



The Hong Kong Society of Gastroenterology

香港腸胃學會

June 2008

The Council

President

Dr. YEUNG Yat Wah

Vice-President

Prof. Benjamin CY WONG

Hon. Secretary

Dr. Judy WC HO

Hon. Treasurer

Dr. LAO Wai Cheung

Council Members

Prof. Francis KL CHAN
Dr. SZETO Ming Leung
Prof. Joseph JY SUNG
Dr. YUEN Hon
Dr. HUI Wai Mo
Dr. Vincent KS LEUNG
Dr. YUEN Man Fung
Dr. LAM Chi Ming
Dr. Ambrose CP KWAN

Co-opted Members

Dr. Annie OO CHAN
Dr. Justin CY WU

The Secretariat

Room C, 7/F, Thomson
Commercial Building
8 Thomson Road
Wanchai, Hong Kong
Tel: (852) 2869 5933
Fax: (852) 2869 9533
Email: gastro@hksge.org
Website: www.hksge.org

President Message

Message from Dr. Yeung Yat Wah, President

To achieve our objective to promote the advancement of gastroenterology in Hong Kong, this Society continued to organize the Annual General Meeting & Scientific Meeting in March and the Joint Annual Scientific Meeting in September this year. The Annual General Meeting & Scientific Meeting on March 13, 2008 was very successful and well attended. The Joint Annual Scientific Meeting will be held on September 6, 2008 with both local and overseas speakers.

The Secretariat has moved to Wanchai for economic benefits and will continue to provide administrative support to the Society and serve as a centre of communication between the Council and all members and fellows.

I wish to thank the following friends and contributors to this Society: all fellows and members for their continuous support and contributions; Dr. Hui Wai Mo for editing this Newsletter; Prof. Eamonn Quigley, Prof. Willis Maddrey, Dr. On On Chan and Prof. Ching Lung Lai for sharing with us their scientific updates, local and overseas speakers of our scientific meetings and session chairpersons. On behalf of the Society, I wish to express our heartfelt thanks to friends from the pharmaceutical industry for their generous sponsorship and contributions throughout the years. I look forward to the continued support and active participation of all in the future.

Scientific Updates

Irritable Bowel Syndrome: An Inflammatory Disorder?

Professor Eamonn MM Quigley

Department of Medicine, Cork University Hospital
National University of Ireland
Cork, Ireland

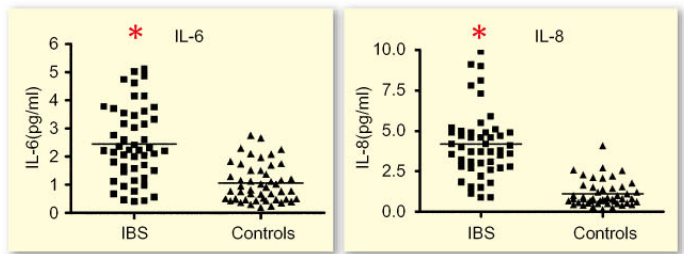
Irritable bowel syndrome (IBS) is a chronic and relapsing condition characterized by dysmotility, visceral hypersensitivity and disturbed gut-brain interactions. Stress, psychological factors and/or food often precipitate IBS, and females, individuals with pre-morbid psychopathology and those with a genetic predisposition appear to have a higher susceptibility to the disorder.¹ Despite this level of understanding of the condition, its aetiology remains unclear. Professor Quigley presented some of the accumulating evidence supporting the concept of IBS as an inflammatory disorder.

Firstly, around 10% to 14% of individuals who suffer an enteric bacterial infection develop post-infectious IBS (PI-IBS).^{2,3} Risk factors for PI-IBS include female gender and persistent inflammation, as demonstrated by elevated numbers of T lymphocytes and enterochromaffin cells in rectal biopsies.² Professor Quigley explained that, although PI-IBS accounts for only a minority of IBS patients, it has clearly established the link between disturbances in the enteric flora, immune activation and the development of IBS symptoms.

