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). We 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 colleagues1 and others, that immune responses detected by immunoblotting to retroviral proteins with homology to HIV p24 and other retroviral antigens, such as HIAp, 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.2 Contradictory data have appeared more recently based on a possible high relevance of anti-Helicobacter antibodies in saliva and sera of these patients.3 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.4 Nilsen 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 immunoblot analyses of patients with these and other chronic liver diseases.5 Conflicting negative PCR results on bile were reported by Tanaka and colleagues6 while studies in Taiwan and Korea regularly seem to detect Helicobacter in human bile in chronic cholestatic bile tract disease.7 Recently, Bulajic et al reported on a strong correlation between bile duct malignancies and the presence of H pylori DNA in bile.8 Since bile acids, intestinal acids, 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.9 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 intestine to the liver, and we speculate that this may involve uptake and intracellular survival in macrophages and other professional phagocytes 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,2 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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Measurement of tumour necrosis factor α

EDITOR—In their paper (Gut 2000;47:281–7), von Baehr et al suggest that the high sensitivity Quantikine ELISA for tumour necrosis factor α (TNF-α) is capable of

Figure 1 (A) Cluster of 3 μm Helicobacter species in primary sclerosing cholangitis damaged liver (arrows). Immunohistopathology with specific antibodies raised against H pylori. Magnification ×1250. (B) Helicobacter species in liver tissue portal zone, demonstrating white energy spots, pepsin- P granule (arrows). Transmission electron microscopy, magnification ×8800.
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 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 immunoreactive (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. We observed a strong correlation between the bioassay 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 (fig 4 in Asadullah and colleagues). 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 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 reactive TNF split product in addition to the bioactive TNF trimer 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 loci. 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 the two European groups and other 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 Satanghi and Vermeire is very valid. There has been significant variation in the presence of linkage in different populations.1,2 From a preliminary re-analysis of the data presented in our paper (Gut 2001;48:191–7), the hypothesis raised through Satanghi 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. I HAMPE
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Indecision and irritable bowel

EDITOR,—The Guidelines for the management of the irritable bowel syndrome supplement was a commendable effort 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 not quoted. In addition, indecision and “fence sitting” has been found to be the most common stressful life situation present 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

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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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. I hope 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 Tori published 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 gastrointestinal biology were beginning to recognize the opportunities offered by this rapidly differentiating organ to understand the interactions of genetic endowment and environmental influences in early life. The focus of much research was on the process of adaptation to 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 realized that this 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 development of 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 an extra-uterine fetus, and breast and gut are analogous to the uterine-placental interface.

This book goes a long way to recognising this. Each chapter (the one on the end plate alone a 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 from an essentially descriptive science, with some experimental work in vivo, to the level of the cell and gene. This has shifted away from the womb, breast, or incubator and into the laboratory. This book is certainly of interest to physicians and surgeons, particularly as 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 the busy pathologist. The oesophagus has certainly changed much of that perspective and the fact that entire books can now be devoted to the study of the oesophagus. As a result Dr Takubo’s book follows the excellent, but now time expired, efforts of Drs Enterline and Thompson in producing a comprehensive pathological survey of the oesophagus. This book is certainly all inclusive and most comprehensive. It is a handsome and well produced book.
Western and Japanese pathologists have recently been highlighted. In this book, inevitably favouring Japanese methodology, such differences are perhaps less marked, for the oesophagus at least, than other areas of the gut and these do not cause particular concern for the Western pathologist. The only slight irritations of this book are the references which are all lumped together at the end and I found referring to these a little labourious. Otherwise, I have no complaints. All those with a keen interest in diseases of the oesophagus will find this well written, well illustrated, and extremely well researched and referenced textbook an informed read and an invaluable source of reference. I thoroughly recommend it.

N A SHEPHERD


In his preface, Dr Heatley states “Having been involved in the production of one of the established texts on this subject, and also having contributed to others, I have written this book with the generalist in mind. It is intended mainly for those in primary care but, hopefully will also be of value to many in other disciplines including hospital specialities not principally involved with *H pylori* management, including those in care of the elderly patients, paediatrics, general bacteriology, chemical pathology, nuclear medicine and as such well as others.” To what extent do I think Dr Heatley has succeeded in his aims in producing a book suitable for general practitioners and other non-gastroenterologists?

This is actually the second edition of a book originally published in 1995 and has been extensively updated. The book is only 64 pages in length with some 22 figures and 24 tables. It is divided into four chapters entitled: (1) *Helicobacter pylori* the organism; (2) *Helicobacter pylori*: the clinical problem; (3) Management of *Helicobacter pylori* infection, and (4) Managing dyspeptic conditions in the elderly patient. Considering the fact that thousands of original papers have been written in the 16 or so years since Barry Marshall and colleagues first “discovered” the bacteria, I thought this author has summarised the relevant literature very well indeed. However, there are no references or lists of suggested reading, and I think this is a weakness.

I thought the first three chapters were excellent but was frankly a bit disappointed with the fourth and final chapter dealing with the management of dyspeptic conditions. In this era of “evidence based medicine” perhaps the author is correct to be so critical of, for example, the evidence that *Helicobacter pylori* is indeed a class I carcinogen, as suggested by the WHO or the Maastricht Consensus report’s recommendations on the management of *Helicobacter pylori* positive NZ. I think the real world may differ. If patients told they have a bug in their stomach which if left might have a 1:4 chance of later causing a peptic ulcer or a 1:100 chance of turning into stomach cancer, will opt for eradication therapy. Textbooks in such a fast moving field as *Helicobacter pylori* are in danger of being out of date almost as soon as they are published. Perhaps a short term compromise would be for the interested general practitioner to first read Dr Heatley’s little book to “get up to speed” on the subject and then look at the relevant clinical sections of the excellent supplement of Gut reporting on the 5th Education Training Workshop in *Helicobacter pylori* held in Bologna in 1998. They would find NJ Tailey’s article entitled “How should a doctor handle positive dyspeptic patients be managed?” particularly useful to balance out the rather negative views expressed by Dr Heatley in his book.

