

PostScript

LETTERS

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Paris staging system for primary gastrointestinal lymphomas

Currently, much effort is put into the development of specific therapeutical approaches that are tailored to specific lymphoma entities. As a consequence, more specific information needs to be collected to support the choice of therapy. In gastric marginal zone B cell lymphoma of MALT-type, the need for specialised documentation of tumour extent in the organ wall has been stressed by the high predictive value for failure of response after *Helicobacter pylori* eradication of infiltration beyond the mucosa, a predictive parameter irrespective of the presence of the t(11;18) translocation.¹⁻³ With new techniques such as endosonography, this infiltration depth can be assessed with improving accuracy. However, the information cannot be translated into the current staging systems. The dissemination patterns of extranodal lymphomas are also essentially different from primary nodal lymphomas.⁶ As tumour stage is one of the most important guidelines in the choice of local therapy (surgery, radiotherapy) and chemotherapy, adequate documentation of tumour localisation in the organ related lymph nodes and beyond is essential.

The Ann Arbor staging system, developed for and routinely used in nodal non-Hodgkin's lymphoma, is not optimal for documentation of the specific relevant features of primary extranodal lymphoma in the gastrointestinal tract. Several modifications and alternatives have been proposed. However, neither differentiation of stage I₁ (confinement of lymphoma to the mucosa and submucosa) from stage I₂ (tumour extension beyond the submucosa according to Radaszkiewicz and colleagues⁷) nor discrimination of stage II₁ (involvement of regional lymph nodes) from stage II₂ (node involvement beyond the regional area, as assessed in the Musshoff modification⁸) is sufficiently serving the demand for documenting all features of lymphoma. To meet these shortcomings, the Lugano classification was constructed by Rohatiner and colleagues⁹ introducing stage IIE for "serosa penetration" without lymph

Table 1 Paris staging system for primary gastrointestinal lymphomas*†

TX	Lymphoma extent not specified
TO	No evidence of lymphoma
T1	Lymphoma confined to the mucosa/submucosa
T1m	Lymphoma confined to mucosa
T1sm	Lymphoma confined to submucosa
T2	Lymphoma infiltrates muscularis propria or subserosa
T3	Lymphoma penetrates serosa (visceral peritoneum) without invasion of adjacent structures
T4	Lymphoma invades adjacent structures or organs
NX	Involvement of lymph nodes not assessed
NO	No evidence of lymph node involvement
N1‡	Involvement of regional lymph nodes
N2	Involvement of intra-abdominal lymph nodes beyond the regional area
N3	Spread to extra-abdominal lymph nodes
MX	Dissemination of lymphoma not assessed
MO	No evidence of extranodal dissemination
M1	Non-continuous involvement of separate site in gastrointestinal tract (eg, stomach and rectum)
M2	Non-continuous involvement of other tissues (eg, peritoneum, pleura) or organs (eg, tonsils, parotid gland, ocular adnexa, lung, liver, spleen, kidney, breast etc.)
BX	Involvement of bone marrow not assessed
B0	No evidence of bone marrow involvement
B1	Lymphomatous infiltration of bone marrow
TNM	Clinical staging: status of tumour, node, metastasis, bone marrow
pTNMB	Histopathological staging: status of tumor, node, metastasis, bone marrow
pN	The histological examination will ordinarily include 6 or more lymph nodes

*Valid for lymphomas originating from the gastro-oesophageal junction to the anus (as defined by identical histomorphological structure).

†In case of more than one visible lesion synchronously originating in the gastrointestinal tract, give the characteristics of the more advanced lesion.

