Ulcerative colitis is more strongly linked to chromosome 12 than Crohn's disease

Chances of successful replication. We concur imply that studying the Crohn's disease sub-group of IBD should thus maximise the chance of successful replication. We concur entirely that genetic heterogeneity is important, and we have recently reported strong evidence that it does indeed apply to chromosome 12. However, our study of 367 multiply affected families suggested a significantly stronger contribution of this locus to ulcerative colitis than Crohn's disease. The difference between the linkage results for ulcerative colitis (LOD=3.01) and Crohn's disease (LOD=1.66) reached statistical significance in two separate tests for heterogeneity. In the light of these results, the validity of the exclusion map drawn by Lesage et al is undermined. The exclusion map was based on an assumed locus specific s value for this locus with regard to Crohn's disease is much less than 2. Thus the contention that Lesage et al can exclude a contribution of IB2 to Crohn's disease susceptibility is probably not valid. As pointed out in the accompanying editorial, simulation studies have demonstrated that lod scores can be expected to vary, particularly when the study population is relatively small. Furthermore, the implication that a panel of 157 affected relative pairs should provide sufficient power to detect linkage if this locus is contributing to disease susceptibility is at marked variance with the power calculations derived by Suarez et al, Mandal et al, and others.

In many respects, the surprising feature is that IB2 has been replicated in as many as five independent panels. The datasets that have failed to detect linkage at this locus have all contained predominantly or exclusively Crohn's disease pairs. Although IB2 probably does contribute to Crohn's disease susceptibility, the effect is likely to be weak and thus would require very large panels of multiply affected families to have a realistic expectation of replicating (or excluding) the linkage result.

It is our view that attempts at fine mapping IB2 probably have the greatest chance of success if they concentrate on panels of families and individuals with ulcerative colitis, which appears to be significantly more strongly linked to this locus than Crohn's disease.

M PARKES
J SATSANGI
D P JEWELL
Gastroenterology Unit, Radcliffe Inflammatory, Oxford, UK
D E WEEKS
M M BARMADA
Department of Human Genetics, Graduate School of Public Health, University of Pittsburgh, Pittsburgh, Pa, USA

Correspondence to: M Parkes. miles.parkes@well.ox.ac.uk


Reply

EDITOR,—In 1996, Satsangi et al reported a positive linkage between inflammatory bowel disease (IBD) multiplex families (including Crohn's disease (CD), ulcerative colitis (UC) and mixed families) with a locus (called IBD2) located on chromosome 12. The attributable risk in siblings of IBD2 multiplex families (n=157, all CD pairs) compared with expected relatives pairs should provide sufficient power to detect linkage if this locus is contributing to disease susceptibility at marked variance with the power calculations derived by Suarez et al, Mandal et al, and others.

In many respects, the surprising feature is that IB2 has been replicated in as many as five independent panels. The datasets that have failed to detect linkage at this locus have all contained predominantly or exclusively Crohn's disease pairs. Although IB2 probably does contribute to Crohn's disease susceptibility, the effect is likely to be weak and thus would require very large panels of multiply affected families to have a realistic expectation of replicating (or excluding) the linkage result.

It is our view that attempts at fine mapping IB2 probably have the greatest chance of success if they concentrate on panels of families and individuals with ulcerative colitis, which appears to be significantly more strongly linked to this locus than Crohn's disease.
Intestinal permeability: the cellobiosemannitol test

EDITOR,—I should like to bring to your attention a conceptual error in the paper by Daniele et al (Gut 2001;48:28–33) regarding the cellobiosemannitol test. The authors suggest that improvement in the cellobiose/mannitol ratio reflects improvement in permeability from the use of oral glutamine. However, only mannitol excretion improved significantly with glutamine; cellobiose excretion remained unchanged. As the authors explain in their methods section, it is the increased cellobiose excretion that reflects increased permeability, not the decrement in mannitol excretion. Therefore, modifications in sugar transport induced by 5-fluorouracil (5-FU) reflected only an absorptive, not a permeability, defect. The decrement in mannitol excretion parallels the decrement in xylose excretion, probably reflecting decreased transcellular passage of the test sugars due to 5-FU and improved with glutamine.