Secondly, immune activation and inflammatory responses have been consistently observed in patients with IBS. For example, increased numbers of intraepithelial lymphocytes and activated mast cells have been found in

mucosal biopsies of the rectum, colon and ileum.^{4,5} The positive correlation between the proximity of mast cells to sensory neurones in the mucosa and pain severity provides the direct link between immune activation and IBS symptoms.⁵ Other studies have demonstrated elevated blood levels of proinflammatory cytokines (Figure), and increased protease and defensin excretion in the stool, as well as immune imbalance in the mucosa, of IBS patients.⁶⁻¹⁰

Figure. Elevated plasma levels of proinflammatory cytokines interleukin-6 (A) and interleukin-8 (B) have been observed in IBS patients.⁷



Finally, both quantitative and qualitative changes in the colonic flora (microbiota) have been described in IBS.¹¹ A dysfunctional interaction between the indigenous flora and the intestinal mucosa might lead to immune activation in the colonic mucosa. However, further study is needed before the relationship between the intestinal microbiota and IBS is fully elucidated.

Professor Quigley outlined a possible mechanism linking the luminal flora with the development of IBS symptoms. Changes in intestinal barrier function in response to stress, infection or an altered microbiota permit access of luminal flora to the sub-epithelial compartment resulting in immune and mast cell activation. Adjacent sensory neurones are stimulated by released proinflammatory mediators, giving rise to IBS symptoms, such as muscle spasm, visceral hypersensitivity and hyperalgesia.

The putative role of inflammation in the pathophysiology of IBS could have both diagnostic and therapeutic implications; cytokines could be used as disease biomarkers to aid diagnosis, while anti-inflammatory drugs and probiotics could provide targeted therapy. In addition, the involvement of the enteric flora in IBS suggests a potential role for antibiotics or, in particular, probiotics in treatment.

References:

1. Levy RL, et al. *Gastroenterology* 2001;121:799-804.
2. Dunlop SP, et al. *Am J Gastroenterol* 2003;98:1578-1583.
3. Mearin F, et al. *Gastroenterology* 2005;129:98-104.
4. Barbara G, et al. *Gastroenterology* 2004;126:693-702.
5. Chadwick VS, et al. *Gastroenterology* 2002;122:1778-1783.
6. Liebrechts T, et al. *Gastroenterology* 2007;132:913-920.
7. Dinan TG, et al. *Gastroenterology* 2006;130:304-311.
8. Cenac N, et al. *J Clin Invest* 2007;117:636-647.
9. Quigley EM, et al. *Am J Gastroenterol* 2007;102:S514-S515.
10. Langhorst J, et al. *Gut* 2007;56:1325-1326.
11. Quigley EM. *J Dig Dis* 2007;8:2-7.

Does Functional Dyspepsia Exist?

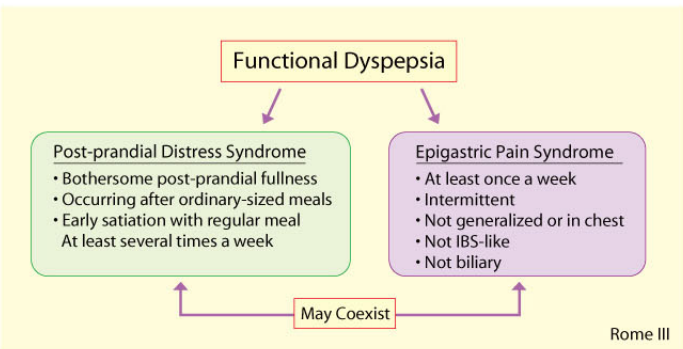
Professor Eamonn MM Quigley

Department of Medicine, Cork University Hospital
National University of Ireland
Cork, Ireland

Patients who presented with ulcer-like or dyspeptic symptoms with no apparent mucosal pathology were originally diagnosed with “non-ulcer dyspepsia”. The Rome Foundation replaced this term with “functional dyspepsia” (FD).¹ In Rome III, FD was redefined to help differentiate it from gastroesophageal reflux disease (GERD) and IBS.² Patients with FD may present with postprandial distress and epigastric pain syndromes (Figure) with pain that may be of a burning quality but without a retrosternal component. However, FD does not have a predictable and characteristic pathophysiology, and shows extensive overlap of symptoms and shared aetiological features with IBS and GERD. The situation is further complicated by differences in transcultural interpretation of “dyspeptic” symptoms and confusion over the definition of these disorder-defining symptoms.

Professor Quigley explained that although a number of motor and sensory abnormalities have been described in FD, their correlation with symptoms is poor. Furthermore, treatment responses are unpredictable and correlate best with overlapping and coincident diseases. The introduction of the term FD has not assisted clinical diagnosis, has not helped to establish its pathophysiology and no new therapies have been identified. Professor Quigley believes that this evidence questions the existence of FD as a distinct disease entity.

