gastroenterologists. It has been written by Andrew Robinson, whose self management programme for patients with colitis lead to fewer outpatient visits, more rapid treatment of relapse, and improved patient satisfaction (Gut 1999; 44:A12), and Anne Kennedy, a research fellow who in writing has been assisted by a professional writer and sensibly had the guide endorsed by the Plain English Campaign.

The guide consists of two booklets in a single plastic folder. Part 1 includes an overview of ulcerative colitis, its causes, tests, treatment, and surgery. Part 2 is an individual patient record. There is much to be commended, with detailed information helpfully summarised in coloured boxes (“Things to Remember”), or treatment options discussed (“Your Choice”) and anecdotals from patients that give a personal appeal. Clinical views and opinions are, on the whole, well balanced and I could see this guide being a valuable contribution to patient information. Faults however qualify this commendation. The surgical subsection on ileorectal anastomosis for ulcerative colitis is wholly inappropriate and there is confusion in terminology in the section on pouch surgery. Factual errors (such as a “2% risk” of ulcerative colitis in offspring, and surgery) is far too fast but that a full understanding of gut development and function also required an understanding of the composition and properties of human milk and the metabolism of the newborn. The developing gastrointestinal tract and lactating mammary gland are complementary organs, jointly involved in the transfer of nutrients and other substances from mother to infant. Until weaning the neonate is at extra-gestal fetus, and breast and gut are analogous to the uterine-placental interface.

This book goes a long way to recognising this. Each chapter (or section of each chapter stand alone review) is written by a leading figure or group expert in its field. Together they cover the major aspects of gut development and function but apart from a short preface there is no overview or attempt to synthesise the book’s contents. It would be impossible for one author to write this book. The impact of molecular biology has moved the subject away from the oesophagus. This book is certainly all inclusive and most comprehensive. It is currently regarded as the province of medicine and science, and a determination to produce a review article overnight). However, the opportunity to print out chapters will abolish the tedium of photocopying, and also preserve the spine of this handsome and well produced book.


While the oesophagus has provided much interest to physicians and surgeons, particularly since the advent of endoscopy, manometry, and pH monitoring, to many pathologists it remained a muscular tube of relative pathological disinterest. Perhaps compared with the stomach and the intestines there is a relative paucity of interesting pathological conditions to study. But the oesophagus has certainly changed much of that perspective and the fact that entire books can now be devoted to the study of the pathology of the oesophagus truly underlines how the use of numerous endoscopic images will be of interest not only to practising endoscopists but also to pathologists and researchers.

Dr Takubo has developed an international reputation for his work in various aspects of the pathology of the oesophagus and his efforts in producing this book are to be applauded. Differences in the pathological assessment of tumours in the gut between

Nevertheless, these points are correctable and if asked by a patient I would broadly recommend the guide. There is nothing else like it on the market and it gives far more useful information than can be readily gleaned from the Internet or from pharmaceutical sponsored freebies. However, the authors will stand by their commitment to update the guide every two years. This means that they should be working on the 2001 edition now.

S P L TRAVIS


When I was a fellow with Allan Walker 15 years ago, gut development was a topic of interest to a handful of researchers worldwide. A classic review by Grand, Watkins, and Torii publishing in 1976, and Koldovsky’s monograph Development of the Functions of the Small Intestine in Mammals and Man in 1969 brought together much of what was then known about the ontogeny of the human digestive system. Developmental biologists were beginning to recognise the opportunities offered by this rapidly differentiating organ to understand the interactions of genetic endothem and environmental influences in early life. The focus of much research was on the process of adaptation of milk feeding. With the survival of ever more preterm infants, the function of the immature gut and its capacity to deal with enteral feeds prematurely were questions of increasing practical concern.

I had the grand idea at that time to produce a short book bringing the field together. But I quickly realised that it was growing too fast but that a full understanding of gut development and function also required an understanding of the composition and properties of human milk and the metabolism of the newborn. The developing gastrointestinal tract and lactating mammary gland are complementary organs, jointly involved in the transfer of nutrients and other substances from mother to infant. Until weaning the neonate is at extra-gestal fetus, and breast and gut are analogous to the uterine-placental interface.

