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Message from the WGO Foundation Chair

WGO training and education programs improve digestive health internationally



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We are all well aware of the prominence of digestive disorders—from diarrhea to obesity to hepatitis to cancer—among global health-care issues, as well as the significant burden they place on national budgets. In its role as the global representative for gastroenterology and hepatology, the World Gastroenterology Organization (WGO) is continually seeking to both promote, to the general public and health care professionals, an awareness of the worldwide prevalence and optimal care of digestive disorders by providing high-quality, accessible, and independent education and training. One of the many ways in which we achieve this is through World Digestive Health Day (WDHD).

Established in 2004, WDHD is celebrated annually on May 29 (the anniversary of the founding of the WGO in Washington, DC, in 1958) by highlighting an important topic in the area of digestive health and

disease. What was initially a single-day public health campaign has grown dramatically since its inception and has evolved into outreach and educational activities throughout the year. Over forty national member societies participated in WDHD 2009, and we look forward to even more being involved this year.

The theme for WDHD 2010 is inflammatory bowel disease (IBD), and our focus is on optimizing diagnostic approaches and maximizing patient care to enhance the quality of life of IBD patients and to help inform their health-care providers worldwide. WGO and the WGO Foundation would like to thank our corporate partners, Takeda Pharmaceuticals and Shire, for their support of WDHD 2010. It is through their generosity that we will be able to raise awareness of IBD around the world.

There is much more to the WGO

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training and education programs than WDHD. Our other initiatives include:

- *Training centers*, which seek to raise the level of practice and help retain skilled doctors and other related health professionals in low-resource nations through hands-on training
- The *International Digestive Cancer Alliance* (IDCA), to promote awareness, screening, early detection, primary prevention, and treatment of digestive cancers through educational activities
- The *Train-the-Trainers* workshops, which offer professional development courses to help enhance the educational and training skills of clinician-educators
- *Global guidelines*, which provide locally relevant treatment options through practical tools that utilize cascades that can be adapted to available resources and infrastructure
- The *Outreach Program*, which helps equip the WGO Training Centers and medical institutions around the world with endoscopic instruments to improve both training and care

Established in 2007, the WGO Foundation is the philanthropic arm of the WGO and serves as the primary mechanism to secure support for and ensure the sustainability of each of these critical initiatives. We are vigorously pursuing a strategy to deliver these programs on a larger scale and to those who need them most by raising much-needed funds, strengthening existing relationships, and forging new ones. This dynamic plan includes expanding the reach of each program and linking them together electronically so that we can leverage the resources, knowledge, talent, and skills of each to complement the others. In the coming months, we will be providing updates on the progress of this undertaking.

This is a historic time for WGO and the WGO Foundation, as we are uniquely positioned not only to begin a new phase in our own development, but also to have an unprecedented effect on the prevention and treatment of digestive disorders. By increasing the number of trained gastroenterologists and related health personnel, we will raise the standard of care in the countries that need it most. We are seeking to work more closely with our member national societies and partners from industry on this expansion, and together we will make a difference by significantly improving the quality of life for many around the world. ■

World Digestive Health Day 2010: Special Scientific Highlight

Interview with the WDHD 2010 campaign leader, Dr. Charles Bernstein

Has the epidemiology of inflammatory bowel diseases (IBD) changed, especially during the past 10–20 years—and if so, are there differences in these changes between ulcerative colitis and Crohn’s disease?

CB: IBD emerged in the early to mid-20th century in the developed world. When it emerged, there was a predominance of ulcerative colitis (UC) over Crohn’s disease, and Crohn’s disease was 1.3 times more common in women. In the last decade in the developed world, Crohn’s disease has become the predominating disease, with incidence rates higher than those for UC in most countries. There has also been more of a balance between women and men. In fact, several studies on children have shown that there now is a greater incidence among boys than girls. Meanwhile, over the past 10–15 years, IBD has emerged to a greater extent in the developing world and even in developed countries where it was previously uncommon, such as Japan. However, UC is predominant in these emerging nations, much as it was in the West several decades ago.

It is not known why UC presents first and is then overtaken by Crohn’s disease in areas in which IBD emerges. The province of Manitoba in Canada is interesting. Like other Canadian provinces, Manitoba has among the highest incidence rates of Crohn’s disease in the world, at approximately 15 per 100,000. However, 10% of Manitoba’s population belong to the First Nations, or North American

Indians. This community has very low rates of IBD, but UC predominates over Crohn’s disease by a factor of four within their IBD population. Members of this ethnic community often live in crowded housing and in areas with a high prevalence of infections that are transmitted by the fecal–oral route, such as hepatitis A and *Helicobacter pylori*. These communities living in Canada share the same epidemiological profile for IBD that developing nations have.

