Acute toxic gastric mucosal damage induced by Lugol’s iodine spray during chromoendoscopy

Lugol’s solution, named after the French Physician JGA Lugol (1786–1851), has a high affinity for glycogen in non-keratinised squamous epithelium. Since the 1960s when Lugol’s iodine was first used to investigate oesophageal diseases,6 advances in the field of diagnostic endoscopy have resulted in its increasing use to detect early mucosal abnormalities and to target biopsies from unstained areas.4 We have been performing chromoendoscopy using Lugol’s solution for the last 10 years, carrying out 10–15 procedures every year. Here we report the first case of an acute toxic reaction affecting the gastric mucosa.

At gastroscopy of a 67 year old woman with reflux symptoms, a small nodule was noted at the gastro-oesophageal junction together with reflux oesophagitis (LA grade B). Biopsies from the nodule raised the possibility of dysplasia within the squamous epithelium. One month later a repeat examination was performed to reassess the squamous epithelium and target biopsies using Lugol’s chromoendoscopy; 10 ml of 5% Lugol’s iodine was sprayed using an Olympus PL spraying catheter. Multiple biopsies were targeted to the unstained areas together with random biopsies from the distal oesophagus. At the end of the examination, the stomach was again entered to remove any stagnant iodine. The gastric mucosa underlying the pool of iodine was intensely oedematous and haemorrhagic (fig 1A, 1B) The patient did not complain of any symptoms either during or after the procedure. Gastric biopsies confirmed acute oedema of the lamina propria with loss of the superficial epithelium consistent with an acute toxic gastric mucosal injury induced by Lugol’s iodine solution (fig 1C) The oesophageal biopsies showed no dysplasia.

During a follow up examination performed three months later to reassess the lower oesophagus, the gastric mucosa appeared endoscopically and histologically unremarkable.

Chromoendoscopy using Lugol’s solution is not without hazards. Local irritation of the oesophageal mucosa may cause retrosternal pain. General allergic reactions include laryngospasm, bronchospasm, and even cardiac arrest. The concentration of the solution used in studies ranges from 0.5% to 5%, and higher concentrations (3–5%) may be associated with a higher risk of complications. A Japanese study reported that washing the mucosa with sodium thiosulphate may neutralise the iodine solution and reduce retrosternal discomfort. Only two cases of gastric mucosal erosions have been reported after the application of iodine.6

In this case, the histological features of localised oedema and loss of superficial gastric epithelium in the absence of significant inflammatory cell infiltrate supported an acute toxic injury to the gastric mucosa. The toxic reaction was confined to the columnar epithelium in the greater curve of the stomach that was in direct contact with the pooled 5% Lugol’s iodine while the squamous oesophageal mucosa remained unremarkable both endoscopically and histologically. Gastric columnar epithelium may be more susceptible to the toxic effect of Lugol’s iodine and mucosal injury may go unrecognised unless the stomach is re-examined after application of the dye. To reduce the risks, we now use 10–20 ml of 1.5% Lugol’s solution and routinely aspirate the gastric pool before assessing the oesophageal mucosa.

Previous studies have shown that Lugol’s staining is useful in screening for early oesophageal cancer in high risk populations such as patients with previous or current non-oesophageal malignancy and those with a high alcohol intake.7 However, none of these studies commented on the adverse reactions to Lugol’s staining during endoscopy. We suggest that the adverse reactions and safety profile of iodine staining need to be addressed, in particular before recommending its routine use for screening purposes. Also, where it has to be used, a lower concentration of 1.5% may be less toxic to the gastric mucosa and is thus recommended.

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References
Erosions or not in GORD? The potential role of oesophageal cell proliferation

Gastro-oesophageal reflux is an almost universal daily occurrence, but only a small percentage of the population develops gastro-oesophageal reflux disease (GORD) and, among them, a small number develop erosive oesophagitis (ERD) or one of its complications. It is well accepted that the pathophysiology of GORD is related to failure of antireflux mechanisms but several phenomena are not fully explained on the basis of this sequence. There is no apparent relation between damage and the amount and quality of refluxate. It is not known why the same amount of refluxate determines GORD in one patient and not in another. It is also unclear whether there is a relation between these unexplained questions and the possible influence of proliferative responses of epithelial proliferating cells to damage. Hence we evaluated cell proliferation of the oesophageal epithelium using Ki67 immunostaining in normal subjects and in patients with GORD, with or without erosions.