‡Anatomical designation of lymph nodes as "regional" according to site:

(a) stomach: perigastric nodes and those located along the ramifications of the coeliac artery (that is, left gastric artery, common hepatic artery, splenic artery) in accordance with compartments I and II of the Japanese Research Society for Gastric Cancer (1995);

(b) duodenum: pancreaticoduodenal, pyloric, hepatic, and superior mesenteric nodes;

(c) jejunum/ileum: mesenteric nodes and, for the terminal ileum only, the ileocolic as well as the posterior caecal nodes;

(d) colorectum: pericolic and perirectal nodes and those located along the ileocolic, right, middle, and left colic, inferior mesenteric, superior rectal, and internal iliac arteries.

node involvement into the Ann Arbor system. This represents a change in meaning of stage II that originally indicated lymph node involvement. Therefore, the Lugano system is causing more confusion than benefit.

TNM staging for tumours of epithelial origin has also been proposed as an alternative in gastrointestinal lymphoma to describe localised disease. The "T" part of this system pertains to the anatomical structure of the organs and sufficiently fulfills the requirements for staging of local extent of the disease.

The European Gastro-Intestinal Lymphoma Study Group (EGILS) is a multidisciplinary group of investigators, including clinical investigators such as gastroenterologists, medical oncologists, radiotherapists, and pathologists, as well as basic researchers such as cellular and molecular biologists. Several groups from the UK, France, Germany, the Netherlands, Spain, and Austria now take part and have come together regularly since 1999 to discuss and study subjects in epidemiology and molecular and cell biology of gastrointestinal lymphoma. Clinical protocols and trials have been developed and performed

as a collaborative effort. As a result of discussions on staging protocols and reporting systems over the past years, we would like to propose a modified TNM staging system, named after the first venue of the group in Paris. The staging system adequately records: (1) depth of tumour infiltration; (2) extent of nodal involvement; as well as (3) specific lymphoma spreading (table 1). It is adjusted to the gastrointestinal origin of the lymphoma, considering histopathological characteristics of extranodal B and T cell lymphomas. The use of this system in future studies will permit accurate comparison of the reported cohorts and should allow rapid accumulation of good data for proper stratification of patients for risk assessment and treatment options.

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A new cause for CA19.9 elevation: heavy tea consumption

Serum carbohydrate associated antigen (CA19.9) is a reliable tumour marker of biliopancreatic malignancies. A number of benign diseases are also known to be related to CA19.9 elevation. Here we report a case of markedly raised levels of CA19.9 associated with heavy tea consumption.

Case report

A 52 year old woman was referred to our unit for epigastric pain and anorexia of two months' duration. She also complained of nausea and a 2 kg weight loss. Her past medical history was unremarkable. She was a non-smoker and denied alcohol abuse or medication intake but remarked on overconsumption of warm black tea for several months (1.5–2 litres/day). Physical examination was normal.

Laboratory data demonstrated normal ranges for electrolytes, liver function tests,

Table 1 Non-malignant causes of CA19.9 elevation (medline research)

Aetiology	Serum CA19.9 level	
	Mild elevation (<200 U/ml)	High levels (>1000 U/ml)
Obstructive jaundice ^{2,3}		+
Acute liver failure and acute hepatitis ^{4,5}		+
Chronic liver disease ⁴	+	
Alcoholic liver disease ⁴⁻⁶	+	+
Non-alcoholic liver disease ^{4,5}	+	
Pancreatitis		
Acute ^{6,9}	+	+
Chronic ^{8,9}	+	
Diabetes mellitus ¹⁰	+	
Interstitial pulmonary disease ¹¹	+	
Collagen vascular diseases ¹²	+	

Anecdotally reports: hydronephrosis,¹³ endometriosis,¹⁴ splenic cyst,¹⁵ bronchogenic cyst,¹⁶ sigmoid diverticulitis,¹⁷ and hypothyroidism.¹⁸

blood count, urea, creatinin, C reactive protein, amylase, and lipase. Fasting blood glucose, triglycerides, cholesterol, and thyroid function tests were normal. Autoantibodies were negative. Serum CA19.9 was 1432 UI/ml (normal <37), and CEA was 2 ng/ml (normal <5).