Figure. Rome III criteria for functional dyspepsia.²



References:

1. Drossman DA, et al., eds. *ROME II. The Functional Gastrointestinal Disorders. Diagnosis, Pathophysiology and Treatment: A Multinational Consensus*. 2nd ed. McLean, Va: Degnon Associates; 2000.
2. Drossman DA. *Gastroenterology* 2006;130:1377-1390.

Functional Constipation: The Hong Kong Experience

Dr Annie OO Chan

Endoscopy Director, Department of
Medicine, Queen Mary Hospital
Associate Professor, Department of Medicine,
The University of Hong Kong

Functional constipation affects approximately 14% of the Hong Kong population¹ and its aetiology is probably multifactorial. To facilitate better diagnosis and severity assessment in local patients, Dr Chan and her colleagues developed the first Chinese version of the constipation questionnaire.²

While researching various aspects of the pathophysiology of functional constipation in Hong Kong, Dr Chan's research group have found:

- An impaired afferent pathway leading to abnormal urge sensation in patients.³
- Patients with functional constipation have defective coping mechanisms, which may contribute to internalization of stress and manifestation of psychosomatic diseases like functional constipation.⁴
- Familial aggregation of constipation occurs, supporting a genetic or intrafamilial environmental component, such as learning/culture or environmental exposure.⁵
- Familial clustering is associated with younger age at onset, more complications and lower likelihood of precipitating factors at onset.⁶
- Functional constipation does not increase the risk for colorectal cancer.⁷

Treatment of functional constipation is often unsuccessful, and research into more reliable treatments is ongoing. Dr Chan's research group is investigating various treatment options including polyethylene glycol, increased dietary fibre, biofeedback and colonic hydrotherapy.^{8,9}

References:

1. Cheng C, et al. *Aliment Pharmacol Ther* 2003;18:319-326.
2. Chan AO, et al. *Aliment Pharmacol Ther* 2005;22:483-488.
3. Chan A, et al. Presented at: Digestive Disease Week 2007; May 19-24, 2007; Washington, USA. ID# 119.
4. Chan AO, et al. *World J Gastroenterol* 2005;11:5362-5366.
5. Chan AO, et al. *Am J Gastroenterol* 2007;102:149-152.
6. Chan AO, et al. *Clin Gastroenterol Hepatol* 2007;5:197-200.
7. Chan AO, et al. *Gut* 2007;56:451-452.
8. Chan AO, et al. *World J Gastroenterol* 2007;13:4771-4775.
9. Chan A, et al. Presented at: Digestive Disease Week 2007; May 19-24, 2007; Washington, USA. ID# S1202.

Alcohol-induced Liver Disease

Professor Willis C Maddrey

Professor of Internal Medicine and
Executive Vice President for Clinical Affairs
The University of Texas Southwestern Medical Center
Dallas, Texas, USA

Although the amount of alcohol ingested is generally related to the risk of developing liver disease, there is no predictable dose response; the alcohol consumption threshold for hepatic injury varies among individuals depending on genetic background, sex (females are more susceptible), duration of exposure, and health and nutritional status. Fatty liver (hepatic steatosis) almost invariably develops in response excessive alcohol consumption but does not inevitably lead to alcoholic hepatitis or cirrhosis. Patients may progress through acute alcoholic hepatitis to frank cirrhosis, but in some patients cirrhosis is the primary disease manifestation. Only about 10% to 20% of heavy drinkers eventually develop cirrhosis,¹ supporting the concept that factors other than alcohol exposure are involved.

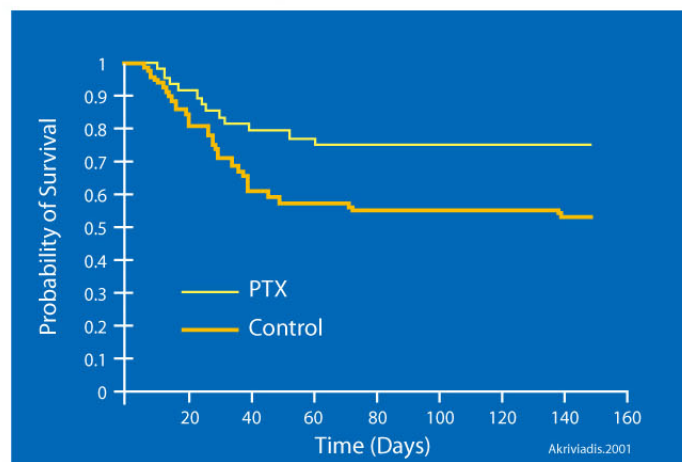
Alcohol-induced liver injury promotes transformation of the stellate cells lining the sinusoids from simple storage cells into migratory collagen-producing cells responsible for fibrogenesis. The exact triggers for activation of stellate cells are not known, but may include direct injury from reactive oxygen species or intermediate steps that include the expression of tumour necrosis factor- α (TNF- α) and subsequent release of proinflammatory interleukins (IL) such as IL-1, IL-6 and IL-8. TNF- α levels in patients with alcoholic hepatitis have been found to correlate with mortality.²