Patients gave written informed consent to participate in the study which was approved by the ethics committee. The design was blinded for epithelial cell kinetic evaluation. Thirty five subjects were enrolled: nine were healthy voluntary controls with normal pH parameters, seven had acid reflux disease (NERD) while 13 had ERD (table 1).

All subjects underwent gastroscopy; six biopsies were obtained within the lower 5 cm of the oesophagus from areas of macroscopically intact oesophageal mucosa. The presence of oesophagitis was graded according to the Los Angeles classification.

| Table 1 | Demographic, endoscopic, pH monitoring, histological, and ultrastructural data of the studied population (normal healthy controls, and gastro-oesophageal reflux disease patients with erosive oesophagitis (ERD) and those with a normal appearing oesophageal mucosa (NERD)) |
|---------------------------------|---------------------------------|-----------------|-----------------|
| **Normal** | **NERD** | **ERD** |
| No of subjects | 9 | 13 | 13 |
| Sex (M/F) | 4/5 | 4/9 | 7/6 |
| Age (y) (mean (SD)) | 38.67 (17.36) | 41.62 (11.77) | 42.54 (13.33) |
| % Time oesophageal pH <4 | 26-63 | 22-59 | 25-65 |
| Endoscopy | | | |
| Normal | 9 | 13 | 0 |
| Mild | 0 | 0 | 0 |
| Moderate | 0 | 0 | 0 |
| Severe | 0 | 0 | 0 |
| Histology | | | |
| Normal | 9 | 13 | 8 |
| Mild | 0 | 0 | 5 |
| Moderate | 0 | 0 | 0 |
| Severe | 0 | 0 | 0 |
| TEM value (mean (SD)) | 0.54 (0.08) | 2.24 (0.53) | 2.39 (0.44) |

**Figure 1** Box plots of Ki67-labelling index (LI), L1 median (bold line in the box), and interquartile range (upper and lower lines of the box) in human oesophageal mucosa of healthy controls and of patients with erosive oesophagitis (ERD) and a normal appearing oesophageal mucosa (NERD). Whiskers indicate lowest and highest values.

p < 0.001
p < 0.01

References

Role of IL-10 promoter haplotypes in Helicobacter pylori associated gastric inflammation

We read with great interest the article by Rad et al (Gut 2004;53:1082–9) on the influence of cytokine gene polymorphisms on mucosal cytokine expression, gastric inflammation, and host specific colonisation in Helicobacter pylori infection. The authors reported an association of the contrainflammatory interleukin (IL)-10 (IL-10) promoter haplotype (GCC), with higher mucosal mRNA levels and colonisation with more virulent cagA+, vacA−S1, and babA2+ strains in 207 patients with H pylori induced chronic gastritis. Rad et al identified pathogenicity genes of H pylori isolates by polymerase chain reaction based techniques from gastric biopsies.

However, the human stomach is colonised by more than one strain of H pylori, which obscures the investigation of germline mutations and host specific colonisation.1 Moreover, within an apparently homogenous population, remarkable genetic differences exist among single colony isolates. The capacity of H pylori to lose and possibly acquire exogenous DNA is consistent with a model of continuous microevolution within a population.2 The evidence exists among single colony isolates. The capacity of H pylori to lose and possibly acquire exogenous DNA is consistent with a model of continuous microevolution within a population.2 The evidence exists among single colony isolates. The capacity of H pylori to lose and possibly acquire exogenous DNA is consistent with a model of continuous microevolution within a population.2

We recruited 614 consecutive Caucasian patients from Northern Germany who underwent gastroscopy with confirmed H pylori infection by rapid urease test or histology. Endoscopic findings and results of histopathological examination of biopsies, classified according to the Sydney classification, were recorded. In total, 316 patients presented with chronic gastritis and served as controls and 124 patients suffered from gastric ulcer. DNA was extracted by standard techniques from 5 ml of EDTA blood. All patients were genotyped for IL-10 −1082, −819, and −592 and TaqMan technology. Samples were recorded and genotypes assigned without knowledge of clinical status. Single marker and haplotype analysis was conducted to assess associations with development of gastric ulcer.

There were no associations between any of the single nucleotide polymorphisms tested and H pylori related pathological findings (data not shown). The proinflammatory low secreting haplotype ATA did not confer a risk factor for the development of an ulcer and the contrainflammatory haplotype GCC did not protect patients from gastric ulcer (table 1). Our results are in agreement with the study of Hida et al who reported higher IL-10 mRNA expression in cagA+ H pylori gastritis, with no relation to endoscopic diagnosis.3 Therefore, we conclude that genetic variations in the IL-10 promoter may influence mucosal cytokine expression but pro- and contrainflammatory haplotypes do not influence the clinical course of gastric inflammation, at least in Northern Germany. Furthermore, we suggest that association studies of germline polymorphisms with the outcome of chronic H pylori infection should focus on clearly defined phenotypes such as ulcer disease, gastric carcinoma, or primary gastric B cell lymphoma.