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L WEAVING
ties not principally involved with other disciplines including hospital speciali-
but, hopefully will also be of value to many in having contributed to others, I have written

The invaluable source of reference. I thoroughly agus will find this well written, well illus-

The only slight oesophagus at least, than other areas of the recently been highlighted. In this book, inevi-

Western and Japanese pathologists have been extensively updated. The book is only

This is actually the second edition of a

24 tables. It is divided into four chapters 64 pages in length with some 22 figures and

Helicobacter pylori

(3) Management of Helicobacter pylori

M. heatley’s little book to “get up to speed” on the subject and

regard to slowing retroviral progression, cryptosporidiosis or microsporidiosis. In ad-

individuals in the developing world develop to the burden of diar-

better outbreak investigation in the future

They would find NJ Talley’s article entitled

Helicobacter pylori

Letters, Book reviews, Notes

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Microsporidiosis is a timely reminder of the

for opportunistic infections. Sadly few people

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his aims in producing a book suitable for general practitioners and other non-

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his/her outbreak in the USA, a

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leading to a full scale enquiry by the Austral-

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In the developing world, parasitic disease

In the last 5 years, more than 200 000 new vir-

moral importance of these infections providing an

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few pages are dedicated to small bowel bacte-

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Helicobacter pylori

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It is a book that covers many pages of the book that

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This book is much more than a

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Absorption of nutrients is presented in a very


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D iabetes and their physiology of the small intestine. On the

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gastroenterology in the western world. Nevertheless, the chapter on coeliac disease provides some practical information on following a gluten free diet that will be very useful not only for gastroenterologists but also for general practitioners and dieticians dealing with patients affected by coeliac disease. Patients too may find this chapter of great use. The chapter on Crohn’s disease is up to date and interesting. Disappointingly, the use of ultrasonography in both the diagnosis and follow up of this condition is only briefly described.

The references are somewhat disappointing. Although in some chapters they are up to date, in others most date back to the 80s.

In conclusion, this book will be useful for clinicians with a specific interest in the small intestine. Otherwise, however, gastroenterologists may not find this book much more useful than the chapters dedicated to the small bowel in the major gastroenterology textbooks. Moreover, gastroenterologists working in tertiary referral centres for the small bowel may find some chapters out of date and not of much use for the most difficult decisions. Hence clinicians practising in the tropics or dealing with patients traveling in less industrialised countries are those that will benefit most.

F BIAGO

Sir Francis Avery Jones British Society of Gastroenterology Research Award 2002

Applications are invited by the Education Committee of the British Society of Gastroenterology who will recommend to Council the recipient of the 2002 Award. Applications should be made to the Honorary Secretary, British Society of Gastroenterology, 3 St Andrews Place, London NW1 4LB by 1 December 2001.

Christina F. M. Schooling

Hopkins Endoscopy Prize 2002

Applications are invited by the Endoscopy Committee of the British Society of Gastroenterology who will recommend to Council the recipient of the 2002 Award. Applications should include:

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

Entrants must be 40 years or less on 31 December 2001 but need not be a member of the Society. The recipient will be required to deliver a 30 minute lecture at the Annual meeting of the Society in Glasgow in March 2002. Applications (TEN COPIES) should be made to the Honorary Secretary, British Society of Gastroenterology, 3 St Andrews Place, London NW1 4LB by 1 December 2001.

A written statement confirming that all or a substantial part of the work has been personally conducted in the UK or Eire.

Falk Symposium No 123: VI International Symposium on Inflammatory Bowel Diseases

This Falk Symposium will be held on 3–5 September 2001 in Istanbul, Turkey. Further information: Falk Foundation e.V., Congress Division, Leinenwebersr. 5, PO Box 6529, D-79041 Freiburg, Germany. Tel: +49 761 15 14 0; fax: +49 761 15 14 359; email: symposia@falkfoundation.de

Falk Symposium No 124: Medical Imaging in Gastroenterology and Hepatology

This Falk Symposium will be held on 28–29 September 2001 in Hannover, Germany. Further information: see Falk Symposium No 123 above.