Recent studies suggest that concomitant alcohol abuse and hepatitis C virus (HCV) infection may exert a multiplicative effect on the risk for liver disease. Alcohol has been shown to stimulate the replication of HCV RNA and interfere with the effectiveness of interferon therapy.³ Moreover, HCV-positive alcohol abusers develop more severe fibrosis with higher rates of cirrhosis and hepatocellular carcinoma (HCC) than HCV-positive nondrinkers.⁴

Abstinence is the cornerstone of management of alcohol-induced liver injury and calls for both intermediate and long-term support. Identification and treatment of superimposed infectious complications is also key and nutritional support therapy to correct the nutritional deficits commonly found in alcohol abusers is an important adjunctive approach. Corticosteroid therapy has proven useful in treating patients with severe acute alcoholic hepatitis, with prednisolone significantly improving short-term survival.⁵ Pentoxifylline, a phosphodiesterase inhibitor that reduces the production of TNF- α and other cytokines and chemokines, also improves short-term survival in patients with severe alcoholic hepatitis (Figure), largely through a significant decrease in the risk of developing hepatorenal syndrome.⁶ The future of alcoholic

hepatitis treatment probably lies in anti-TNF α therapies, a number of which are currently under investigation.^{7,8} Liver transplantation has also been an increasingly popular option over the past decade for persistently abstinent patients who have alcohol-induced cirrhosis.⁹

Figure. Pentoxifylline significantly improves short-term survival in severe alcoholic hepatitis⁶



References:

1. American Liver Foundation. Alcohol-Induced Liver Disease. Available at: <http://www.liverfoundation.org/education/info/alcohol>. Accessed October 9, 2007.
2. McClain CJ, et al. *Semin Liver Dis* 1999;19:205-219.
3. Zhang T, et al. *Hepatology* 2003;38:57-65.
4. Singal AK, Anand BS. *J Clin Gastroenterol* 2007;41:761-772.
5. Ramond MJ, et al. *N Engl J Med* 1992;326:507-512.
6. Akriviadis A, et al. *Gastroenterology* 2000;119:1637-1648.
7. Tilg H, et al. *J Hepatol* 2003;38:419-425.
8. Menon KV, et al. *Am J Gastroenterol* 2004;99:255-260.
9. Tome S, Lucey MR. *Aliment Pharmacol Ther* 2004;19:707-714.

Drug-induced Liver Toxicity

Professor Willis C Maddrey

Professor of Internal Medicine and
Executive Vice President for Clinical Affairs
The University of Texas Southwestern Medical Center
Dallas, Texas, USA

Many commonly used drugs cause some degree of liver alteration, often minor and transient alanine aminotransferase (ALT) elevations. However, some drugs can pose greater risk for patients with underlying liver diseases such as steatohepatitis, alcohol-induced liver disease, metastatic liver disease and chronic viral infections. For example, patients with chronic hepatitis C (CHC) or B (CHB) are at increased risk of isoniazid-induced liver injury,¹ and HIV infection increases the risk of sulfonamide hepatotoxicity.² Furthermore, symptoms of underlying liver injury can divert attention from the possibility of drug-induced hepatotoxicity, which itself can mask or mimic other liver-related conditions.

There are a wide variety of risk factors for drug-induced liver disease, the most important of which include age, female gender, alcohol use and obesity. Multiple mechanisms have been implicated in drug-induced hepatotoxicity. Professor Maddrey highlighted two: the production of reactive toxic drug intermediates, largely via the cytochrome P450 (CYP) enzyme superfamily; and the failure of the hepatic systems of some individuals to adapt to mild, transient hepatic injury through changes in the innate immune system or the use of alternative metabolic pathways. Failure to adapt, which may be influenced by genetics as well as age and gender, leads to clinical injury.