R M CRAIG
Department of Gastroenterology and Hepatology, Northwestern University, Chicago, Illinois 60611, USA
r-craig@northwestern.edu

Reply
EDITOR,—I thank Dr Craig for raising this issue but I do not see any conceptual error. The apparent inconsistency that he points out in our paper (Gut 2001;48:28–33) is due to the controversy surrounding transcellular permeation of mannitol, as well as of other monosaccharides. While transcellular permeation of mannitol is well known, its use for osmotic shrinkage of membrane vesicles and as an extracellular fluid volume marker suggests that, at least in part, mannitol diffuses through the intercellular tight junctions. Thus it seems justified talking of permeability for mannitol. One of the reasons for its use in combination with cellobiose is the different molecular sizes of the two probes: the smaller size of mannitol allows its passage through the small tight junctions of the villi while the larger cellobiose passes through the larger tight junctions of the crypts.

Finally, we did find an increase in cellobiose excretion after fluorouracil (5-FU) that was in part prevented by oral glutamine. Although this difference did not reach statistical significance, overall the data indicate increased intestinal permeability after 5-FU, partially prevented by oral glutamine.

B DANIELE
Divisione Di Oncologia Medica B, Istituto Nazionale Tumori, via M Semmola, 80131 Napoli, Italy
bdaniele@irso-oncology.it

Evaluation of the role of CFTR in alcohol related pancreatic disease

EDITOR,—In up to 30% of patients with idiopathic pancreatitis (IP) a mutation of at least one or both alleles of the cystic fibrosis transmembrane conductance regulator (CFTR) gene can be identified. The study by Malats et al (Gut 2001;48:70–4) addressed the question of whether CFTR mutations, possibly together with environmental factors such as alcohol, may be associated with chronic pancreatitis or pancreatic cancer. The vast majority of the pancreatic patients (86.4%) investigated by Malats et al were diagnosed as having alcoholic pancreatitis (AP), and 75.4% of the cancer patients were daily drinkers. The authors found no statistically significant differences in the prevalence of delta-F508 (0%, 2.4%) and the 5T allele (10.5%, 5.5%) in the AP or cancer groups compared with the expected prevalence in the general population. The lack of a positive association of both delta-F508 and the 5T allele with AP is neither surprising nor argues against involvement of CFTR variations in the development of AP, considering the following.

In cystic fibrosis (CF), the degree of correlation between CFTR genotype and CF phenotype varies between clinical components but is highest for pancreatic involvement. CFTR mutations can simplify be divided into “severe” and “mild” with respect to the degree to which mutations impair CFTR function. Approximately 85% of CF patients suffer from pancreatic insufficiency (PI). Generally patients with two “severe” mutations are associated with at least one “mild” mutation (fig 1). In CF, pancreatic exocrine is seen rather frequently in PS patients but not in PI patients.

The apparent inconsistency that he points out in our paper (Gut 2001;48:28–33) is due to the controversy surrounding transcellular permeation of mannitol, as well as of other monosaccharides. While transcellular permeation of mannitol is well known, its use for osmotic shrinkage of membrane vesicles and as an extracellular fluid volume marker suggests that, at least in part, mannitol diffuses through the intercellular tight junctions. Thus it seems justified talking of permeability for mannitol. One of the reasons for its use in combination with cellobiose is the different molecular sizes of the two probes: the smaller size of mannitol allows its passage through the small tight junctions of the villi while the larger cellobiose passes through the larger tight junctions of the crypts.

Finally, we did find an increase in cellobiose excretion after fluorouracil (5-FU) that was in part prevented by oral glutamine. Although this difference did not reach statistical significance, overall the data indicate increased intestinal permeability after 5-FU, partially prevented by oral glutamine.

B DANIELE
Divisione Di Oncologia Medica B, Istituto Nazionale Tumori, vía M Semmola, 80131 Napoli, Italy
bdaniele@irso-oncology.it

Fig 1 Disease manifestation according to residual cystic fibrosis transmembrane conductance regulator (CFTR) function as a result of the combination of severe or mild CFTR genotype. CF, cystic fibrosis; PS, pancreatic sufficiency; PI, pancreatic insufficiency; CB/IVB, congenital absence of the vas deferens.

British and US Caucasian, but not in Australian or Spanish AP patients.

Up to now no environmental or genetic cofactor was identified in patients with mutant CFTR alleles associated IP, suggesting that impairment of CFTR function alone may be enough to induce pancreatitis. On the other hand it may be speculated that patients with an abnormal CFTR allele, who develop pancreatitis in conjunction with alcohol abuse, may be characterised by a higher residual CFTR function, which by itself is not capable of inducing pancreatitis.