What can be hypothesized about the etiology of the diseases from what is known about the epidemiology of IBD?

CB: When exploring the etiology of IBD, it can be as revealing to study communities that do not get the disease as it is to study those that do (such as the indigenous population in Manitoba juxtaposed to the Caucasian population). However, as the disease emerges in the developing world, it will be of great interest to explore environmental changes in those countries, as this may make it possible to define what may be spurring this emergence of IBD. One hypothesis that has evolved in relation to many chronic immune diseases is the “hygiene hypothesis.” This hypothesis posits that in communities where there is a reduction of communicable infectious diseases (some of which may even be fatal), there is a parallel rise in chronic immune diseases. If the developing immune system is not exposed to

microorganisms in childhood, it may not become tolerant of microorganisms with similar antigenicity later in life. Another angle to the hygiene hypothesis is the possibility that it is not a lack of pathogenic organisms in youth, but rather a reduction in saprophytic, potentially probiotic-type organisms that are able to down-regulate injurious immune responses by triggering regulatory T cells. What could alter this microbial ecology? Is it the increasing use of antibiotics? Is it a change in diet? Is it a change away from an agricultural milieu to a more industrial one? These are the types of issue that require investigation.

Are there gender differences in either form of IBD, and do they provide any clues to the etiology?

CB: As I mentioned, there used to be a predominance of females over males in Crohn’s disease, but this gender inequity has disappeared over time and more recently there have been similar rates among females and males. In UC, there is no gender predilection. The initial female predominance led to questions regarding the impact of female sex hormones on Crohn’s disease. Some data have suggested an increase in the incidence among users of oral contraceptives, but no clear etiologic connection has ever been established.

What is the peak age of onset of IBD, and what implications does that have

for investigating the etiology of the diseases?

CB: In Crohn's disease, the peak age of onset is the third decade, although the disease can present at any age. There do not seem to be major phenotypic differences between patients in whom it develops in childhood and those in whom it appears over the age of 40 in that fistulas and small bowel involvement can be seen at all ages. However, children are more likely to present with ileal disease and person presenting later in adulthood are more likely to present with isolated colonic disease.

It seems logical to pursue etiological studies intensely in children—especially as they have not yet been exposed to as wide a variety of life experiences and influencing factors, so that it may be easier to catalogue dietary and environmental influences. In UC, the incidence starts to rise in childhood and reaches a peak in the third decade, when it plateaus and remains fairly constant across all adult age groups. These differences in the age of presentation may therefore have implications for the etiology of Crohn's disease in comparison with UC. Perhaps microorganisms that are relevant or acquired in childhood may be more relevant to the etiology of Crohn's disease than UC.

Are there differences in the presentation of Crohn's disease in different areas of the world?

CB: This is the type of issue that really requires further exploration. In the recent Practice Guidelines prepared by a number of gastroenterologists from around the world under the auspices of the WGO, we created a cascades approach to diagnosing and managing IBD in different regions of the world (*Inflamm Bowel Dis* 2010;16:112–24). Crohn's disease is distinguished from UC by disease proximal to the colon, perineal disease, fistulas, histologic granulomas, and full-thickness as opposed to mucosa-limited disease. In Crohn's disease, granulomas are evident in up to 50% of patients and fistulas in 25%. It is noteworthy that the presentation of Crohn's disease and UC is quite similar in such disparate areas of the world as North America, South America, Europe, Australia, and New Zealand.

But there are also differences. In Pakistan, for example, there is much less extraintestinal disease with both UC and Crohn's disease than is reported in the West (where up to 25% of patients have extraintestinal manifestations, if arthralgias are included). In Pakistan, few patients have perianal or fistulizing disease. In India, for example, the age of presentation of CD is a decade later than in the West, colonic involvement is more common, and fistulization appears less common. More information is needed in order to discern whether there are differences in the IBD phenotype in some of the nations where it is now newly emerging.

Are there any unifying hypotheses about what might cause either form of IBD?

CB: Currently, the leading candidate etiologic agent is some type of microorganism that triggers an aberrant immune response. It is unclear whether this microorganism might be an exogenous infection or is an organism that emerges from an imbalance in the host's gut flora. No obvious candidate exogenous microorganisms have emerged, although interest still abounds as to what role an atypical mycobacterium might play. *Mycobacterium paratuberculosis* causes Johne's disease in cattle (a Crohn's-like disease), but to date it has not been proven that this organism is zoonotic—that is, that it definitely causes human disease. A leading candidate microorganism in the gut flora is an adherent invasive form of *Escherichia coli*; this was first identified by a French group, but has since been isolated by several independent laboratories in Europe and North America. If a microbe from within the gut flora emerges as a key contributor to the pathogenesis of IBD, it will still be unclear what it is that alters the flora ecology. Is it diet? Is it antibiotics? Is it other infections?