Upper endoscopy, colonoscopy, and barium study of the small bowel showed normal results. Abdominal ultrasonography and computed tomography scan found mild enlargement of the body of the pancreas without hepatobiliary abnormalities. Endoscopic ultrasonography showed no pancreatic malignancy or biliary abnormalities. The pancreas was homogenous and mildly enlarged in the body without pathological significance.

Spirometry, chest x ray, bronchoscopy, and bronchoalveolar lavage fluid examination were normal.

The patient was advised to stop tea consumption. Four weeks later she became symptom free and gained the 2 kg weight loss. Another serum CA19.9 assay showed a considerable drop in levels to 42 UI/ml. A rechallenge test was then attempted. The patient restarted tea consumption as previously. Four weeks later CA19.9 increased to 745 UI/ml followed by a fall to 25 UI/ml one month after withdrawal. Follow up one year later revealed no clinical abnormalities. Abdominal and chest computed tomography scan were normal.

Discussion

CA 19.9 (sialyl lewis-a) is a monosialoganglioside with small increases in a number of benign diseases, with highest levels in pancreatic adenocarcinoma, hepatocellular, and cholangiocellular cancer, and also in gastric, colorectal, and occasionally other cancers.¹

Physiologically elevated concentrations are present in many secretions of healthy individuals with the Lewis a positive phenotype in contrast with low serum levels of CA19.9 in Lewis a negative individuals (7–10%).¹

Among non-malignant causes, obstructive jaundice is frequently associated with increases in CA19.9. Relief of jaundice is often associated with a fall in CA19.9 in benign cases and mostly in patients with malignancy.² Normal biliary epithelial cells secrete mucins carrying the epitope of CA 19.9. Unspecific elevation of CA 19.9 in serum reflects both inflammatory hypersecretion and leakage of biliary mucins into serum.³

In addition, there is a strong correlation between serum CA19.9 concentration and standard parameters of cholestasis; alkaline

phosphatase and bilirubin during acute liver failure, acute hepatitis, and chronic liver diseases of any aetiology.⁴⁻⁶

Other benign causes of CA19.9 increases are shown in table 1. The common underlying mechanism for each is probably inflammatory hypersecretion of CA19.9 by normal epithelial cells.

The constituent flavonoids of tea beverage are known to be potent antioxidants. It appears that the flavonoids impact on a wide range of molecular targets that influence cell growth and pathways of angiogenesis.⁷

In this case, the usual causes of CA19.9 elevation were ruled out before a link with the tea beverage was suspected. After tea consumption withdrawal, a dramatic improvement occurred and the patient became symptom free, suggesting the diagnosis of tea intoxication. At the same time, serum CA19.9 levels dropped and a positive rechallenge test proved the relationship between tea overconsumption and raised levels of CA19.9.

To our knowledge, this is the first reported case of markedly raised levels of CA19.9 associated with heavy tea consumption. The mechanism of this relation remains unclear. In addition, the epithelial tissue target involved in CA19.9 secretion by tea overuse is unknown. Individual susceptibility to abnormal CA19.9 secretion triggered by tea overconsumption could not be excluded.

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Non-alcoholic steatohepatitis and hepatic steatosis in patients with adult onset growth hormone deficiency

Adult onset growth hormone (GH) deficiency closely resembles syndrome X.¹ Patients with syndrome X often suffer from obesity, dyslipidaemia, insulin resistance, and hypertension, and hepatic steatosis and non-alcoholic steatohepatitis (NASH) have also been characterised as symptoms of syndrome X.^{2,3} However, the relationship of GH deficiency to hepatic steatosis and NASH remains unclear.

We looked for the presence of hepatic steatosis using computer tomography (CT) in a

Table 1

	Without GH deficiency	With GH deficiency	p Value
Hepatic steatosis (+/-)	0/5	7/6	0.035
BMI (kg/m ²)	21.29(1.2)	23.54(2.21)	0.35
TG (mg/dl)	135.8 (25)	95.4 (10)	0.30
T-ch (mg/dl)	202.5 (18)	180 (6.0)	0.44

Values are mean (SD).