Therefore, to delineate the genetic background of pancreatic disease in AP it seems to be more appropriate to investigate the prevalence of uncommon mild variants (“atypical mutations”) in large cohorts of AP patients than to test for the more common (“severe, typical”) mutations of the CFTR gene in small patient groups. It has to be considered that the test kits for CFTR mutations often used in routine screening are usually designed to detect the more severe CF mutations. This would result in missing a substantial number of patients with PS. It may be speculated that the CFTR mutations, as suggested by preliminary data on more comprehensive genetic testing in patients with ICP.

J OCKENGA
Department of Gastroenterology, Medical School Hannover, 30625 Hannover, Germany
M STUHRMANN
Department of Human Genetics, Medical School Hannover, 30625 Hannover, Germany
M P MANNS
Department of Gastroenterology, Academic School Hannover, 30625 Hannover, Germany

Correspondence to: Dr J Ockenga
Ockenga.Johann@mh-hannover.de


Reply
EDITOR,—We agree with the view of Ockenga et al that from an ideal research perspective a complete analysis of the cystic fibrosis transmembrane conductance regulator (CFTR) gene should be performed for cases of pancreatitis before a definitive statement on

www.gutjnl.com
We first described the strong correlation between obesity and serum TNF-α in 1998. Adipose tissue synthesises a number of proinflammatory cytokines. The negative correlation found in the Adelaide study is surprising given the findings in larger studies of non-NASH subjects and may be due to the small study numbers and not correcting for modest alcohol intake.

Alcohol consumption is considered a risk factor for the development and progression of liver disease in patients with fatty livers. We previously showed a strong negative correlation between any alcohol consumption and serum TNF-α levels in a general population sample. Moderation of alcohol consumption is known to suppress TNF-α production by monocytes, possibly by suppressing post-transcriptional TNF-α production. Furthermore, alcohol also has effects on TNF-α function mediated via high density lipoprotein (HDL). Alcohol enhances HDL levels by stimulating lipoprotein lipase activity in adipose tissue. HDL not only inhibits TNF-α release from macrophages but also protects certain cells against TNF-α induced damage.

If TNF-α is important, then modest alcohol intake should be protective via suppression of TNF-α. This raises the possibility that TNF-α is not important in early steatohepatitis.

In defining patients with NASH, alcohol consumption must be rigorously excluded. In the Adelaide study, 10 of 22 patients drank a total of 20 g of alcohol per day; however, even modest amounts of alcohol have effects on TNF-α levels and function.

The known interaction between alcohol and obesity in the pathogenesis of fatty liver and steatohepatitis suggests that investigators must look to factors other than TNF-α in studying the early pathogenesis of this condition. In the same way that altered cytokine homeostasis has been implicated in alcoholic liver disease, NASH is probably caused by changes to more than one proinflammatory cytokine. Interleukin 6 (IL-6) is a proinflammatory cytokine, a hepatocyte stimulating factor and function of inflammatory cytokine, a hepatocyte stimulating factor and inhibitor of hepatic apoptosis. It has been suggested that hepatic steatosis is due to the rate of hepatocyte apoptosis becoming insufficient to match the rate of hepatocyte proliferation. IL-6 induced liver regeneration may render the liver more susceptible to the effects of other insults. Unlike TNF-α, serum IL-6 exhibits a positive correlation with both obesity and alcohol intake (fig 1). So far IL-6 has not been studied in the aetiology of NASH.

Future studies examining the link between TNF-α and NASH will need to rigorously control for alcohol consumption and assess many other aspects of the inflammatory cytokine network.