Elevated ALT levels signal potential liver injury; ALT levels of more than three times the upper limit of normal (ULN) without overt clinical symptoms are suggestive of intermediate-level injury and should be carefully investigated. It is important to note that an ALT of >3 x ULN combined with clinically apparent jaundice has an ominous prognosis - the associated mortality rate is 10% or more.³ Drugs tend to have a hepatotoxicity "signature" related to the latency and pattern of injury, which can aid diagnosis or at least rule out drug involvement.

Genetic studies of CYP polymorphisms show potential in this field; for example, patients with homozygous wild genotype CYP2E1 c1/c1 have a higher risk of isoniazid hepatotoxicity than those with the mutant allele c2.⁴ Also, individuals with defective epoxide hydrolase cannot adequately detoxify the reactive metabolite of phenytoin,⁵ which provides a potential test for susceptibility to phenytoin toxicity. These data signal the future-toxicogenomics. Toxicogenomic studies may lead to screening tests to identify patients at risk for drug-related liver injury. However, until such tests are a reality, clinicians need to be aware of the potential hepatotoxicity of drugs they prescribe, particularly where underlying liver disease or concomitant drug or alcohol consumption may exacerbate the risk or confound the clinical signs.

References:

1. Saukkonen JJ, et al.; ATS Hepatotoxicity of Antituberculosis Therapy Subcommittee. *Am J Respir Crit Care Med* 2006;174:935-952.
2. Levy M. *Drug Saf* 1997;16:1-8.
3. Zimmerman HJ. *Clin Liver Dis* 2000;4:73-96,vi.
4. Huang YS, et al. *Hepatology* 2003;37:924-930.
5. Spielberg SP, et al. *N Engl J Med* 1981;305:722-777.

Update on Treatment of Chronic Hepatitis B

Professor Ching-Lung Lai

Chair Professor of Hepatology and Medicine, and
Chief of Gastroenterology and Hepatology Division
Department of Medicine, The University of Hong Kong
Queen Mary Hospital, Hong Kong

Guidelines and recommendations for management of CHB developed by different groups still vary in the recommended disease markers and thresholds for guiding treatment decisions. Professor Lai discussed the discrepancies between two recently published treatment guidelines,^{1,2} placing particular emphasis on the need to consider both hepatitis B virus (HBV) DNA and ALT levels.

HBV DNA level is the primary treatment determinant for hepatitis B e-antigen (HBeAg)-positive patients in a treatment algorithm developed by a panel of American hepatologists (Keeffe et al).¹ In contrast, the American Association for the Study of Liver Diseases (AASLD) 2007 guidelines base treatment initiation largely on ALT levels, with ALT >2 x ULN being a threshold for definite treatment.² For HBeAg-negative patients, the AASLD 2007 guidelines are again primarily based on ALT level and the threshold of >2 x ULN also features here, but HBV DNA levels are also considered for these patients. Professor Lai explained that the threshold of 2 x ULN appears somewhat arbitrary as there are no published data to support the view that patients with ALT >2 x ULN are especially at risk for disease progression. Even patients with near normal ALT levels can be at considerably increased risk for cirrhosis and HCC.³⁻⁵ Professor Lai believes that ALT levels should not be used alone in making treatment initiation decisions; HBV DNA levels should always be considered as well.

Recommended treatment endpoints also vary between the two publications. For HBeAg-positive patients, the treatment endpoint in the Keeffe et al. algorithm is HBeAg seroconversion followed by 6 to 12 months of undetectable (by PCR) HBV DNA levels, while the AASLD 2007 guidelines recommend treating until 6 months after seroconversion, but set no criteria for HBV DNA levels. In HBeAg-negative patients, the two publications largely concur, with the Keeffe et al. algorithm recommending "long-term treatment", and the AASLD 2007 guidelines promoting continuation of treatment to hepatitis B surface antigen (HBsAg) clearance, which Professor Lai explained equates to long-term treatment.

Professor Lai emphasised that with the current understanding of the disease course of CHB, it is unacceptable to use HBeAg seroconversion as the sole criteria of treatment endpoint, without consideration of HBV DNA level. In patients who acquire HBV infection at birth or in early childhood (as is typical in Asian countries), disease progression continues after HBeAg seroconversion despite HBV DNA levels <2,000 IU/mL and ALT levels between 0.5 and 2 x ULN.⁶ Furthermore, the Risk Evaluation of Viral Load Elevation Associated Liver Disease/cancer (REVEAL) studies have shown that the risk for HCC and cirrhosis rises with increasing HBV DNA levels, independent of HBeAg status and ALT levels.^{3,7}