BMI, body mass index; TG, serum triglyceride level; T-ch, serum total cholesterol.

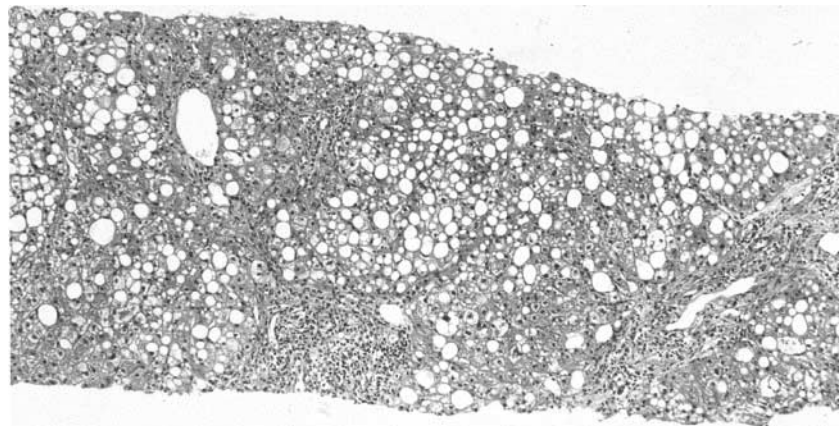


Figure 1 Histology of the liver biopsy. Macrovesicular steatosis, pericellular and centrilobular fibrosis, and ballooned hepatocytes are all observed in this section. (Method: needle biopsy; haematoxylin-eosin staining.)

study of 18 patients with adult onset anterior lobe pituitary hormone deficiency, with or without GH deficiency. We also performed a liver biopsy in one patient with adult onset GH deficiency. None of the patients was positive for hepatitis B surface antigen, hepatitis C virus antibody, antinuclear antibody, or anti-mitochondrial antibody. The criterion used for diagnosing hepatic steatosis was a ratio for the liver/spleen CT value of less than 0.9.⁴ In 13 patients with GH deficiency, seven showed hepatic steatosis (53.8%) while in five patients without GH deficiency no hepatic steatosis was present ($p=0.035$). Body mass index (BMI kg/m²), serum triglyceride level (TG mg/dl), and serum total cholesterol (T-ch mg/dl) were similar in patients with or without GH deficiency (table 1). The significance of the per cent hepatic steatosis was evaluated by χ^2 test. BMI, TG, and T-ch were analysed using an unpaired Student's *t* test.

The male patient on whom we performed a liver biopsy was diagnosed with GH deficiency at 58 years old. He showed hepatic steatosis, measured by CT, at 64 years. Serum aspartate aminotransferase level fluctuated from 20 to 80 U/l. The patient did not have a history of alcohol abuse but was slightly obese (BMI 30 kg/m²). Liver biopsy was performed when the patient was 72 years old, and this showed the presence of NASH (fig 1).

We have demonstrated that hepatic steatosis is more frequently observed in patients with GH deficiency than in those without GH deficiency. Furthermore, by liver biopsy, we have shown that a patient with GH deficiency also had NASH. These results indicate that adult onset GH deficiency is a possible risk factor for hepatic steatosis and NASH.

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Expression of thiopurine methyltransferase in South Asians

Azathioprine and its metabolite 6-mercaptopurine (6-MP) are widely used in the treatment of Crohn's disease, ulcerative colitis, autoimmune hepatitis, and a range of other inflammatory and autoimmune diseases, as well as for prevention of transplant rejection. Thiopurine methyltransferase (TPMT) converts 6-MP to 6-methylMP.^{1,2} Deficiency of its expression predisposes treated patients to bone marrow depression; it may also enhance the efficacy of thiopurines by increasing their metabolism to 6-thioguanine nucleotides.^{1,2} Previous reports in Caucasians have shown that approximately 0.3% are homozygously and 10% heterozygously deficient in TPMT.^{1,2} One study

suggests that the frequency of individuals with the mutant TPMT allele, TPMT*3A, is lower in Southwest Asians than in Caucasians³; this variant allele causes reduced enzyme activity *in vitro*.³ However, there are no data on the phenotypic expression of TPMT in South Asian populations.