A POULLIS
Mayday University Hospital, Thornton Heath, Surrey, UK
Correspondence to: M A Mendall.
mike.mendall@mhc-tr.sthames.nhs.uk

Reply
Editor,—Our recent paper found increased small bowel bacterial overgrowth (50% versus 22%) and twofold increased systemic levels of tumour necrosis factor α (TNF-α) in patients with non-alcoholic steatohepatitis (NASH) compared with control age and sex matched subjects (Gut 2001;48:206–11). Poullis and Mendall question the finding of elevated TNF-α levels in blood in NASH subjects and quote their own work of elevated TNF-α levels in obese non-NASH subjects.1 There was no correlation between TNF-α levels and obesity in our study whereas their study showed a correlation with obesity. How can this be explained? The question comes down to whether TNF-α is being produced predominantly in adipose tissue or in the liver, and which of these contributes to elevated systemic levels. At the moment this cannot be resolved. TNF-α will need to be investigated in liver biopsies and TNF-α levels sampled from the hepatic vein (not entirely impossible). The same should be done in animal models of obesity. In the meantime, it would be important to ascertain what proportion of obese patients have unrecognised NASH and whether this could explain the elevated TNF-α levels in obesity. Several lines of evidence suggest TNF-α is upregulated in the liver in alcoholic liver disease and presumably this is reflected in increased serum levels. We doubt therefore whether a low (<20 g/day) consumption of alcohol reduces systemic TNF-α levels but this could be formally studied. We have re-examined our data and found that there is no difference in mean TNF-α levels between those who


Reply
reported no alcohol consumption and those who drank alcohol. Finally, we would also comment on our recent work that shows that the C\textsuperscript{-}D-xyllose/H\textsubscript{2}-CH\textsubscript{3} breath test is only positive in 60–69% of cases of small bowel bacterial overgrowth, mostly because it depends on bacterial overgrowth being present on the day of testing. Thus small bowel overgrowth may have contributed even more to NASH than indicated in our paper.

\[ \text{alain.wigg@flinders.edu.au} \]

\[ \text{Correspondence to: Dr AJ Wigg, Department of Gastroenterology and Hepatology, Flinders Medical Centre, Bedford Park, SA 5042, Australia.} \]

\[ \text{A J WIGG} \]

\[ \text{A G CUMMINS} \]

\[ \text{Department of Gastroenterology and Hepatology, Queen Elizabeth Hospital, Woodville South, SA, Australia} \]

\[ \text{and Department of Gastroenterology and Hepatology, Flinders Medical Centre, Bedford Park, SA, Australia} \]

\[ \text{Correspondence to: Dr AJ Wigg, Department of Gastroenterology and Hepatology, Flinders Medical Centre, Bedford Park, SA 5042, Australia.} \]

\[ \text{alan.wigg@flinders.edu.au} \]

---

**BOOK REVIEWS**

\[ \text{Dyspepsia: The Clinical Consequences. Edited by V Healey, P Moncur (\$69.50).} \]


Dyspepsia is like pornography—everyone thinks they know what it is but no one can agree on a definition. This is where the analogy ends, however, as there are plenty of books on pornography but few have been written on dyspepsia compared with other areas of gastroenterology. Out of idle curiosity I searched the Internet for books on irritable bowel syndrome and found 25 published in the past five years compared with only three titles specifically on dyspepsia. This is surprising given that dyspepsia represents 50% of a gastroenterologist’s workload and it is Refreshing to see an up to date book on the subject.

The book discusses the epidemiology, pathophysiology, diagnosis, and treatment of dyspepsia in a methodical fashion. There are contributions from an illustrious list of authors many of whom have international reputations in the field of dyspepsia research. By searching the Internet for books on irritable bowel syndrome and found 25 published in the past five years compared with only three titles specifically on dyspepsia. This is surprising given that dyspepsia represents 50% of a gastroenterologist’s workload and it is refreshing to see an up to date book on the subject.

The book discusses the epidemiology, pathophysiology, diagnosis, and treatment of dyspepsia in a methodical fashion. There are contributions from an illustrious list of authors many of whom have international reputations in the field of dyspepsia research. By searching the Internet for books on irritable bowel syndrome and found 25 published in the past five years compared with only three titles specifically on dyspepsia. This is surprising given that dyspepsia represents 50% of a gastroenterologist’s workload and it is refreshing to see an up to date book on the subject.


Nausea is an extraordinarily common and under appreciated symptom that affects patients and non-patients alike. In a North American population, approximately 15% of subjects surveyed had moderate to severe nausea in the past month. Thankfully, the nausea most of us experience is brief and self limited. Almost all of the medical and surgical subspecialties however have patients who are intermittently and chronically nauseated. Unfortunately, the medical response in the study of nausea from a pathophysiological and treatment viewpoint.

The authors of the book seek to change this situation and have produced a most interesting and readable book about nausea and vomiting for students, primary care physicians, and researchers interested in these unique human symptoms. Gastroenterologists with a clinical or research interest in nausea and vomiting will also find this book helpful in that it brings together a large amount of information that is not easily accessible to us.

