LETTERS

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The editors will decide as to whether to also publish it in a future paper issue.

Long term follow up of Helicobacter pylori induced gastric diffuse large B cell MALT lymphoma following eradication treatment alone

I was interested to read the article by Alsohan and colleagues on the long term follow up of gastric diffuse large B cell lymphoma after eradication of Helicobacter pylori (Gut 2003;52:507–9).

Gastric lymphomas represent approximately 5% of all gastric malignancies and are frequently due to mucosa-associated lymphoid tissue (MALT) B cell gastric lymphomas. Acquired MALT due to infection provides the tissue of origin for the B cell lymphoma. Monoclonal proliferation of B cells in the germinal centres of lymphoid tissue with epithelial invasion—"lymphoepithelial lesions"—are the histological hallmark of MALT lymphoma. H pylori induced chronic gastritis through genetic mutation of trisomy 3 and 18 leads to the cal hallmark of MALT lymphoma.

Eradication of H pylori with triple therapy (two antibiotics and double dose proton pump inhibitor) is curative for low grade gastric MALT lymphoma. There are reports of long term studies in the literature from the major centres around the world on the efficacy and safety of this modality of treatment for low grade MALT lymphoma.

District General Hospital (DGH) experience of treating MALT lymphoma is limited due to the rarity of the disorder. However, MALT lymphoma can be managed at a DGH with long term follow up. Regular endoscopic surveillance is required following eradication of H pylori.

Primary diffuse large B cell gastric lymphoma (previously known as high grade MALT lymphoma) is not considered treatable with antimicrobial agents alone. I agree with the authors that it is important to differentiate between patients who may benefit from H pylori eradication as a single modality of treatment and patients who require conventional chemotherapy in this group. The authors have cautioned that although some patients with diffuse large B cell gastric lymphoma might benefit from eradication treatment, this should not be considered standard therapy at present.

However, it was encouraging to note that high grade gastric MALT lymphoma can be treated with a single modality of antibiotic eradication of H pylori, provided the patient is willing to undergo close observation and endoscopic surveillance. This is particularly pertinent for a DGH to heed this message as in a rare situation of being faced with a high grade gastric MALT lymphoma, one would feel confident to try antibiotic eradication of H pylori alone with careful endoscopic surveillance, as often is employed in the case of low grade gastric MALT lymphoma.1

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References

Is hepatobiliary scintigraphy insensitive for the diagnosis of sphincter of Oddi dysfunction?

We read with interest the article by Craig and colleagues (Gut 2003;52:352–7) who reported disappointing results on the value of quantitative hepatobiliary scintigraphy (QHBS) in patients with a suspected sphincter of Oddi dysfunction (SOD). As our paper documented, contrary results were referred to,1 we must add a few words of comment.

Firstly, it should be emphasised that in patients with SOD there is an up to fivefold risk of post-endoscopic retrograde cholangiopancreatography and post-manometry pancreatitis, and therefore there is a strong need for any objective non-invasive method. Hence it is crucial to know whether QHBS can be applied to predict abnormal manometric results. Two European groups recently published concordant results2 which clearly showed abnormal results of QHBS and endoscopic sphincter of Oddi manometry (ESOM). These findings and those of Craig et al are so different that there must be some explanation. We believe this may be due to differences in study design and cholecystokinin (CCK) administration in particular.

In fact, the Australian group changed the CCK augmentation method during QHBS, as originally suggested by Sostre and colleagues,3 whereas Sostre’s group injected a short three minute bolus of 20 ng/kg/body weight of CCK octapeptide (CCK-OP), completed 12 minutes before initiation of QHBS, Craig et al infused 20 ng/kg/body weight CCK-OP over 45 minutes, starting 15 minutes before QHBS, and continued the infusion with sphincter of Oddi dysfunction which may benefit from treatment. At present, we are developing a new catheter assembly system for manometric recording of the sphincter of Oddi, which we believe will eliminate the risk of pancreatitis. This catheter may replace the manometry and may become the new standard while we await the development of non-invasive reproducible diagnostic tests of sphincter of Oddi dysfunction.