If the incidence rates of IBD are lower in the developing world than in the developed world, what happens to the incidence rates amongst immigrants to the developed world? Is there a

difference between UC and CD?

CB: It seems that immigrants retain the incidence rates of their birth countries for diseases like IBD, so that immigrants from the developing world to the developed world have low rates of IBD. However, there are suggestions from Britain and Vancouver that the offspring of immigrants who are raised in the West have incidence rates that are comparable to the Caucasian populations of those countries. This has been published for UC, and it may be similar in Crohn's disease. This type of finding supports the notion that the environment has a stronger impact on the development of the diseases than genetics.

Should the approach to treating IBD be similar around the world?

CB: The approach to treating IBD around the world will have to be tailored to the availability of treatments in the various countries. It would be optimal if the best approach was available universally, but it is less likely that the expensive therapies used in the West will become as widely available in developing nations. Furthermore, there may be issues of access to health care in developing nations that are not as problematic in the West. This is reviewed in the WGO Practice Guidelines.

How might the search for the etiology of IBD best be carried out?

CB: The search can be pursued in areas where IBD is well established. This is mostly in the developed nations, where resources are available to investigate complex genetic, immunological, and microbiological studies. However, etiologic hypotheses should also be pursued amongst children and in particular in countries where IBD is emerging. These communities provide an opportunity to study IBD early in its evolution. This has also been reviewed in a recent paper ([Gut 2008;57:1185-91](#)). ■



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Colorectal cancer in inflammatory bowel disease



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Inflammatory bowel disease (IBD)—i.e., ulcerative colitis (UC) and Crohn's disease (CD)—ranks among the high-risk conditions for colorectal cancer (CRC), together with the hereditary syndromes of familial adenomatous polyposis and hereditary nonpolyposis colorectal cancer. The increased risk of CRC in patients with IBD has been recognized since the second half of the 20th century, and CRC is a major cause of long-term mortality in these patients.

Epidemiology of colorectal cancer in patients with IBD

Incidence. There has been some fluctuation in estimates of the cumulative incidence of CRC in patients with UC. One study reported a very high figure of nearly 60% after 40 years, but lower figures have usually been reported. A meta-analysis of 41 studies estimated the cumulative

incidence of CRC at 2% after 10 years and 18% after 30 years. A study conducted at St Mark's Hospital, London, estimated the cumulative incidence at only 7.6% after 30 years. Overall, when the relative risk of CRC in patients with UC is compared with that in the general population, the odds ratios vary between 2.5 and 5.5. The risk of CRC has also been confirmed in CD, with neoplastic lesions being detected in 6.2% of 259 patients with colonic CD. The relative risk in comparison with the general population was estimated at 2.5 in a meta-analysis.

Temporal trends in incidence and mortality. Registries of IBD have been developed in Scandinavian countries. A cohort study with long-term follow-up to 2004 has been conducted by Söderlund et al. at the Karolinska Institute in Stockholm in association with several Swedish hospitals,

including 7607 patients in whom IBD was detected in the period 1954–89 (Table 1).¹ The records for 4125 patients with UC and 3482 with CD represented 198,227 patient-years. In this series, the crude incidence of CRC was 95 per 100,000, and the crude mortality 47 per 100,000. During the period 1970–2005, the incidence of CRC in IBD did not decrease significantly, in contrast to a significant decrease in mortality, which in the more recent period (2000–2004) reached the same level as for the general population. This trend may be explained by CRC being diagnosed at an earlier stage, with improved results of treatment and better survival.

Risk factors for CRC in IBD. The increased risk for CRC in IBD is the result of a combination of chronic inflammation and genetic predisposition. In a patient with UC, the following risk factors have been listed: severity of inflammation in the epithelium; extensive involvement of inflammation with pancolitis; young age at diagnosis; family history of CRC (the risk is twice as high when there

Beginning of surveillance	Patient-years	Incidence of CRC	Mortality from CRC
1970–1979	36,456	3.0	3.2
1980–1989	62,910	2.4	1.7
1990–1999	62,463	2.1	1.7
2000–2004	28,406	1.8	0.7

Table 1 Odds ratios for the relative risk of colorectal cancer between 1970 and 2004 in Swedish patients with inflammatory bowel disease, in comparison with the general population. In 2000–2004, the adjusted incidence was higher than that of the general population, but the mortality from colorectal cancer (CRC) had returned to normal

are relatives who have had CRC); and primary sclerosing cholangitis. The risk of CRC increases with the duration of UC, and the risk becomes sizable after 8 years of disease. In CD, the increased risk of CRC does not correlate with the duration of disease.