G D BELL

**Cryptosporidiosis and Microsporidiosis**.


There is a great need to raise the profile of parasitic diarrhoeal disease, and this book serves the cause well. World wide the prevalence closely parallels the burden of diarrhoeal disease. Malnourished children are especially at risk of increased morbidity and mortality, both HIV negative and positive, using crude oocyst antigen but has yet to be fully evaluated. Farthing emphasises the clinical importance of childhood infection in the developing world and flags up new therapeutics, including nitazoxamide, which has good efficacy and is at least a potentially viable option in the tropics. McDonald and coworkers review developments in our knowledge of the host immune response to *Cryptosporidium* highlighting the role of T cell mediated responses. Work by the author has revealed the central role of interferon γ production by intraepithelial lymphocytes in the control of infection. Strong and Nelson tackle new developments in gene discovery of *C. parvum*. Using a combination of characterisation of expressed sequence tags and the use of “HAPPY” mapping technology, approximately 30% of the *C parvum* genome has been surveyed. A full scale genome mapping project is needed to allow comparison with the better characterised related parasites *Toxoplasma* and *Plasmodium*.

Topics relating to microsporidiosis are similarly covered. Weber and colleagues review diagnostic methods although information on the relevance of this infection in the tropics, where most disease now occurs, is limited. Our knowledge of the immune response to Microsporidium is relatively limited but as with Cryptosporidium, Didier points out the importance of interferon γ mediated T cell responses in control of infections. The book is divided into chapters on Cryptosporidium and water supplies which emphasised the need for better outbreak investigation in the future (www.dswi.detr.gov.uk/pubs/bouchier).

In the developing world, parasitic disease contribute heavily to the burden of diarrhoeal disease. Maltreated children are especially at risk of increased morbidity and mortality, both HIV negative and positive, from Cryptosporidiosis and microsporidiosis. Twenty years ago it was a major component of HIV positive individuals in the developing world develop cryptosporidiosis or microsporidiosis. In addition to slowing retroviral progression, HAART has emerged as the best treatment for opportunistic infections. Sadly few people with AIDS in the developing world have access to antiretroviral therapy.

For these reasons Cryptosporidiosis and Microsporidiosis is a timely reminder of the importance of these infections providing an authoritative up to date summary of the many recent developments in the field. Franz Petry, the editor, has done well to bring together a panel of authors with an international reputation in their subjects.

Tzipori and Widmer as well as Morgan and colleagues put forward evidence for two distinct genotypes of *C. parvum*: a “human-type” which may infect only humans or immunocompromised animals and an “animal-type” which may infect both humans and a range of animals. This has important applications in the molecular epidemiological characterisation of samples collected during waterborne outbreaks. For example, almost all isolates tested from the London/ Hertfordshire waterborne outbreak were of human origin, suggesting failure of water treatment. Petry summarises the diagnostic approaches for the identification of *C. parvum* infection. Particularly of interest is the recent development of ELISA for the detection of serum antibodies to *C parvum* immunodominant antigens. The technique appears to be both more sensitive and specific than ELISA using crude oocyst antigen but has yet to be fully evaluated. Farthing emphasises the clinical importance of childhood infection in the developing world and flags up new therapeutics, including nitazoxamide, which has good efficacy and is at least a potentially viable option in the tropics. McDonald and coworkers review developments in our knowledge of the host immune response to *Cryptosporidium* highlighting the role of T cell mediated responses. Work by the author has revealed the central role of interferon γ production by intraepithelial lymphocytes in the control of infection. Strong and Nelson tackle new developments in gene discovery of *C. parvum*. Using a combination of characterisation of expressed sequence tags and the use of “HAPPY” mapping technology, approximately 30% of the *C parvum* genome has been surveyed. A full scale genome mapping project is needed to allow comparison with the better characterised related parasites *Toxoplasma* and *Plasmodium*.


This is a book that covers not only small intestinal disorders but also the anatomy and physiology of the normal small bowel. Absorption of nutrients is presented in a very detailed way. Although these aspects are very well dealt with, it is somewhat curious that in a book entitled Small Bowel Disorders almost 50% of the text is related to the anatomy and physiology of the small intestine. On the other hand, chapters on symptoms and signs of small intestinal diseases and those on the use of the most important diagnostic tests will be of great help to clinicians. I was favourably impressed by the quality of the very interesting figures.

The book is particularly aimed towards tropical conditions affecting the small bowel. Many pages are specifically dedicated to infectious diseases and these are always kept in mind in the chapters describing diagnostic tests. In contrast, some non-infectious disorders are less extensively described. Only a few pages are dedicated to small bowel bacterial overgrowth, vascular disorders, radiation enteritis, and graft versus host disease, conditions that are relevant in the practice of tropical medicine.
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 (TWENTY COPIES) 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 (TWENTY COPIES) should be made to the Honorary Secretary, British Society of Gastroenterology, 3 St Andrews Place, London NW1 4LB by 1 December 2001.

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 (TEN COPIES) 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.

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 affecting women and children. Further information: Professor M K Bhan, Coordinator, Centre for Diarrheal Disease and Nutrition Research, All India Institute of Medical Sciences, New Delhi. Tel: +91 11 6963822; fax: +91 11 6862662; 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 4277848; fax: +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 Museu de les Ciències Príncep Felipe, Ciutat de les Arts i les Ciències, Avda. Institut Obreiro, 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.

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