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References
Table 2  Sensitivity and specificity of scintigraphic results as compared with manometry

<table>
<thead>
<tr>
<th></th>
<th>Sensitivity (%)</th>
<th>Specificity (%)</th>
<th>PPV (%)</th>
<th>NPV (%)</th>
<th>Accuracy (%)</th>
</tr>
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<tbody>
<tr>
<td>QHBS positive</td>
<td>79</td>
<td>71</td>
<td>88</td>
<td>55</td>
<td>77</td>
</tr>
<tr>
<td>CBD=12 mm</td>
<td>42</td>
<td>86</td>
<td>89</td>
<td>35</td>
<td>54</td>
</tr>
<tr>
<td>Abnormal LFT</td>
<td>26</td>
<td>71</td>
<td>71</td>
<td>23</td>
<td>38</td>
</tr>
</tbody>
</table>

QHBS, quantitative hepatobiliary scintigraphy; CBD, common bile duct; LFT, liver function test; PPV, positive predictive value; NPV, negative predictive value.

Worsening of steatosis and fibrosis progression

We read with great interest the article by Castera and colleagues (Gut 2003;52:288–92) and acknowledge that worsening of steatosis in chronic hepatitis C is associated with fibrosis progression. However, in our view there are no data supporting a causal role for this clinical association or any specific relation of this finding to chronic hepatitis C.

Firstly, the authors provide no explanation as to why steatosis worsened in patients under consideration. Overweight, diabetes, and alcohol consumption are the main causes of fibrotic liver disease. There are no data throughout the study indicating whether patients in whom steatosis worsened simply gained weight or developed any of the complications associated with insulin resistance. The latter can develop within the course of liver injury before cirrhosis is present or be epidemiologically linked to infection by hepatitis C virus (HCV) for reasons that have yet to be determined. High serum glucose, as well as diabetes, are associated with liver fibrosis progression and might contribute to enhanced fibrogenesis. However, for alcohol consumption, a thorough evaluation is needed before ruling out the possibility of even slight increases in daily alcohol consumption translating over the course of several years, into enhanced steatosis. There is a theoretical possibility that progression of steatosis reflects the natural course of HCV infection if steatosis were to occur later than the necroinflammatory lesions defining chronic hepatitis.

However, as current knowledge stands, this is purely speculative and also, the outcome of this study is that patients in whom steatosis progressed had a longer duration of infection than those in whom it did not. Hence there appears to be no data in this study suggesting that progression of steatosis is HCV-related or that confounding proteagogenic factors have been ruled out.

The second issue is that it has not been made entirely clear what “worsening” of steatosis means. This was defined as an increase of at least one point on a grading scale that is not evenly distributed (0%; 1–10%; 10–30%; >30%). Since many patients had no steatosis on the biopsy, such a definition would mean that in most cases an increase from 0% to 5% would be qualified as “worsening” of steatosis. This may explain the authors’ statement that “there were less patients with progression of steatosis than patients with steatosis progression between the two biopsies”. In any event, the biological relevance of minor increases in the amount of steatosis appears highly improbable, especially if the total amount of steatosis at the second biopsy was not associated with the amount of fibrosis, as noted in this study. This biological relevance could have been strengthened had the authors provided quantitative data on a correlation between progression of steatosis and progression of fibrosis.

Although the idea that steatosis progresses rather than that the amount of steatosis associated with fibrosis progression warrants further study, it is hard to reconcile with lessons from non-alcoholic fatty liver disease where patients with massive steatosis do not develop liver fibrosis although they obviously experienced steatosis progression. This argues against a simple and direct link between steatosis and fibrosis. We propose an alternative view in which both steatosis and fibrosis are the result of a common underlying condition, insulin resistance, which operates through proinflammatory mediators to enhance fibrogenic pathways to promote steatosis.

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Correspondence to: Dr V Ratziu; vratziu@hesear.fr

References
6 Fehrenbach H, Weiskirchen R, Kasper M, et al. Up-regulated expression of the receptor for advanced glycation end products in cultured rat hepatic stellate cells during...
Colorectal screening guidelines in acromegaly