We have compared expression of TPMT in South Asian and Caucasian patients attending gastroenterology, renal, rheumatology, and dermatology clinics who were being considered for treatment with thiopurines. TPMT activity was assayed by tandem mass spectrometry for 6-methylMP (assays kindly provided by Purine Research Laboratory, Guy's Hospital, London SE1 9RT, UK). Homozygous deficiency of TPMT was defined as a level < 10, heterozygous as 10–25, and normal as > 25 pmol/h/mg haemoglobin.

Of 83 Caucasian patients, one (1.3%) was homozygously deficient, 10 (12%) were heterozygous, and 72 (86.7%) had normal expression of TPMT. The corresponding values in 77 South Asian patients were 0, 7 (9%), and 70 (91%), respectively.

The prevalence of deficiency of expression of TPMT in South Asian patients resembles that in Caucasians. Use and monitoring of azathioprine and 6-MP should therefore follow similar principles in both ethnic groups.

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Fatigue is associated with high circulating leptin levels in chronic hepatitis C

We read with great interest the recent paper from Piche and colleagues (*Gut* 2002;**51**:434–9) relating leptin levels to fatigue in chronic hepatitis C patients. On the surface, their observation that serum leptin levels correlate with the fatigue impact scale are very intriguing. However, leptin shows a gender based difference and women have circulating plasma leptin concentrations that are at least three times higher than men, even when matched for body fat mass.¹ Thus sex needs to be considered when investigating any association with leptin levels, and multivariate analysis is necessary to reduce the bias caused by this confounding variable. Indeed, in the study by Piche *et al* the fatigue impact scale was found to be significantly higher in females. Therefore, without a multivariate analysis the authors cannot affirm that serum leptin levels are implicated in fatigue development in patients with chronic hepatitis C.

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Mutant K-ras2 in serum

Ryan and colleagues' careful and well conducted study (*Gut* 2003;**52**:101–8) raises once again the interesting issue of whether molecular analysis and knowledge of mutations of the Kirsten ras gene in particular, have a role in the management of patients with colorectal cancer.

The two RASCAL (Kirsten Ras in Colorectal Cancer Collaborative group) studies^{1,2} which eventually enrolled data from 4266 patients from 42 centres in 21 countries showed that although the frequency of Kirsten ras mutations at codons 12 and 13 may vary a little between populations, overall they are only present in just over one third of patients. This is significantly less frequent than quoted by Ryan *et al*. In addition, the RASCAL study also showed that Kirsten ras mutations are not associated with sex, age, tumour site, or Dukes' stage.

Much more importantly, however, they also showed that of the 12 different possible specific point mutations at codons 12 or 13, only a guanine to thymidine mutation, which are found in less than 10% of all patients, is an independent variable for relapse and death. Indeed, the most common mutation, guanine to adenine, exerted an effect on survival barely different from wild-type ras. Others have shown there is a reasonable biological basis for these findings.^{3,4}

As a collaborative group, therefore, we felt it was generally unhelpful to consider Kirsten ras mutations collectively, rather than the effect of each individual mutation separately when considering the prognosis of patients with colorectal cancer. With this in mind, we believe that two crucial questions are left unanswered by the study of Ryan *et al*. Without definitive answers to these, it is unlikely that serum detection of mutated Kirsten ras gene will ever be clinically useful.