Table 1 Haplotype analysis of the interleukin 10 (IL-10) promoter in 440 patients with chronic gastritis and gastric ulcer disease

<table>
<thead>
<tr>
<th>IL-10 promoter</th>
<th>Chronic gastritis (n=316)</th>
<th>Gastric ulcer (n=124)</th>
<th>OR</th>
<th>p (χ²)</th>
</tr>
</thead>
<tbody>
<tr>
<td>−1082 −819 −592</td>
<td>48.0%</td>
<td>48.3%</td>
<td>1.01</td>
<td>0.939</td>
</tr>
<tr>
<td>G C C</td>
<td>23.6%</td>
<td>24.2%</td>
<td>1.03</td>
<td>0.862</td>
</tr>
<tr>
<td>A T A</td>
<td>28.2%</td>
<td>27.5%</td>
<td>0.97</td>
<td>0.842</td>
</tr>
</tbody>
</table>

Conflicts of interest: None declared.

References


Gastric ulceration due to chronic mesenteric ischaemia treated by stenting of the inferior mesenteric artery

We report a case of gastric ulceration due to visceral ischaemia treated successfully by stenting of the inferior mesenteric artery (IMA) alone. Gastric ulceration has very rarely been described as a result of chronic mesenteric ischaemia.1–3 Four of the five cases described in these reports were treated surgically and one by angioplasty to the superior mesenteric artery (SMA). All were reported to have successful resolution of ulceration. To our knowledge, there is no other case of successful stenting of the IMA alone, with resolution of gastric ischaemia described in the medical literature.

Our patient was a 50 year old woman presenting with abdominal pain, loss of appetite, vomiting, and weight loss. Pain was maximal in the epigastrium and precipitated by meals. Apart from being a smoker there was no other significant medical history. She was empirically started on omeprazole. Oesophagogastroduodenoscopy (OGD) revealed multiple serpiginous ulcers affecting the body of the stomach with extension to the cardia (fig 1D). Histology from the ulcers demonstrated ulceration with regenerative hyperplasia with no evidence of Helicobacter pylori infection. An abdominal computed tomography scan showed non-specific thickening of the pylorus and first part of the duodenum but was otherwise normal. A small bowel follow through revealed no abnormality. Fasting gut hormone levels, including ghrelin level, after stopping omeprazole were normal and a vasculitis screen (including serum ANCA) was negative.

Her abdominal pain was controlled by morphine 120 mg/day. A repeat OGD 10 weeks after treatment with omeprazole 40 mg once daily showed continuing ulceration with no improvement since the previous examination. Further histology showed similar findings as before.

Abdominal angiography demonstrated complete occlusion of the SMA origin (fig 1B), and tight ostial stenoses of the IMA (fig 1A) and coeliac axis. The SMA branches filled sluggishly and were reconstructed almost exclusively via the left colic branch of the IMA.

Attempts to bypass the coeliac axis stenosis and proximal SMA occlusion were
unsuccessful. The IMA was catheterised; initial pressure measurements demonstrated a mean IMA pressure of 20 mm Hg (mean pressure gradient between the IMA and the aorta of 70 mm Hg) which persisted after angioplasty with a 5 mm balloon. A 5 mm diameter, 16 mm long balloon mounted stent was therefore placed across the ostial stenosis (fig 1C) resulting in marked improvement of the angiographic appearance and almost complete obliteration of the mean pressure gradient. The patient was commenced on aspirin and warfarin and an international normalised ratio of 2 was maintained. She was weaned off the opiate analgesia and was discharged on omeprazole, aspirin, warfarin, and amitriptyline.

Her symptoms resolved completely and amitriptyline and warfarin were discontinued three months later. A repeat OGD showed complete healing of the ulcers. She was continued on aspirin 150 mg and omeprazole 10 mg daily and advised to stop smoking, but unfortunately was not successful in doing so. She has been followed up for 18 months and has had no recurrence of her symptoms.