The book is basically divided into three parts: in the first 14 chapters the relevant anatomy and physiology of nausea and vomiting, various research methodologies, therapeutics, relevant neuropathies, and the economic impact of nausea and vomiting are covered. Next there are 6 chapters on the treatment of nausea and vomiting. Finally, the last chapter in an excellent and extensive essay on nausea and vomiting as an evolutionary response of the ancient reptilian brain. The reptilian brain appears to be responding to an increasing number of nausea/gastrointestinals precipitated by their modern lifestyles, technologies, and therapeutics, as well as specific diseases/disorders. Why is this?

The authors raise many provocative issues. They reject the simplistic notion that nausea and vomiting are regularised as a response to a putative ingestion of toxic substances. This time honoured concept simply does not reflect the many situations where nausea and vomiting occur in the absence of toxic ingestants. A recent emphasis on the role of nausea and vomiting during pregnancy. The authors review an interesting concept that relates nausea and vomiting and gravity, as gravitational forces affect the basic organisation of brain function. Refreshing ideas and perspectives on nausea and vomiting are offered that encompass philosophy and psychology viewpoints, as well as physiology and pharmacology.

Nausea is more debilitating than vomiting. The authors argue that nausea should be clearly separated from vomiting by studying pathophysiological mechanisms and developing therapies. Indeed, vomiting is the cure for nausea (at least temporarily)! Nausea is an “early warning system” evoked as the organism attempts to maintain homeostasis in response to the stimulus. Vomiting is described as an “accident” of cascading stimuli that ultimately overwhelm homeostasis and the inhibitory circuits that prevent the uncontrollable and potentially injurious vomiting reflex.

Gastroenterologists are not the only medical providers dealing with the problems of nausea and vomiting. It is in the air; nausea is everywhere” is a phase I often use when lecturing about the multidisciplinary problem of nausea and vomiting. The second major portion of the book incorporates 18 chapters in which a practical approach to the diagnosis and treatment of nausea and vomiting is described for many medical and surgical specialties. From allergy and immunology to gastroenterology, oncology, surgery, and sports and space medicine these chapters are an introduction to the treatment of nausea and vomiting by various specialists. The authors are a bit uneven in their thoroughness and somewhat redundant in that each specialty ultimately uses similar drugs and comfort techniques for their patients. The tremendous lack of progress in the therapy for nausea and vomiting makes this area an open field for drug and non-drug development.

The final chapter is an extensive essay on nausea and vomiting that encompasses stimulating paragraphs that are well worth reading for any student of nausea and vomiting symptoms. Topics range from the adaptive value of nausea as a warning sign of ongoing problems in the internal/external environment, as marshalling social support for the sufferer, and as a powerful stimulus for problem solving to avoid these symptoms in the future.

I highly recommend this book as thoughtful and thought provoking reading for anyone interested in the common and sometimes debilitating symptoms of nausea and vomiting. The authors provide excellent reviews and new insights that are now necessary to consider in the fight against nausea and vomiting.

Would I feel tempted to buy this book? At £12.00 it is a give away price and an excellent buy. It provides an up to date and easily read guide to our present understanding of the cause, diagnosis, and management of Crohn's disease and ulcerative colitis. It certainly provides an authoritative handbook for specialist registrars or even concerned patients. Its one weakness is in the absence of references - but within 100 pages could one realistically expect to achieve this? Its role as a handbook for a consultant is less clear. Most of the information it contains should be already known to him or her, but it certainly could refresh that knowledge.

Inflammatory bowel disease is laid out in an attractive format with clear subtitles, useful summary tables, and a good range of illustrations. The impact of inflammatory bowel disease on aspects of life such as fertility, sexual relations, education, employment, and the consequences of the disease in childhood are dealt with in a limited way. The growing role of the specialist nurse in counselling and support is not considered in the book although it could provide useful background reading for anyone working in such a role.

I was particularly impressed by the inclusion of such esoteric treatments as arsenic suppositories in the text although this was omitted from the index. Remicaide was also absent from the index but dealt with in the text. In future editions the index could be strengthened by a more uniform inclusion of key terms. Another useful innovation could strengthened by a more uniform inclusion of key terms. Another useful innovation could be the listing of reliable web sites.