We write with concern regarding the recent "new colorectal screening guidelines for colon and polyps in patients with acromegaly" (Gut 2002;51(suppl V):V13–14). While there is little doubt that patients with acromegaly have an increased risk of developing colorectal cancer, the exact nature of this risk is far from clear. The endocrine literature has witnessed a significant debate, polarising two separate views. Jenkins and Fairclough advocate screening, while Renesch and colleagues suggest that the risk of colorectal cancer formation does not warrant screening or surveillance.1 The recommendations by Jenkins and Fairclough for a single screening programme, endorsed by the BSG and ACBP, are based largely on a series of 222 patients enrolled in a colonoscopy programme in one centre. The principal finding of this study was a 13–14-fold increase in the risk of colorectal cancer in acromegics relative to the general population. This is at odds with larger studies (e.g. 662 vs 1041, n = 1634) which overall indicate an increased colorectal cancer risk of 2.5–3-fold. Jenkins and Fairclough advocate an intensive screening protocol beginning at 40 years of age (the age of the youngest case of this 22 case series occurring at 39 years although mean age of the 10 patients with cancer was 67 years). They advocate repeat colonoscopy at five years, or three yearly if at increased risk (as determined by adenoma at initial colonoscopy or increased IGF-1 levels). Renesch et al however conclude that there is no increased incidence of colorectal cancer for acromegalic patients with an apparently normal control population (generated from postmortem and colonoscopy data). These data may differ because of the control population used; this study may also explain why their incidence increases with age. Based on the current literature, an independent view is that patients might benefit from a single sigmoidoscopy or colonoscopy at approximately 55 years of age. However, no study suggests a risk of proximal neoplasia that warrants the risks and difficulties of a colonoscopy. Guidelines for familial colorectal cancer screening suggest that colonoscopy surveillance is only warranted for a lifetime risk of 1 in 10 or greater.

It is therefore both worrying and disappointing that the published guidelines reflect only one point of view of a very polarised debate. This may reflect the process of guideline formation in which experts were requested to submit guidelines.1 At present there is insufficient data to advocate an intensive colorectal cancer screening programme for patients with acromegaly. The increased risk of colorectal cancer is modest and the potential risk of colonoscopy in acromegalic patients is considerable. These guidelines need to be challenged before gastroenterologists are forced into a practice which is not evidence based and may be detrimental to patient well being.

References

Authors’ reply
We thank Drs Perry and Kane and Professor Stewart for their contribution to the debate on this topic. The correspondents acknowledge that patients with acromegaly have an increased risk of developing colorectal cancer; the question really concerns the magnitude of the cancer risk and the relative risk of colonoscopy.

As stated in the guidelines, our recommendations were based on aggregated data from a total of 13 prospective colonoscopy studies involving almost 700 patients with acromegaly. The relative risk was derived from the prevalence of colorectal cancer in patients with acromegaly compared with the asymptomatic matched control populations in the same studies. On this basis, there is also a clear increase in the risk of tubular adenomas. We believe this to be the best quality data on which to base recommendations. We have previously given the reasons why we think that control data generated from the postmortem and colonoscopy studies referred to by Renesch et al are of poorer quality.

One of the major aims of a screening programme is to prevent the development of colorectal cancer by detection and removal of adenomas. We therefore feel that it is sensible to begin this preventative screening at the age of 40 years in this group of patients who seem to be at relatively high risk. We, and we might add, “how extensively to biopsy?” to correctly identify any presence or progression of gastric atrophy.

Concerning the first point on the classification of atrophic gastritis, the current literature is largely biased by the inconsistency of the histological criteria used to categorise atrophy.1 To amalgamate the different viewpoints and also test interobserver agreement in atrophy classification, an international group of pathologists recently published an extensive description of the different phenotypes of gastric atrophy.

The proposed classification also introduces a new diagnostic category (that is, indeterminate atrophy) which necessitates an evaluation of atrophy when high grade inflammation—mostly related to Helicobacter pylori infection—interferes with a reliable assessment of the "loss of appropriate glands".

As for the number and location of biopsies for atrophy assessment, the recommendations of the updated Sydney system seem a suitable compromise between the excessive pathologists’ demands and the operating limits of routine practice.

The question of “where to biopsy?” is more intriguing. No doubt both the oxyntic and antral mucosa need to be tested, but endoscopists too often neglect the recommendation to take an additional angular sample. We studied 504 consecutive H pylori positive patients who underwent gastroscopy for untreated non-ulcer dyspepsia. In all patients, biopsies were obtained (Pentax, Japan: KW2415S) from: (i) oxyntic mucosa (one biopsy from the lesser curvature 4–6 cm proximal to the antrum and one from the greater curvature 8–10 cm proximal); (ii) antral mucosa (one biopsy each from the greater and lesser curvatures, 3–5 cm proximal to the pyloric ring); and (iii) only one additional biopsy from a focal ulcer. Histological categories included the basic distinction between non-atrophic and atrophic gastritis. Two pathologists independently...
assessed the biopsies with a 93% consistency (Fleiss’ K value = 0.91). Table 1 shows the atrophy prevalence according to biopsy location.