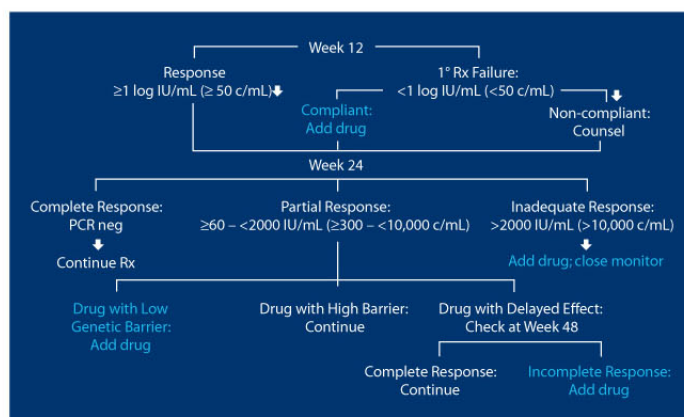
In Professor Lai's opinion, the ideal approach to treatment would be⁶:

- Treat patients as early as possible when the immune clearance phase is prolonged
- Treat HBeAg-negative patients long term, since the majority of complications occur after HBeAg seroconversion
- Do not use HBeAg seroconversion in HBeAg-positive patients as the sole treatment endpoint
- Continue treatment until HBV DNA levels are permanently undetectable by PCR assays and ALT levels are maintained at <0.5 x ULN

Studies have shown that prediction of resistance risk and response to long-term therapy is possible early in nucleos(t)ide analogue therapy. Only 8% of HBeAg-positive patients treated with long-term lamivudine (median follow up, 29 months) who had undetectable HBV DNA levels (<200 copies/mL) at week 24 developed lamivudine resistance compared with 64% of those who had a HBV DNA level of >2,000 IU/mL at week 24.⁸

Professor Lai also highlighted an algorithm developed recently in an international workshop that promotes early monitoring of serum HBV DNA levels during nucleos(t)ide therapy to identify treatment outcomes. The algorithm, named the "roadmap concept", provides strategies for managing patients based on virologic responses at week 12 and 24 (Figure), but does not extend to guiding long-term therapy.⁹ Even earlier prediction of future response may be possible. A very recent study has shown that a good 5-year response to lamivudine therapy is more likely in HBeAg-positive patients if HBV DNA levels at week 4 are <2,000 IU/mL.¹⁰

Figure. The "roadmap" concept for guiding therapy decisions in CHB⁹



References:

1. Keeffe EB, et al. *Clin Gastroenterol Hepatol* 2006;4:936-962.
2. Lok ASF, McMahon BJ. *Hepatology* 2007;45:507-539.
3. Chen CJ, et al. *JAMA* 2006;295:65-73.
4. Uchenna HI, et al. *Gastroenterology* 2006;130:678-686.
5. Yuen MF, et al. *Gut* 2005;54:1610-1614.
6. Lai CL, Yuen MF. *Ann Intern Med* 2007;147:58-61.
7. Iloeje UH, et al. *Gastroenterology* 2006;130:678-686.
8. Yuen MF, et al. *Hepatology* 2001;34:785-791.
9. Keeffe EB, et al. *Clin Gastroenterol Hepatol* 2007;5:890-897.
10. Yuen MF, et al. *Hepatology* 2007 (in press).

Highlights from Annual General Meeting and Scientific Meeting 2008

March 13, 2008 (6:15 p.m. - 10:00 p.m.)

Ballroom, Level 7, Langham Place Hotel, Mongkok, Kowloon

Organizing Chairmen: Dr. YUEN Man Fung & Dr. Vincent K.S. LEUNG

This was a very successful scientific meeting attended by 118 doctors and medical professionals. Professor Francis Chan, the President of the Society, welcomed the participants to the Scientific Meeting. After Professor Joseph Sung gave a citation on the outstanding work and contributions of Professor Anthony Axon, the distinguished guest from the United Kingdom, Professor Chan presented him a plaque of the Society.

Professor Axon then delivered his presentation on "The clinical relevance of dysplasia in Barrett's oesophagus" which was deemed highly interesting and informative. The discussion followed was actively participated.

The Annual General Meeting was attended by 46 fellows and members. The Society's annual report and financial statement for the year of 2007 were adopted as presented by the Chairman and Hon. Treasurer. Eight members were re-elected to the Council for the term 2008-2010.

Nine pharmaceutical companies participated in the exhibition and they were AstraZeneca, Bristol-Myers Squibb, Eisai, Ferring, GlaxoSmithKline, Novartis, Nycomed, Roche and Takeda. A certificate of appreciation was presented to each of the sponsors in appreciation of their support and contributions.