Firstly, detection of any new mutation in the serum of a patient who has had colorectal cancer raises the possibility of tumour recurrence, although the presence of a Kirsten ras mutation is not colon specific. However, more than one third of patients in Ryan's study did not clinically relapse despite the presence of a serum mutation for very long periods. Others have also detected tumour DNA in patients for long periods without evidence of overt clinical recurrence.³ Therefore, screening for the presence of Kirsten ras mutations *per se* is unlikely to be clinically helpful very often. However, it would have been very interesting to know whether the patients who did not relapse in this study were those who had what the RASCAL group have defined as "benign" rather than "aggressive" mutations.

Secondly, the authors do not say whether each postoperative serum mutation which

they detected corresponded with the genotype of the primary tumour. While a number of studies have suggested that occasional metastases may carry a different mutation to the primary, this is very uncommon. If the mutation is not the same as the primary tumour, this raises many issues, not least that despite the very careful use of controls, nested polymerase chain reaction techniques may be too sensitive for clinical practice.

For an individual with colorectal cancer, a multitude of factors may shape the clinical course. To improve our knowledge, we must seek to study the most subtle variation in molecular profiles possible, as small differences may be very important. While rigorous small prognostic clinical studies may be intriguing, they may have significant limitations. Some of us have argued previously that there is only one way forward.⁶ All bodies funding prospective therapeutic studies in colorectal cancer should insist that a panel of molecular markers are measured and recorded in all patients using a standardised technique, even if they are not included as an end point, and make these results readily available for subsequent analysis. Studies which are performed prospectively carry the least risk of methodological flaws.

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BOOK REVIEW**Radiological Imaging of the Small Intestine**

Edited by N C Gourtsoyiannis. Berlin: Springer, 2002, €214.0, pp 477. ISBN 3-540-65520-4

Springer Verlag are producing an extensive range of books entitled *Medical Radiology* under the general editorship of Professor AL Baert, and this volume on the small intestine is the latest in the line. Professor NC Gourtsoyiannis is its distinguished editor. This is now a very well established series with topics which vary from organ based (that is, pancreas) to technique based (that is, spiral computed tomography (CT)) of the abdomen. As with all Springer publications, the illustrations are outstanding and the more than 800 in this book are no exception.

Imaging of the small intestine remains the last redoubt for the gastrointestinal radiologist due to the remorseless advance of endoscopy from both ends of the alimentary tract; a point noted in the foreword by Professor Baert. Virtual colonoscopy has rekindled some interest but this is strictly a polyp/tumour detection exercise and the more subtle and interesting colonic abnormalities elude it. Study of the small bowel however retains that interest, both because of the myriad of processes which may affect it as well as the fact that it is not so easy to initiate the "knee jerk" response of biopsy it. It therefore remains more of a deductive radiological process where observations need to be carefully integrated with the clinical state of the patient before a reasoned differential can be given. This coupled with the multitude of imaging techniques, from good old fashioned

barium to ultrasound, CT, and magnetic resonance (MR), etc, still means that it provides a diagnostic challenge.

How does *Radiological Imaging of the Small Intestine* help? The answer is enormously, as it presents an encyclopaedic review of all of the small bowel abnormalities with their attendant investigative techniques. The only problem (and it is one which affects all radiological texts) is the question of whether to go for chapters which reflect technique or for those which reflect pathological conditions. In this case the editor has gone for both and while this results in a superlative volume, there is bound to be some repetition. For example, reference to Crohn's disease or small bowel tumours will appear both in their own section as well as under CT/MR, etc. However, as all of these chapters are written by different authors with their own experiences, this can prove to be an advantage enabling the reader a "second bite of the cherry".