In this series, the importance of sampling the incisura angularis is emphasised by the percentage of atrophic gastritis (46%) that would have been missed if sampling had not included the angular mucosa. The NND (number needed to diagnose) values in detecting atrophy calculated for incisura, antrum, and corpus sampling were 2.17, 4.85, and 63.29, respectively.

Because of different pricing policies in different countries, it is difficult to estimate the additional cost of processing and interpreting the extra biopsy sample taken from the incisura angularis. In most countries, however, the cost of the endoscopy procedure is far higher than the cost of histological examination, and the price of an angular biopsy seems amply balanced by the benefit of an appropriate assessment of gastric disease and a suitable evaluation of the patient’s cancer risk.

**Table 1** By site prevalence of atrophic gastritis (distinguishing between atrophy with and without IM) in 504 consecutive *Helicobacter pylori* positive patients. Patients with phenotype “indeterminate for atrophy” and/or “foveolar restricted IM” were included among cases of non-atrophic gastritis.

<table>
<thead>
<tr>
<th>By site prevalence of gastric atrophy (504 H pylori positive consecutive patients)</th>
<th>Gastric atrophy</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>IM absent</td>
</tr>
<tr>
<td>Corpus only</td>
<td>0</td>
</tr>
<tr>
<td>Antrum only</td>
<td>3</td>
</tr>
<tr>
<td>Antrum and incisura angularis</td>
<td>6</td>
</tr>
<tr>
<td>Incisura angularis only</td>
<td>9</td>
</tr>
<tr>
<td>Total</td>
<td>18</td>
</tr>
</tbody>
</table>

**Diversion colitis with a mucosal tear on endoscopic insufflation**

I read with interest the report of Cruz-Correa and colleagues (Gut 2002; 51: 600). They described three cases of collagenous colitis with mucosal tears on endoscopic insufflation and stated that as far as they were aware there were no reports in other gastrointestinal diseases. We would like to present the case of a similar mucosal tear on endoscopic insufflation in a patient with diversion colitis.

A 46 year old Japanese man presented with an acute abdomen caused by ascending colon diverticular perforation. He underwent drainage of the abdominal cavity with loop colostomy. He had been suffering from systemic lupus erythematosus and chronic renal failure for 25 years. He had received more than 90 g of oral steroid at the time of referral and was taking 10 mg/day. After operation, he was free from symptoms and gave no history of haematemesis or blood in stools. On surveillance colonoscopy, the dysfunctional colon mucosa, which was 10 cm away from the loop colostomy, was torn with slight bleeding, and the muscularis mucosal was exposed on endoscopic insufflation with air (fig 1). The lumen of the colon was narrowed and the remaining colon mucosa showed mild colitis with a decreased vascular pattern and oedema. The post endoscopic course was uneventful without any treatment. Routine laboratory investigations revealed: white blood cell count 10600/µl (normal range 4000–9000/µl), haemoglobin 12.2 g/dl (normal range 14–18 g/dl), haematocrit 36.1% (normal range 40–48%), blood urea nitrogen 76.4 mg/dl (normal range 9–21 mg/dl), and serum creatinine 4.29 mg/dl (normal range 0.6–1.2 mg/dl). Cultures for stool pathogens were negative.

Diversion colitis may occur in a part of the bowel that was previously healthy and which has been placed outside the faecal stream because of a proximal stoma. The mechanism of diversion colitis remains unclear but may be associated with changes in the intestinal bacterial flora, absence of essential nutrients, or intestinal toxins. In most cases, there are no symptoms, as in our case. Frisbie et al reported that mucosal erythema and friability were seen in most patients who had undergone diverting colostomy for neuropathic large bowel. Continuous high doses of steroids make human tissue fragile, including the colon mucosa. Taken together, these results
suggest that the mucosal tear in our case may have been attributable to diverticulosis with fragility of the mucosa.

We performed surveillance colonoscopy for patients with a stoma, a dysfunctional colorectum must be surveyed with great care.