The majority of the participants stayed and enjoyed the dinner during which informal discussions and exchange of views continued.



Upcoming Meeting

Tenth Joint Annual Scientific Meeting

September 6, 2008 (1:00 p.m. - 6:20 p.m.)

Ballroom, Level 7, Langham Place, Mongkok Hong Kong

Co-organizers: The Hong Kong Society of Gastroenterology
The Hong Kong Society of Digestive Endoscopy
Hong Kong Society for Coloproctology
Hong Kong Association for the Study of Liver Diseases
The Hong Kong Society of Gastrointestinal Motility

Co-sponsors: AstraZeneca, GlaxoSmithKline

Organizing Chairman: Prof. Man-Fung Yuen

Sessions	Speakers	Topic
Symposium I (GI)	Prof. Angel Lanas (Spain)	"Management of aspirin and NSAID related GI complications: a European perspective"
	Prof. James Lau (PWH)	"Screening of CRC in first-degree relatives"
Symposium II (Liver)	Prof. Pietro Lampertico (Italy)	"Beyond the use of PPI in the management of acute peptic ulcer bleeding"
	Dr. James Fung (QMH)	"Management and prevention of NUC resistance"
Symposium III (Endoscopy Video Forum)	Dr. Philip Chiu (PWH)	"Antiviral treatment of HBeAg negative chronic hepatitis B"
	Prof. W.L. Law (QMH)	"Non-invasive assessment of liver fibrosis"
		"Endoscopic Submucosal Dissection in GI tumours"
		"Colonic Stenting"

Further details will be available soon from website www.hksge.org/event

Welcome new members & fellows

Honorary Fellow

Prof. Anthony Axon

President, World Organisation of Digestive Endoscopy
Professor of Gastroenterology, The General Infirmary at Leeds, U.K.

Member

Dr. Sammy Pak Lam CHEN

Department of Medicine & Geriatrics
Kwong Wah Hospital

Dr. Lindy Tsz Lam NG

Department of Medicine
Pamela Youde Nethersole Eastern Hospital

Dr. TAI Ling Fung

Department of Medicine
Pamela Youde Nethersole Eastern Hospital

Major Meetings

12-15 June 2008

Hong Kong – Shanghai International Liver Congress 2008

Organizers: Shanghai Organizing Committee;
Cheng Si Yuan (China-International) Hepatitis Research Foundation
Location: Hong Kong Convention & Exhibition Centre, Hong Kong
Website: www.livercongress.org

25-28 June 2008

10th World Congress on Gastrointestinal Cancer

Organizers: Imedex in partnership with the European Society of Medical Oncology
Location: Barcelona, Spain
Website: www.worldgicancer.com

11-12 July 2008

Hong Kong Surgical Forum, Summer 2008

Organizers: Department of Surgery, Li Ka Shing Faculty of Medicine, The University of Hong Kong Medical Centre; Queen Mary Hospital & Hong Kong Chapter of the American College of Surgeons
Location: Queen Mary Hospital, Hong Kong
Website: www.hku.hk/surgery

11-13 July 2008

GIHep Singapore 2008

Organizers: Gastroenterological Society of Singapore
Society of Colorectal Surgeons (Singapore)
Chapter of Gastroenterologists College of Physicians, Singapore
Location: Singapore
Website: www.gihp.org.sg

16-20 August 2008

3rd Edition of the World Congress of Pediatric Gastroenterology Hepatology and Nutrition

Organizer: Latin American Society of Pediatric Gastroenterology, Hepatology and Nutrition (LASPGHAN)
Location: Iguassu Falls, Brazil
Website: www.wcpghan2008.com

27-29 August 2008

10th International Congress of ESHGID

Organizer: The Egyptian Society of Hepatology, Gastroenterology and Infectious Diseases in Alexandria in collaboration with Egyptian Hepato-Pancreato-Biliary Society
Location: Alexandria, Egypt
Website: www.alfamedical.com

2-5 September 2008

11th World Congress of Endoscopic Surgery (WCES)

Organizers: International Federation Societies Endoscopic Surgeons
Location: Yokohama, Japan
Website: www.wces2008.com

5-6 September 2008

ELSA 2008

Organizer: Endoscopic & Laparoscopic Surgeons of Asia
Location: Yokohama, Japan
Website: www.elsa2008.com