Other cancers in IBD. In patients with UC, dysplasia may occur in the ileal pouch after colectomy. Patients with UC are also at increased risk for hepatobiliary cancer. In patients with CD and intestinal obstruction, adenocarcinoma has been observed in the terminal ileum. A small increase in the risk for intestinal or extraintestinal lymphoma has been reported in IBD patients who were receiving immunomodulation therapy with azathioprine.

Causal factors for cancer

The role of chronic inflammation. Several lines of research have implicated chronic inflammation of the intestinal mucosa as being a key factor in the risk for CRC:

- The risk increases with the duration and the severity of epithelial inflammation.
- Anti-inflammatory drugs such as 5-amino-salicylic acid (5-ASA) reduce inflammation and the risk of CRC.
- Several animal models in rodents have associated experimental colitis with the development of colonic tumors.
- The colonic mucosa in IBD demonstrates enhanced epithelial turnover, with high rates of mitosis and apoptosis.

Inflammation contributes to tumor

promotion as a result of oxidative stress, with increased expression of COX-2 and nitric oxide synthase (NOS), which activate the antiapoptotic pathway. Chronic inflammation interferes with several molecular pathways of CRC: the chromosomal instability (CIN status) pathway, with inactivating mutation of the APC tumor suppressor gene and of TP53; the microsatellite instability (MSI status) pathway, with alterations in mismatch repair (MMR) genes; and the pathway of epigenetic hypermethylation of CpG islands (CIMP status), with inactivation of regulatory genes.

The role of immunotherapy. Immunomodulating drugs (azathioprine) have been prescribed for IBD patients, but their carcinogenic potential, which has been shown in other indications, is not well established in this situation. Armstrong et al. conducted a nested case-control study on more than 15,000 IBD patients in whom 392 cancers (2.6%) occurred.² The consumption of azathioprine in IBD patients was analyzed against the occurrence of any cancer. No increase in the risk of cancer was shown in individuals with IBD who had taken azathioprine. However, evidence was found for an increased risk of lymphoma, with an odds ratio of 3.22.

Diagnosis of neoplasia

Clinical monitoring of inflamed colonic epithelium in patients with IBD needs to take into account the marked polymorphism in neoplastic precursors of colorectal cancer. The precursors include conventional polypoid adenomas, progressing through the adenoma-carcinoma sequence; dysplasia-associated lesions or masses (DALMs), which are broad-based and

protruding; and flat areas of dysplasia, often multifocal and poorly visible.

Endoscopic diagnosis. Colonoscopy is less sensitive in detecting precancerous lesions in IBD patients than in the general population, because all the surface of the colonic and rectal mucosa has to be scrutinized. Taking into account the multifocal and “invisible” morphology (easily mistaken for inflammation) of dysplasia in flat areas, systematic random biopsies have been recommended, with four-quadrant samples every 10 cm. Novel imaging techniques have improved the effectiveness of detection so much that they are now in balance with the nontargeted biopsy strategy. A major advance has been made with high-resolution endoscopy, magnification, and chromoendoscopy with either indigo carmine dye (nonabsorbed) or toluidine blue dye (absorbed by epithelial cells). A further contribution to diagnosis has been provided by image processing using narrow-band imaging (NBI) techniques and confocal laser endomicroscopy. Trimodal endoscopy imaging, combining white-light endoscopy, autofluorescence imaging (AFI), and NBI has been recommended by the Amsterdam group. Areas of dysplasia appear red on AFI.

The macroscopic classification of dysplasia in IBD includes protruding and flat lesions. Among protruding lesions, a distinction should be made between sporadic adenomatous polyps and DALMs. Sporadic adenomatous polyps are well-defined and develop in sectors free of colitis—for example, the proximal colon in left-sided colitis. DALMs are polypoid structures located in zones of active inflammation and

Findings	Treatment decision
No dysplasia	Repeat colonoscopy (after 1 or 2 y)
Flat neoplastic lesion	
Indefinite for dysplasia	Repeat colonoscopy (after 6 months)
Low-grade dysplasia	Repeat colonoscopy (after 6 months) or colectomy
High-grade dysplasia	Colectomy
Protruding neoplastic lesion	
Sporadic adenoma	Polypectomy
	Repeat colonoscopy (after 1 y)
Adenoma-like DALM	Polypectomy

Table 2 Findings at colonoscopy in patients with ulcerative colitis, and treatment strategies

are associated with a high risk of cancer. In a meta-analysis conducted by Bernstein, 43% of patients undergoing immediate colectomy after detection of a DALM had cancer.³ Some DALMs have an “adenoma-like” morphology, while others are “non-adenoma-like,” with a poorly outlined morphology: irregular nodularity of the surface, ulcerations, and discolored mucosa with velvety patches. These can easily be mistaken for gross inflammatory alterations. Flat areas of dysplasia are more frequent than protruding lesions and can now be detected with high-definition endoscopy. Dysplasia of any grade is associated with a risk of concurrent cancer in another area of the colon.