The interest in this case lies in the fact that only the IMA needed to be stented to achieve an appropriate vascular supply to the stomach, despite severe occlusions and reduced flow in the coeliac and mesenteric axes. This has not been described before, and demonstrates that minimally invasive radiological stenting of only one territory of the mesenteric axis, even the IMA alone, can provide enough blood flow to treat the complications of chronic mesenteric vascular disease. This case is a reminder that chronic mesenteric vascular disease should be considered as a cause of resistant gastric ulceration. This case also demonstrates that minimally invasive radiological stenting of only one territory of the mesenteric axis, even the IMA alone, can provide enough blood flow to treat the complications of chronic mesenteric vascular disease.

Figure 1  (A) Inferior mesenteric artery (IMA) stenosis, (B) superior mesenteric artery occlusion, (C) stented IMA, and (D) gastric ulcer.

Diagnostic radiation exposure and cancer risk

Diagnostic and therapeutic radiological investigations are an essential part of the workup of patients with a number of clinical problems across a variety of medical specialties. Although new non-ionising ray technologies have started to replace traditional investigations these have not lead to a reduction in radiation exposure. In contrast, based on global statistics and projections, radiation exposure of patients is increasing, in particular as a result of new indications and use in cross sectional imaging. In addition, multiple investigations of patients with chronic disease can lead to substantial individual radiation exposure as surgical practice increasingly relies on the use of cross sectional imaging to aid diagnosis and treatment. New imaging techniques, in particular computed tomography (CT) colonography, have become attractive alternatives to conventional colonoscopy. However, the necessity for both prone and supine scanning means that radiation exposure is double that of a conventional abdominal scan which can lead to a theoretical increase in the risk of exposure related cancer and death.

The awareness of hospital doctors about radiation exposure and associated cancer risk is poor. From personal experience, many gastroenterologists involved in diagnostic and therapeutic procedures using ionising radiation do not routinely wear full protective clothing (0.35 mm lead equivalent aprons, thyroid shield, lead glasses) on a regular basis. This is also shown in a survey of endoscopic retrograde cholangiopancreatography (ERCP) practices by Campbell et al in which only 52% of respondents reported wearing a thyroid shield all of the time. An audit of radiation exposure to personnel performing ERCP found that both patients and staff are exposed to significant radiation exposure. This was equivalent to an estimated additional lifetime cancer risk of between 1 in 3500 and 1 in 7000. These studies highlight the substantial underestimation by medical staff of patient and operator related radiation induced cancer risk.

The National Radiological Protection Board (NRPB) has recently revised the radiation dose for typical x ray examinations. For example, an abdominal/pelvic CT scan would typically lead to an effective dose of 10 mSv, which is an equivalent of 4.5 years of natural background radiation. This radiation exposure was estimated to carry a 1:2000 risk of fatal cancer in the 16–69 year old patient population (personal communication from NRPB). For older patients, this may be halved but for younger patients increased up to fivefold. Put another way, this is equal to 250–300 fatal cancers for every 1 million abdominal/pelvic CT scans.

These values are calculated using a probability coefficient, which was developed by the International Commission on Radiological Protection (ICRP), based on historical epidemiological cohorts and other research. It is worth noting that the cancer risk attributed to radiation exposure has constantly risen over time as longer term follow up information from the screening and Nagasaki cohorts have become available. It is therefore possible that radiation attributed cancer risk will continue to rise in the future.

In the UK, diagnostic x rays related cumulative risk of cancer to age 75 years was recently estimated at 0.6%, which is equivalent to approximately 700 cases of cancer per year. In the same report, this rate was estimated to be up to five times higher for countries with a higher use of diagnostic x rays. The British Society of Radiology has made specific recommendations to reduce radiation exposure. Adherence to these guidelines may well be an explanation for the comparatively low frequency of diagnostic x ray in UK practice.

Clinicians should use these recommendations when considering radiological investigations. Protection of operators and nursing staff using recommended protective clothing should also be followed. Change in clinical practice may not be easy to achieve as, for example, endoscopic capacity to reduce the number of alternative radiological investigations such as barium enemas, is limited. In contrast, barium enemas are often used to reduce the demand on endoscopic services. New technologies and methods may well reduce radiation exposure. Examples in gastroenterology include magnetic resonance cholangiopancreatography or ultrasound instead of ERCP and magnetic resonance enteroclysis instead of small bowel enemas. Technological advances, in particular low dose helical CT, may reduce radiation exposure by 40–70%. However, availability of these technologies is limited or only slowly increasing and it is therefore unlikely that their use will influence radiation exposure in the near future.

What remains is the judicious use of radiological investigations and close liaison with radiologists in order to keep the radiation exposure of patients and staff as low as possible.