Is there competition in the market place and how does this book hold up? The inevitable comparison has to be made to *Clinical Imaging of the Small Intestine* by Herlinger, Maglinte, and Birnbaum, interestingly also published by Springer. This is the more mature book, now in its second edition, having been originally published in 1989. Its authorship is primarily North American with two European contributors whereas *Radiological Imaging* is primarily a European work with some distinguished North American contributors. Indeed, two of the editors of *Clinical Imaging* are authors. Both books go for the same layout—that is, chapters which are technique based followed by those on pathological states. Both are totally comprehensive and although *Clinical Imaging* is slightly larger (576 pp versus 477 pp) it is less expensive (€164 versus €214). This price differential is most likely to be due to the inclusion of 141 beautiful colour illustrations of both endoscopic views and pathology specimens in *Radiological Imaging*. This is also the most recent publication (2002 versus 1999) and in areas where there is rapid technical change—typically MR imaging—the reader can appreciate the difference. It has to be said that both books fulfil their remit extremely well but your reviewer, if he had to choose, would opt for spending the extra Euros.

Professor Gourtsoyiannis and his team have to be congratulated on producing a superb book that graces the swelling ranks of *medical radiology*. A must for every radiology department and a continuing source of information for any radiologist with an interest in the gastrointestinal system

A H Freeman

NOTICES**New In Vivo Imaging Modalities for Molecular Biology, Cell Biology and Physiology**

This Jacques Monod conference will be held on 31 May–4 June 2003 in Roscoff, France. Further information: Bertrand Tavitian, INSERM M10103, Service Hospitalier Frédéric Joliot, CEA Direction des Sciences du Vivant, Direction de la Recherche Médicale, 4 place du Général Leclerc, 91401 Orsay Cedex, France. Tel: +33 169 867 779; fax: +33 169 867 739; email: tavitian@shfj.cea.fr

Prague Hepatology Meeting

To be held on 5–7 June 2003 in Prague, Czech Republic. Leading speakers from Europe and

the USA will present new ideas and suggestions on pathophysiology, diagnostics, and therapy of liver diseases in ten programme blocks. Further details: Ms Veronica Revicka. Tel: +420 241 445 759; fax: +420 241 445 806; email: veronika@congressprague.cz

8th International Liver Symposium

This symposium will be held on 13 June 2003 in Manchester, UK. Leading speakers from the USA and Europe will present state of the art reviews on recent advances in hepatology. Further details: Professor TW Warnes, Department of Gastroenterology, Manchester Royal Infirmary, Oxford Road, Manchester M13 9WL, UK. Tel: +44 (0)161 276 4316; fax: +44 (0)161 276 8779; email: judith.harrow@cmmc.nhs.uk

Falk Symposia—New Findings on Pathogenesis and Progress in Management of IBD

Two symposia and a workshop will be held on 10–14 June 2003 in Berlin, Germany. Further details: Falk Foundation e.V., Congress Division, PO Box 6529, Leinenweberstr. 5, 79041 Freiburg/Br, Germany. Tel: +49 761 15 140; fax: +49 761 15 14 359; email: symposia@falkfoundation.de; website: www.falkfoundation.de

Gastroenterology and Endotherapy: XXIst European Workshop

This will be held on 16–18 June 2003 in Brussels, Belgium. Further details: Nancy Beauprez, Administrative Secretariat of the Workshop, Gastroenterology Department, Erasme Hospital, Route de Lennik 808, B-1070 Brussels, Belgium. Tel: +32 (0)2 555 49 00; fax: +32 (0)2 555 49 01; email: beauprez@ulb.ac.be

The Association of Coloproctology of Great Britain & Ireland

This annual meeting will be held on 7–10 July 2003 in Edinburgh, UK. Further details: Conference Secretariat, The ACPGBI at the Royal College of Surgeons of England, 35–43 Lincoln's Inn Fields, London WC2A 3PE. Tel: +44 (0)20 7973 0307; fax: +44 (0)20 7430 9235; email: acpghi@asgbi.org.uk; website: www.acpghi.org.uk

European Helicobacter Study Group (EHSg)

This meeting, on Helicobacter infections and gastroduodenal 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 46 152 564; email: Torkel.Wadstrom@mmb.lu.se; website: www.helicobacter.org