Strategy of endoscopic treatment. Lesions with the appearance of a sporadic polypoid adenoma can be treated safely using polypectomy (Table 2). This also applies to DALMs with an “adenoma-like” appearance. Conversely, DALMs with a non-adenoma-like appearance require colectomy. In flat areas of high-grade dysplasia, colectomy is also

recommended. Whenever polypectomy is performed, a follow-up colonoscopy is recommended after 6 months to 1 year. When a flat area with low-grade dysplasia is detected, colectomy is strongly recommended.

Prevention of colorectal cancer

CRC can be prevented in IBD patients through prophylactic colectomy or colonoscopic surveillance at regular intervals to detect precursor lesions. Endoscopic surveillance of the colon in IBD patients, at intervals of 1–2 years, has a preventive effect against death from CRC, and the 5-year survival of patients with cancer is now much higher because detection is earlier. In addition, colectomy is avoided in the majority (75%) of patients receiving surveillance. It has been suggested that chemoprevention reduces the risk of CRC, but the effectiveness of this is not firmly established. The anti-inflammatory drug 5-ASA, also known as mesalazine or mesalamine, is used to treat mucosal inflammation in ulcerative colitis and mild to moderate Crohn’s disease. This bowel-specific

aminosalicylate drug acts locally in the gut and is an antioxidant that traps free radicals, which are potentially damaging products of metabolism; the drug has few systemic side effects. A meta-analysis of nine studies conducted in 2005 by Velayos et al. confirmed that the risk of CRC or dysplasia was reduced in users of 5-ASA.⁴ The effective dose was 1.2 g/day. The odds ratio for the risk of neoplasia in users as compared to non-users was 0.37. The mechanism of reduction of the inflammation in the epithelium by 5-ASA is similar to that of nonsteroidal anti-inflammatory drugs. A synthetic bile acid, ursodeoxycholic acid, may also provide some protection against CRC by reducing the luminal concentration of the procarcinogen deoxycholic acid. Finally, a small reduction in the risk of CRC through dietary intake of folate has also been suggested in patients with UC.

Conclusions

Improvements in the management of patients with IBD have not

significantly influenced the incidence of CRC. The reduction of mortality from CRC in IBD patients relates to detection of CRC or its precursors at an earlier stage through surveillance colonoscopy, and to the indications for total colectomy in chronic and severe forms of IBD. Chemoprevention with 5-ASA at a daily dose of 1.2 g is also recommended. ■

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IBD Research Review

As part of the WGO's campaign to raise awareness about inflammatory bowel disease (IBD) throughout 2010, an IBD expert will be recommending and highlighting a "gold standard" article on IBD, with a direct link to the original source, in each issue of e-WGN this year.



ARTICLE

Perminow G, Beisner J, Koslowski M, et al. Defective Paneth cell-mediated host defense in pediatric ileal Crohn's disease. *Am J Gastroenterol* 2010;105:452-9 (PMID: 19904243).



IBD EXPERT: CHARLES BERNSTEIN

Professor Bernstein, a Canadian IBD expert, is the chairman of the WGO's IBD guideline review team. He recently completed WGO's new IBD guideline, featuring strong cascades, and is a well-known speaker on the topic of IBD, with more than 200 published papers in the field. Professor Bernstein is also this year's World Digestive Health Day campaign leader.

Charles Bernstein comments on his IBD article of choice: A current hypothesis regarding the trigger for Crohn's disease is that there is a dysbiosis or alteration in the intestinal flora, to which patients with Crohn's disease mount an aberrant immune response. While a number of groups have reported on the relative differences in certain bacterial species in patients with Crohn's disease versus healthy control individuals, and some have reported on specific organisms such as an adherent invasive form of *Escherichia coli* that is associated with Crohn's disease, it is unclear what the underlying mechanism might be that facilitates a change in the intestinal flora.