The book includes chapters dedicated to the clinical management of liver disease, including viral hepatitis, hepatocellular carcinoma, acute liver failure, and living related liver transplantation. There is no doubt that the authors’ extensive experience in the field of living related liver transplantation will appeal to physicians and surgeons alike but the insights brought to these areas by immunological understanding are less clearly stated.

The book is aimed at both clinicians and scientists, and provides much needed background reading in the rapidly evolving field of hepatology. However, it would be hard going for anyone without a background understanding of basic immunology or molecular biology given the complexity of the science involved. One problem with such a book is assessing the target audience. The rapid evolution of the immunology accounts for many parts of this book being out of date by the time it is published and therefore of less relevance to people working directly in the field. It is perhaps more useful for clinicians or scientists working predominantly in other areas who need an introduction to liver immunology. In this context it would have been helpful to include more explanatory diagrams and a rather more “user friendly” style. However, overall this is a useful book and a good introduction to liver immunology.

Molecular Biology and Immunology in Hepatology. Advances in the Treatment of Intractable Liver Diseases

Edited by T Tsuji, H Tsuchiya, M Zaniya, et al.
Amsterdam: Elsevier Science BV, 2002, b/w, pp 342. ISBN 0 444 50653 5

Dysregulated immune responses underlie the pathogenesis of many liver disorders including not only autoimmune diseases but also viral hepatitis and the chronic inflammatory responses stimulated by alcohol. Thus understanding liver immunology and the molecular signals involved in its regulation are critical if we are to gain insights into the pathogenesis of these diseases and develop novel therapies. Recent advances in immunology have been phenomenal and it is not surprising that many clinicians find it difficult to integrate and understand the importance of emerging immunology research. The editors of this book are to be commended for providing a summary of our knowledge of immunology of the liver and how this informs liver diseases, especially viral hepatitis and autoimmune diseases. They have made a credible attempt to demystify the molecular and immune complexities involved and to distil the field into one concise volume.

Chronic hepatitis C infection is one of the greatest challenges facing hepatologists and gastroenterologists alike in the 21st century and it is now clear that both viral and host immune factors determine the outcome of infection. It is thus not surprising that viral hepatitis accounts for a substantial part of the book, which covers issues from viral genetics and host responses to gene therapy of viral hepatitis and transgenic mouse models of viral progression and hepatocellular carcinoma.

Potential mechanisms of autoimmune liver diseases and the clinical features of the various syndromes are also extensively reviewed. This section of the book demonstrates particularly well how an immunological understanding can provide direct insights into a specific disease.

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Irritable Bowel Syndrome: Diagnosis and Treatment


Gastroenterologists derive job satisfaction from performing endoscopic procedures, establishing diagnoses, and explaining and treating symptoms. Patients with irritable bowel syndrome (IBS) usually require endoscopic procedures, diagnosis is often uncertain, symptoms cannot easily be explained, and there is no effective treatment for IBS. The health service will pay for. Its not surprising therefore that few gastroenterologists relish the prospect of seeing a patient with IBS.

The synopsis claims this text to be an “updated and comprehensive description of the most relevant concepts of colorectal cancer…” I beg to differ.

In the preface, Dr de Leon states that he hopes the volume captures his spirit of the place and is an authoritative and critical text to help a new generation of investigators, and to some extent the volume has achieved this aim. However, he also recognises his own limitations in taking on this daunting task.

The author is not explicit as to who the book is aimed at but refers to a “new generation of investigators” — if this means that the
The book is aimed at giving an overview of colorectal cancer to people working in basic science on colorectal cancer then the book is short enough to be digestible. As an overview of colorectal cancer in the 21st century, a single author could not be expected to do justice to the whole topic and this text is not a comprehensive overview of colorectal cancer.

The best sections of the book are not surprisingly those areas where Dr de Leon has written and published on himself, namely the genetics of colorectal cancer and chemoprevention of colorectal cancer. In many respects the excellent description of the state of the art in these areas highlights the inadequacies in other areas such as pathology, surgical technique, mesorectal excision, adjuvant chemotherapy, and the role of radiotherapy, which are covered in a superficial manner. With the exception of the genetics of colorectal cancer, the reviews of the literature are brief and highly selected. The section on adjuvant chemotherapy and the data presented on faecal occult blood screening are far too brief to do them justice given the current interest worldwide in these aspects of the disease. The section on screening by endoscopic means makes no mention of the potential complications of this modality, and surely deserves at least a mention. Unfortunately, there are also some inaccuracies in the book—for example, the section on screening by CT colonography.