6 September 2008

10th Joint Annual Scientific Meeting

Co-organizers:
The Hong Kong Society of Gastroenterology
Hong Kong Society of Digestive Endoscopy
Hong Kong Society for Coloproctology
Hong Kong Association for the Study of Liver Diseases
The Hong Kong Society of Gastrointestinal Motility
Location: Langham Place Hotel, Mongkok, Kowloon, Hong Kong
Website: www.hksge.org/event

10-13 September 2008

11th World Congress of the International Society for Diseases of the Esophagus

Organizer: The International Society for Diseases of the Esophagus
Location: Budapest, Hungary
Website: www.isdecongress2008.com

13-16 September 2008

The Asian Pacific Digestive Week 2008 (APDW 2008)

Organizer: Indian Society of Gastroenterology
Location: New Delhi, India
Website: www.apdw2008.net

17-20 September 2008

Prague Hepatology Meeting 2008

Organizer: Foundation of the Czech Society of Hepatology
Location: Prague, Czech Republic
Website: www.congressprague.cz/en/kongresy/phm2008.html

18-20 September 2008

XXIst International Workshop on Helicobacter and related bacteria in chronic digestive inflammation

Local Organizer: Marcis Leja
Location: Riga, Latvia
Website: www.helicobacter.org

18-19 September 2008

Falk Symposium 166: III FALK GASTRO-CONFERENCE (Part II)

GI Endoscopy - Standards & Innovations
Organizer: Falk Foundation
Location: Mainz, Germany
Website: www.dr.falkpharma.de

20-21 September 2008

Falk Symposium 167: III FALK GASTRO-CONFERENCE (Part III)

Liver Under Constant Attack - From Fat to Viruses
Organizer: Falk Foundation
Location: Mainz, Germany
Website: www.dr.falkpharma.de

25-27 September 2008

Gastrointestinal Oncology Conference 2008

Organizer: International Society of Gastrointestinal Oncology
Location: Arlington, Virginia, USA
Website: www.isgio.org/isgio2008

1-4 October 2008

Japanese Digestive Disease Week (JDDW)

Organizers: Japanese Society of Gastroenterology
Japan Gastroenterological Endoscopy Society
Japan Society of Hepatology
Japanese Society of Gastroenterological Cancer Screening
Japanese Society of Digestion and Absorption
Location: Tokyo, Japan
Website: www.jddw.jp

3-8 October 2008

The ACG 2008 Annual Scientific Meeting & Postgraduate Course

Organizer: American College of Gastroenterology
Location: Orlando, FL, U.S.A.
Website: www.acg.gi.org

8-11 October 2008

18th World Congress of the International Association of Surgeons, Gastroenterologists and Oncologists

Organizer: International Association of Surgeons, Gastroenterologists and Oncologists
Location: Istanbul, Turkey
Website: www.iasg2008.org



18-22 October 2008

16th United European Gastroenterology Week (UEGW)

Organizer: The United European Gastroenterology Federation
Location: Vienna, Austria
Website: www.uegf.org

22-26 October 2008

Australian Gastroenterology Week (AGW)

Organizer: Gastroenterological Society of Australia
Location: Brisbane, Australia
Website: www.gesa.org.au

31 October – 4 November 2008

The Liver Meeting and International Association for the Study of Liver 25th Biennial Meeting

Organizer: American Association for the Study of Liver Diseases
Location: San Francisco, CA, U.S.A.
Website: www.aasld.org

7-10 November 2008

21st World Congress of Digestive Surgery (WCDS2008)

Organizer: International Society for Digestive Surgery
Location: Beijing, China
Website: www.wcds2008.org

13-16 February 2009

19th Conference of the APASL

Organizer: Asian Pacific Association for the Study of the Liver
Location: Hong Kong
Website: www.apasl2009hongkong.org

22-26 April 2009

44th Annual Meeting of the European Association for the Study of the Liver (EASL)

Organizer: The European Association for the Study of the Liver
Location: Copenhagen, Denmark
Website: www.easl.ch

30 May - 4 June 2009

Digestive Disease Week 2009 (DDW)

Organizer: DDW Organizers
Location: Chicago, Illinois, USA
Website: www.ddw.org

21-25 November, 2009

Gastro 2009 (UEGW + WCOG)

Organizers: The United European Gastroenterology Federation (UEGF)
World Gastroenterology Organization (WGO)
World Organisation of Digestive Endoscopy (OMED)
British Society of Gastroenterology (BSG)
Location: London, United Kingdom
Website: www.gastro2009.org