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Conflict of interest: None declared.

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Adalimumab use in pregnancy

Infliximab, a chimeric antibody to tumour necrosis factor alpha (TNF-α), has demonstrated efficacy for the induction and maintenance of remission in patients with Crohn’s disease. Antibodies to the chimeric component of infliximab can lead to infusion reactions and possible loss of response. A human recombinant monoclonal antibody to TNF-α, adalimumab, has recently demonstrated safety and efficacy for induction of remission in Crohn’s disease. It has also been effective in patients who have lost response to infliximab. Currently, this drug is FDA approved for the treatment of rheumatoid arthritis but it is being administered off label for the treatment of Crohn’s disease and rheumatoid arthritis. Am J Gastroenterol 2004; 99:2385–92.


Can gastro-oesophageal reflux be predicted while advancing the endoscope through the laryngeal area?

We read with great interest the article by Mullihan et al regarding examination of the laryngopharyngeal area during upper gastrointestinal endoscopy, after being trained for examination of these anatomical structures (Gut 2004; 53:1232–4). Twenty six laryngeal procedures were examined in 1311 cases, the most important of which was demonstration of an early supraglottic cancer.

Upper gastrointestinal endoscopy has been performed in children for various indications. In paediatric gastroenterology practice, endoscopy is an important procedure beginning from the mouth. After inserting the endoscope into the oral cavity, the uvula, epiglottis, and concomitant laryngeal cartilages with the vocal cords above are seen. While passing through the epiglottis area, the concomitant laryngitis, oedema, hyperaemia or ulceration of the arytenoids, and laryngeal granulomas can be visualised. Examination of the laryngopharyngeal area is not a routine part of the endoscopic procedure in children.

Although supraglottic cancer is extremely rare among children, a more common problem of the laryngeal area during childhood is gastro-oesophageal reflux (GOR), which affects almost 10% of children. Recurrent upper or lower respiratory tract infections, and weight loss are frequent clinical findings with GOR. Extraoesophageal manifestations of GOR have been identified and recognised more recently over the past decade. The phrase “extraoesophageal reflux” refers to the effects of refluxed gastric material far from the oesophagus. It has been shown that the contents of the gastric juice, including hydrochloric acid and pepsin, are damaging not only to the oesophagus but also to pharyngeal and laryngeal tissues. Resistance of the laryngeal mucosa to refluxed gastric content is due to the presence of buffering effects of saliva which are important factors predicting the severity of laryngeal injury. Gaynor and colleagues1 reported the otolaryngological manifestations of extraoesophageal reflux and stated that the presence of erythema, oedema of the arytenoids and posterior part of the vocal cords, or more chronic changes such as the presence of granulomas might suggest GOR in aetiology.

In our paediatric gastroenterology outpatient clinic, 375 upper gastrointestinal endoscopies were performed in children aged

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three months to 17 years, between 2003 and September 2004. The laryngopharyngeal area was investigated in 207 children during the endoscopic procedure and of these, 40 children had oedema of the vocal cords or arytenoids. Sixteen of these cases were due to caustic material ingestion; the remaining 24 had upper gastrointestinal endoscopy for other indications. Among the 24 cases with laryngopharyngeal pathology, 11 had hyperaemia and mucosal nodularity in the proximal, and 14 in the distal, part of the oesophagus. When the proximal and distal oesophageal biopsies were compared in this group, 37% had proximal and 66% had distal histological oesophagitis. Therefore, the presence of laryngeal oedema made us suspect GOR, and it is now routine for us to take oesophageal biopsies from the upper and lower parts of the oesophagus.

In the study of Mullhaupt et al, the importance of macroscopically noticeable laryngeal lesions during endoscopy among adult patients was emphasised and the most important was reported to be discovery of an early supraglottic carcinoma. Upper gastrointestinal endoscopy is also an important procedure for the diagnosis of GOR and its supraglottic manifestations, if it is performed by an endoscopist who has been trained in the normal anatomy and pathology of the laryngeal area. Thus we agree with inspection of the laryngopharyngeal area, not only for evaluation of malignancies (although seen extremely rare among children) but also for extraoesophageal manifestations of GOR.