With the growth of expensive, lengthy, but definitive textbooks in the text although this was omitted from the index. Remicaide was also absent from the index but dealt with in the text. In future editions the index could be strengthened by a more uniform inclusion of key terms. Another useful innovation could be the listing of reliable web sites.

With the growth of expensive, lengthy, but definitive textbooks in the text although this was omitted from the index. Remicaide was also absent from the index but dealt with in the text. In future editions the index could be strengthened by a more uniform inclusion of key terms. Another useful innovation could be the listing of reliable web sites.

With the growth of expensive, lengthy, but definitive textbooks in the text although this was omitted from the index. Remicaide was also absent from the index but dealt with in the text. In future editions the index could be strengthened by a more uniform inclusion of key terms. Another useful innovation could be the listing of reliable web sites.

With the growth of expensive, lengthy, but definitive textbooks in the text although this was omitted from the index. Remicaide was also absent from the index but dealt with in the text. In future editions the index could be strengthened by a more uniform inclusion of key terms. Another useful innovation could be the listing of reliable web sites.

With the growth of expensive, lengthy, but definitive textbooks in the text although this was omitted from the index. Remicaide was also absent from the index but dealt with in the text. In future editions the index could be strengthened by a more uniform inclusion of key terms. Another useful innovation could be the listing of reliable web sites.

With the growth of expensive, lengthy, but definitive textbooks in the text although this was omitted from the index. Remicaide was also absent from the index but dealt with in the text. In future editions the index could be strengthened by a more uniform inclusion of key terms. Another useful innovation could be the listing of reliable web sites.
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.

An applicant need not be a member of the Society. The recipient will be required to deliver a 20 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.

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, Leinenweberstr. 5, PO Box 6529, D-79041 Freiburg, Germany. Tel: +49 761 15 14 0; fax: +49 761 15 14 35; 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 Diarrheal 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 Diarrheal Disease and Nutrition Research, All India Institute of Medical Sciences, New Delhi. Tel: +91 11 6663822; 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 Detlef 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, Middlesex, HA1 3UJ, UK. Tel: +44 (0)20 8235 4046/8; fax: +44 (0)20 8235 4039; email: stmarks@ic.ac.uk; website: www.stmarks hosp-ital.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, Ciatat de los Arts i les Ciencies, Avda. Instituo Obrero, s/n, 46013 Valencia, Spain. Tel: +34 96 197 44 66; fax: +34 96 197 44 70; email: catedrasg@cac.es

42nd Annual Conference of the Indian Society of Gastroenterology
This conference will be held on 23–29 November 2001 in Lucknow, India. The programme includes two pre-conference symposia (on gastrointestinal motility and scientific communication, on 23 November), a one day postgraduate course or CME (24 November), and an endoscopy workshop (28–29 November). Further information: Dr S R Naik, Department of Gastroenterology, SGPGI, Lucknow 226014, India. Tel: +91 522 440700 or 440800, ext 2400; fax: +91 522 440708 or 440017; website: www.sgpgi.ac.in/conf/igsg2001.html

41st St Andrew’s Day Festival Symposium on Therapeutics
This will be held on 6–7 December 2001 in Edinburgh, UK. Further information: Ms Eileen Strawn, Symposium Co-ordinator. Tel: +44 (0)131 225 7324; fax: +44 (0)131 220 4393; email: e.strawn@rcpe.ac.uk; website: www.rcpe.ac.uk

14th Intensive European Course of Digestive Endoscopy
This course will be held on 17–18 December 2001 in Strasbourg, France. Further information: Michele Centonze Conseil, 6 bis rue des Cendriers, 75020 Paris, France. Tel: +33 1 44 62 68 80; fax: +33 1 43 49 68 58; email: mail@m-centonze-conseil.com
Ulcerative colitis is more strongly linked to chromosome 12 than Crohn's disease

M PARKES, J SATSANGI, D P JEWELL, D E WEEKS, M M BARMADA and R H DUERR

Gut 2001 49: 311-312
doi: 10.1136/gut.49.2.311

Updated information and services can be found at:
http://gut.bmj.com/content/49/2/311

These include:

References
This article cites 10 articles, 1 of which you can access for free at:
http://gut.bmj.com/content/49/2/311#BIBL

Email alerting service
Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

Notes

To request permissions go to:
http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to:
http://group.bmj.com/subscribe/