The book is written in a very readable style but with very few illustrations and the quality of the illustrations included is adequate. I found the lack of detail and lack of inclusion of some of the most relevant literature (the last five years) irritating and frustrating. Given the size of the task, I imagine such a book was several years in gestation and this may account for some recent important publications being omitted. It is certainly not a reference book but might provide useful background reading for investigators who are new to the area.

J H Scholefield

NOTICES

Sir Francis Avery Jones British Society of Gastroenterology Research Award 2004

Applications are invited by the Education Committee of the British Society of Gastroenterology who will recommend to Council the recipient of the 2004 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 old or less on 31 December 2004 but 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 2004. Applications (TWENTY COPIES) should be made to the Honorary Secretary, British Society of Gastroenterology, 3 St Andrews Place, London NW1 4LB by 1 December 2003.

British Society of Gastroenterology Hopkins Endoscopy Prize 2004

Applications are invited by the Endoscopy Committee of the British Society of Gastroenterology who will recommend to the Council the recipient of the 2004 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 2004. Applications (TEN COPIES) should be made to the Endoscopy Section Secretary, British Society of Gastroenterology, 3 St Andrews Place, London NW1 4LB by 1 December 2003.

European Helicobacter Study Group (EHSG)

This meeting, on Helicobacter infections and gastroesophageal pathology, will be held on 3–6 September 2003 in Stockholm, Sweden. Further details: Professor Torkel Wadstrom, President- EHSG, Lund University, Department of Infectious Diseases & Medical Microbiology, Division of Bacteriology, Solvegatan 23, SE-223 62 Lund, Sweden. Tel: +46 46 173 241; fax: +46 46 152 364; email: torkel.wadstrom@mmhu.lu.se; website: www.helicobacter.org

Falk Symposium 135—Immunological Diseases of Liver and Gut

This symposium will be held on 12–13 September 2003 in Prague, Czech Republic. Further details: Falk Foundation e.V., Congress Division, PO Box 6529, Leinwenaberstr. 5, 79041 Freiburg/Br, Germany. Tel: +49 761 15 140; fax: +49 761 15 14 359; email: symposia@falkfoundation.de; website: www.falkfoundation.de

The European Society of Parenteral and Enteral Nutrition (ESPEN)

ESPEN will celebrate its silver anniversary at the time of the annual congress, which is to be held on 20–23 September 2003 in Cannes, France. Further details: www.espen.org

XII Falk Liver Week

The XII Falk Liver Week, in honour of Hans Popper’s 100th birthday, will be held on 15–22 October 2003 in Freiburg, Germany. Further details—see Falk Symposia above.

3rd Congress of the European Chapter of the American College of Nutrition

This meeting will be held on 14–15 November 2003 in Göttingen, Germany. Abstract deadline: 01 October 2003. Main topics: Metabolic Syndrome, Plant-genomics, Treatment of Obesity, Hormonal Regulation of the Body Weight, Pediatric Nutrition, Malnutrition, Food-induced Diseases, Food and Allergy. Further details: G Schickedanz, Congress Secretary, Department of Gastroenterology and Endocrinology, University of Göttingen, Robert-Koch-Str. 40, 37075 Göttingen, Germany. Tel: +49 551 396326; fax: +49 551 3919125; email: nutrition2003@med.uni-goettingen.de; website: www.nutrition-europe.org

European Course on Laparoscopic Endoscopy

This course will be held on 18–21 November 2003 in Brussels, Belgium. Further details: Secretary to Professor Cadière, Service de Chirurgie Digestive, Rue Haute 322, Brussels 1000, Belgium. Tel: +32 (0)2 648 07 60; fax: +32 (0)2 647 86 94; email: straeb.asmb@proximedia.be; website: www.straeb-asmb.com

Hong Kong-Shanghai International Liver Congress 2004

This conference will be held on 14–17 February 2004 in Hong Kong. The topic of the conference is the “Liver Diseases in the Post-Genomic Era”. Further details: Ms Kristie Leung, Room 102–105 School of General Nursing, Queen Mary Hospital, 102 Pokfulam Road, Hong Kong. Tel: +852 2818 4300/8101 2442; fax: +852 2818 4030; email: kristieleung@hepa2004.org; website: www.hepa2004.org

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