Paneth cells are secretory epithelial cells located at the base of crypts and are a major source of antimicrobial peptides known as defensins. The differentiation and maturation of

Paneth cells are regulated by the Wnt signaling pathway, of which TCF-4 is an important effector molecule. In this study, Perminow et al. report on the expression of the mRNA for human α -defensin-5 (HD-5) and TCF-4 in pediatric Crohn's disease tissue. They found that in those with ileal Crohn's disease, the ileal expression of HD-5 and TCF-4 was reduced, while in the same individuals, the colonic expression of both HD-5 and TCF-4 was unchanged from that in healthy controls. On assessing the colonic expression of these proteins, they found that while the colonic expression of HD-5 was unchanged in those with ileal disease, it was markedly increased in those with disease of the colon. Colonic Paneth cell metaplasia is a typical finding in chronic IBD affecting the colon. Notably, the changes in HD-5 in the ileum were unrelated to fecal calprotectin levels (and hence

unlikely to be merely a response to inflammation), whereas colonic HD-5 levels did correlate with fecal calprotectin—raising the possibility that this could be a response to inflammation. Colonic expression of TCF-4 was reduced in those with ileal disease and unchanged in those with colonic disease.

The authors concluded that changes in small-bowel HD-5 and TCF-4 mRNA may play a primary role, impairing the host defense against local microbes, whereas in colonic disease the changes in HD-5 are a response to the Paneth cell metaplasia that arises secondary to inflammation. These findings underscore the different pathogenetic mechanisms between ileal and colonic Crohn's disease and the potentially important role of Paneth cells and defensins in ileal disease. ■

IBD and the limits of PubMed



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Introduction

PubMed, with its more than 19.5 million citations for biomedical articles from MEDLINE and life science journals, is the world's most powerful online medical information platform. We are all using it—well, you should be. So it was a brilliant decision by the then US Vice-President, Al Gore, to make it available free for everyone in 1996. Figure 1 shows the historic moment of the very first search in PubMed.

This article deals with the following topics:

- What is covered in PubMed and what is not?
- Getting it out—MeSH thesaurus entries for IBD.
- Planning a search for IBD—can we go “evidence-based”?
- Who's who in IBD?—citation and publication analyses
- The WGO's Ask a Librarian (AAL) service

Coverage—what's not in there?

Before we delve into what is and what is not covered in PubMed, are you aware of “PubMed On Tap Lite”? With mobile phones playing an increasingly important role in access to health information in developing countries (see the world's premier health information service for developing

countries at www.HIFA2015.org), did you know you can run a “Lite” version of PubMed from your iPhone? [Click here](#) for the latest details.

So, what is covered in PubMed? Well, all of the information included in PubMed comes from 5000 or so journals. Any non-journal-based information is not covered. And even if it is covered in a journal in PubMed—for example, the very recent IBD guideline from the World Gastroenterology Organization, chaired by Charles Bernstein (PMID: 19653289) you will have to go elsewhere for the full text in six different languages (on the [WGO web site, here](#)).

Table 1 gives a summary of useful sources for IBD information that are not available in PubMed.

It may be in there, but how do you get it out? MeSH thesaurus entries for IBD

The MeSH (Medical Subject Headings) thesaurus is the controlled vocabulary resource used to index MEDLINE. MeSH terminology provides a consistent way of retrieving information in which different terminology may be used for the same concepts. So any article reviewing “morbus Crohn” is indexed with “Crohn disease” and can then be retrieved by exploding the broader term “inflammatory bowel diseases.”

How can one find MeSH terms? Type sufficiently unique words—for example, “inflammatory bowel,” into the MeSH database for details about IBD. This supplies a definition:

- **Inflammatory Bowel Diseases**
Chronic, non-specific inflammation of the GASTROINTESTINAL TRACT. Etiology may be genetic or environmental.

This higher-level term includes Crohn disease and ulcerative colitis as “narrower” terms.

Alternatively, you can type a few keywords into the PubMed search box—e.g., to look for articles that have the words “enteritis” or “inflammatory bowel” in the title field, and then view the indexing for this record. This way you are also likely to find the relevant MeSH term “inflammatory bowel diseases.”



Fig. 1 June 26, 1997: the first search of PubMed, by Vice-President Al Gore at the U.S. Capitol. [PD] Photo: National Center for Biotechnology Information.