9th Asian Conference on Diarrhoeal Diseases and Nutrition

This meeting will be held on 28–30 September 2001 in New Delhi, India. The organisers hope the meeting will promote meaningful and effective collaboration among individuals/institutions towards control of the major health problems in Asia, particularly those affecting women and children. Further information: Professor M K Bhan, Coordinator, Centre for Diarrhoeal Disease and Nutrition Research, All India Institute of Medical Sciences, New Delhi. Tel: +91 11 6963828; fax: +91 11 686266; email: ascodd2001@rediffmail.com

VI Congress of the International Xenotransplantation Association

This congress will be held on 29 September to 3 October 2001 in Chicago, USA. Further information: Felicissimo & Associates Inc., 205 Viger Avenue West, Suite 201, Montreal, Quebec, Canada H2Z 1G2. Tel: +1 514 874 1998; fax: +1 514 874 1580; email: info@ixa2001chicago.com; website: www.ixa2001chicago.com

Falk Symposium No 125: Cytokines in Liver Injury and Repair

This Falk Symposium will be held on 30 September to 1 October 2001 in Hannover, Germany. Further information: see Falk Symposium No 123 above.

Falk Symposium No 126: Hepatocyte Transplantation

This Falk Symposium will be held on 2–3 October 2001 in Hannover, Germany. Further information: see Falk Symposium No 123 above.

EASL Single Topic Conference

The EASL Single Topic Conference “Liver fibrosis: from basic science to clinical targets” will be held on 12–13 October 2001 in Florence, Italy. Organisers: Massimo Pinzani (University of Florence) and Delele Schuppan (University of Erlangen-Nuernberg). The aim of the conference is to provide the latest information on this key area of hepatology and to translate the current knowledge into clinical terms. It is directed at both the expert in the field and the general hepatologist. Further information: Massimo Pinzani, Dipartimento di Medicina Interna, Università degli Studi di Firenze, Viale GB Morgagni, 85, I-50134 Firenze, Italy. Tel: +39 055 4277845; fax: +39 39 055 417123; email: m.pinzani@dfc.uniﬁ.it

Lecture Course in Coloproctology

This course will be held on 15–17 October 2001 in Harrow, UK. Professor Russell Stitz from Australia will be the Sir Alan Parks Visiting Professor and, for the first time, there will be a Sir Francis Avery Jones Visiting Professor which will be Professor Paul Rutgeerts from Belgium. Further information: The Administrator, St Mark's Academic Institute, St Mark’s Hospital, Northwick Park, Harrow, Middx, HA1 3UJ, UK. Tel: +44 (0)20 8235 4046/8; fax: +44 (0)20 8235 4039; email: stmarks@ic.ac.uk; website: www.stmarkshospital.org.uk

International Symposium on Hyperammonemia, Liver Failure and Hepatic Encephalopathy

This symposium will be held on 20–22 October 2001 in Valencia, Spain. Further information: Cátedra Santiago Grisolía, Fundación Museo de las Ciencias Príncipe Felipe, Ciutat de les Arts i les Ciències, Avda. Institut Obrero, s/n, 46013 Valencia, Spain. Tel: +34 96 197 44 66; fax: +34 96 197 44 70; email: catedrasg@cac.es

ICGH-2: The Second Iranian Congress of Gastroenterology and Hepatology

The main Iranian meeting of gastroenterologists and researchers in this field will be held on 27 October to 1 November 2001 in Tehran, Iran. Further information: Dr Shahin Merat, Digestive Diseases Research Center, Shariati Hospital, N. Kargar Street, Tehran 14114, Iran. Tel: +98 911 717 3966; fax: +98 21 225 3635; email: merat@ams.ac.ir; website: www.ams.ac.ir/icgh

Falk Symposium No 127: Autoimmune Diseases in Pediatric Gastroenterology

This Falk Symposium will be held on 8–9 November 2001 in Basel, Switzerland. Further information: see Falk Symposium No 123 above.

Falk Symposium No 128: Autoimmune Diseases in Pediatric Gastroenterology

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