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Is stool DNA multigene testing an unreliable strategy for colorectal cancer screening?
The availability of a simple non-invasive test capable of detecting colorectal cancer specific genetic signatures with reasonable sensitivity and specificity might avoid the invasiveness, unpleasant bowel preparation, and risk of bleeding and perforation related to colonoscopy. Molecular marker combinations in faecal DNA testing have been shown to produce high rates of both colorectal cancer and advanced adenoma detection in selected patient populations, and observations from large representative groups are emerging. Imperiale and colleagues have recently reported the results of the first large study of faecal DNA testing in asymptomatic subjects. A total of 4404 average risk adults, who were at least 50 years old, underwent faecal occult blood testing, faecal DNA testing, and colonoscopy, which was considered the reference standard. Comparing test results in a random subgroup of 2907 persons, the authors found that the faecal DNA test was much more sensitive than faecal occult blood testing in detecting colorectal cancer and adenomas with high grade dysplasia. However, the sensitivity for both the faecal DNA panel and faecal occult blood testing was low. In particular, the faecal DNA test detected only 52% of colorectal cancers and 15% of adenomas, rates that were far lower than those previously reported in the literature for multigene target testing.1,2 Furthermore, in the same study, the sensitivity of faecal occult blood testing (13%) was unexpectedly low.

Interestingly, in the results section, Imperiale and colleagues reported that “among 1423 subjects with negative findings on colonoscopy, 79 had a positive faecal DNA panel and 68 had a positive Hemoccult II test, for specificities of 94.4% and 95.2%, respectively.” The question immediately arises as to whether these patients subsequently developed “advanced neoplasia” or precursor polyps and consequently whether the results from the DNA stool test or faecal occult blood test were falsely or truly positive. In fact, colonoscopy could have produced false negative results for several reasons, including misinterpretation of what was visualised or failure to perform adequate biopsy of the lesions seen.3 In this case, it could be intriguing to compare the ability of faecal DNA panel versus that of the Hemoccult II test in predicting the early occurrence of “advanced neoplasia” when colonoscopy misses the disease.

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have described that apart from T cells, activated platelet also express CD40L. Platelets display enhanced levels of membrane bound CD40L in CD and ulcerative colitis patients, and secrete higher amounts of soluble CD40L, compared with healthy controls. These phenomena have biological relevance in terms of intestinal microvascular activation as IBD activated platelets trigger chemokine production, VCAM-1, ICAM-1, and CD40L upregulation, and T cell adhesion to the gut endothelium. Therefore, abnormality of endothelial or platelet CD40 expression would not only block T cell-endothelial interactions but also interrupt platelet-endothelial and platelet-leucocyte cell cross-talk in the gut microvasculature.

Taken together, these observations suggest that the CD40 antisense oligonucleotide used by Gao et al. exerts its beneficial effect not only by disrupting the interaction between CD40 bearing monocytes and endothelial cells and CD40L positive T cells, but also by acting on a much wider array of cell types that the CD40 antisense oligonucleotides appears to be a very promising therapeutic approach to turn off intestinal inflammation, by disconnecting a crucial and almost ubiquitous communication system used by multiple cell types during inflammation.

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Conflict of interest: None declared.

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NOTICES

Masterclass in Crohn’s disease

A masterclass in Crohn’s disease will be held in Oxford on Wednesday 31 August 2005. This masterclass has been designed for consultants and registrars, including those who do not specialise in gastroenterology. Topics will include aetiology, differential diagnosis, and management. The course fee is £110 and board and accommodation is available at Wadham College at extra cost.

Six bursaries will be available for applicants training in gastroenterology or in research posts in British hospitals. For further details and application forms contact: Professor Derek J Jewell, Radcliffe Infirmary, Woodstock Road, Oxford OX2 6HE, UK; tel +44 1865 224829; fax: +44 1865 790792; email: derek.jewell@ndm.ox.ac.uk; website: http://www.medicine.ox.ac.uk/gastro.

Asian Pacific Digestive Week 2005

This will be held on 25–28 September 2005 in COEX, Seoul, Korea. It is hosted by APDW 2005 Organizing Committee in conjunction with Asian Pacific Association of Gastroenterology (APAGE), Asian Pacific Society of Digestive Endoscopy (APSDE), Asian Pacific Association for the Study of the Liver (APASL), and International Society for Digestive Surgery (ISDS). The theme is “Rediscovery of Asia for gastrointestinal diseases”. The abstract submission deadline is 15 June 2005 and early bird registration deadline is 30 June 2005.

For further information contact the Secretary General, Jin-Ho Kim, University of Ulsan, #1510 Hyoysung Olympic County II, 175-12, Chamsil-dong, Songpa-gu, Seoul 138-220, Korea; tel: +82 2 412 0673; fax: +82 2 412 0674; email: jhkim@amc.seoul.kr; website: http://www.APDW2005.org.

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