Table 1 The limits of PubMed/MEDLINE

What you can't get in PubMed	What you have to do/add:	Notes
All relevant published research about IBD—1 Not all journals are covered	Add Embase at least	Many other databases could still add relevant citations—for example, the large nursing database CINAHL. But this is only relevant if you go “evidence-based”
All relevant published results about IBD—2 Drug-indexing not as “deep” as in Embase	Add Embase at least	For the connoisseurs - use Embase drug indexing to search Medline or use www.embase.com
All relevant published results about IBD—3	Add a regional Medline/Index Medicus for: – Africa – Eastern Mediterranean – Latin America and Caribbean – South-East Asia – Western Pacific	Regional health and medical databases have been compiled to complement MEDLINE. Although most of the significant medical periodicals published in developed countries are indexed in the MEDLINE database, there is still a considerable amount of important and valuable medical and health documentation from countries outside the major industrialized areas that is not included. This material therefore receives less global visibility, in spite of its often higher relevance for other developing countries
Ongoing clinical trials in IBD	1. WHO—ICTRP platform 2. Clinical trial registries	1. Unique search platform for trials —includes Japanese, Chinese, and seven other national registries, including the U.S. one (www.clinicaltrials.gov) 2. Scattered—not so easy to find / Ask a Librarian: www.worldgastroenterology.org/ask-a-librarian.html
All randomized controlled trials on IBD	Cochrane Database of Controlled Clinical Trials (CENTRAL), and top-up with Embase and Medline	Access free through HINARI for many developing countries (but not all); see: http://www.who.int/hinari/eligibility/en/
IBD guidelines	www.ngc.org —the US National Guidelines Clearing House; plus, check the major gastroenterology societies and www.sign.ac.uk	But available in PubMed as a citation with abstract if published in one of the journals covered by PubMed. SIGN can help your country produce guidelines
Evidence-based clinical information resources	1. www.uptodate.com 2. www.tripdatabase.com	1. The market leader—“for profit”; excellent 2. Very good—for free; very interesting features, but there are many others
Ranked analysis of fields—e.g., top IBD authors, top IBD journals, top IBD topics, top IBD institutions	Use MEDLINE on a host with ranking technology, e.g., OVID, Datastar, DIMDI, STN, Dialog, etc.	Or use citation databases such as SCOPUS and the Web of Science
Citation information— e.g., most cited IBD author, most cited IBD article, etc.	Use a citation database such as SCOPUS or Web of Science	No controlled vocabulary, but all references of all articles are included, thus allowing citation trails.

The importance of knowing “term history.” The MeSH database also gives the year the term was introduced—in the case of “inflammatory bowel diseases,” it was 1989. Very importantly, it also tells you how the concept was indexed before 1989:

- **Previous Indexing:**
[Colitis, Ulcerative \(1966–1988\)](#)
[Crohn Disease \(1980–1988\)](#)

So, if you wish to do a search for inflammatory bowel diseases going back further than 1989, you may want to include the previous indexing terms. “Colitis, ulcerative” takes you back to 1966, but “Crohn disease” only to 1980. How was Crohn disease indexed before 1980, then? It wasn’t! Entering “Crohn disease” in the MeSH database does not give an entry for previous indexing, but it does give a number of

synonyms/entry terms:

- Crohn’s Disease
- Crohns Disease
- Ileocolitis
- Ileitis, Terminal
- Ileitis, Regional
- Colitis, Granulomatous
- Enteritis, Granulomatous
- Enteritis, Regional

These could be used if you want to search for Crohn disease articles further back than 1980.

Each database you search has its own controlled vocabulary—in the Embase thesaurus (EMTREE) for example, IBD is searched with the controlled term “enteritis,” and IBS with the controlled term “Irritable colon.” Should you bother to add another database to your search? Well, yes and no. No, because MEDLINE covers all the top 50 or so gastroenterology journals and all the top general journals—so why add Embase? Yes, because if you want to claim to be evidence-based, then your searches must stand up to rigorous scrutiny, and if others find an IBD trial in Embase that you did not find in Medline ... you are in trouble.

Planning a search on IBD

Not just gastroenterologists but librarians too need to differentiate between irritable bowel and inflamed bowel (PMID: [20331581](#)). Not just the way to search, but also the nomenclature itself is a little confusing.

It is so easy to confuse “inflammatory” with “inflamed” and with “irritable”—and even more so for nonnative speakers like me.

A comprehensive search for IBD in PubMed is challenging, because it is a so-called “explosion” term; the term can be exploded, because it has narrower terms below it—ulcerative colitis and Crohn’s disease. And whilst you do not need the highly sensitive search strategies of a Cochrane Trials Search Coordinator, you can do better than just typing IBD in the PubMed search box (this gives 7159 results—compare that with the results shown in Table 2).

In PubMed, explosion (automatically searching all narrower index terms under a broader index term) is turned on by default. It is worthwhile to add all of the results together (using the Boolean “OR”) in order to get a more comprehensive result (now 60,481).

Who’s who in IBD?—citation and publication analyses

There are two comprehensive

citation databases, both of which are commercial publications. [The Web of Science](#) is published by Thomson Reuters, and the more recent SCOPUS is published by Elsevier Science ([www.scopus.com](#)). Let’s have a look at the Web of Science first and see what you can do with IBD.

Which are the most highly cited articles in IBD? If I search for “inflammatory bowel disease” OR “Crohn’s disease” OR “ulcerative colitis” (there is no controlled vocabulary in the Web of Science), I get 55,573 records. The six articles shown in Fig. 2 are the most highly cited ones. That means they occur most frequently in the lists of references of these 55,573 articles. Record no. 1, for example, is cited 2656 times in the 55,573 articles.

Who has published most on IBD? Using the same results of the previous search, the WoS can count how many times an individual author occurs in this population of 55,573 articles. In this way we can arrive at a “league table” for each topic searched. The top

You search this:	Hits	PubMed searches this:
1. Inflammatory bowel disease	55384	“inflammatory bowel diseases”[MeSH Terms] OR (“inflammatory”[All Fields] AND “bowel”[All Fields] AND “diseases”[All Fields]) OR “inflammatory bowel diseases”[All Fields] OR (“inflammatory”[All Fields] AND “bowel”[All Fields] AND “disease”[All Fields]) OR “inflammatory bowel disease”[All Fields]
2. Inflammatory bowel diseases	51717	“inflammatory bowel diseases”[MeSH Terms] OR (“inflammatory”[All Fields] AND “bowel”[All Fields] AND “diseases”[All Fields]) OR “inflammatory bowel diseases”[All Fields]
3. Crohn’s disease	31008	“crohn disease”[MeSH Terms] OR (“crohn”[All Fields] AND “disease”[All Fields]) OR “crohn disease”[All Fields] OR (“crohn’s”[All Fields] AND “disease”[All Fields]) OR “crohn’s disease”[All Fields]
4. Ulcerative colitis	28651	“colitis, ulcerative”[MeSH Terms] OR (“colitis”[All Fields] AND “ulcerative”[All Fields]) OR “ulcerative colitis”[All Fields] OR (“ulcerative”[All Fields] AND “colitis”[All Fields])
All together (1 or 2 or 3 or 4)	60481	#1 or #2 or #3 or #4

Table 2 Searching for IBD in PubMed (note the difference between 1 and 2)*

*No.2 does not have the “exact match” or the broader free-text search in all fields for the singular.

Tabl 3 The six most frequently cited articles on IBD, from the Web of Science.

1.	Title: Nitric oxide, superoxide, and peroxynitrite: The good, the bad, and the ugly • Author(s): Beckman JS, Koppenol WH • Source: AMERICAN JOURNAL OF PHYSIOLOGY- CELL PHYSIOLOGY Volume: 271 Issue: 5 Pages: C1424-C1437 Published: NOV 1996 • Times Cited: 2,656
2.	Title: Mechanisms of disease – Nuclear factor-kappa b – A pivotal transcription factor in chronic inflammatory diseases • Author(s): Barnes PJ, Larin M • Source: NEW ENGLAND JOURNAL OF MEDECINE Volume: 336 Issue:15 Pages: 1066-1071 Published: APR 10 1997 • Times Cited: 2,262
3.	Title: Association of NOD2 leucine-rich repeat variants with susceptibility to Crohn’s disease • Author(s): Hugot JP, Chamaillard M, Zouali H, et al • Source: NATURE Volume: 411 Issue: 6837 Pages: 599-603 Published: MAY 31 2001 • Times Cited: 2,082
4.	Title: A frameshift mutation in NOD2 associated with susceptibility to Crohn’s disease • Author(s): Ogura Y, Bonen DK, Inohara NH, et al • Source: NATURE Volume: 411 Issue:6837 Pages: 603-606 Published: MAY 31 2001 • Times Cited: 1,994
5.	Title: Genome-wide association study of 14.000 cases of seven common diseases and 3.000 shared controls • Author(s): Burton PR, Clayton DG, et al • Source: NATURE Volume: 447 Issue:7145 Pages: 661-678 Published: JUN 7 2007 • Times Cited: 1,720

author—“Rutgeerts P”—was found in the author field for 547 records. Of course, he may not be the most frequently cited author—that would be another search. Fig. 3 shows a list of the top authors—note the very popular author “Anon.”

What are the top 13 journals for IBD?

Another aspect that PubMed does not provide is a way of finding which journals publish most frequently on a given topic—IBD, for example. Is this important? Perhaps. If you were starting a library on IBD, you would want to be sure to subscribe to the best journals. Again, I do my search for “inflammatory bowel disease” OR “Crohn’s disease” OR “ulcerative colitis” in the Web of Science, but now I want to know which journals publish most frequently on IBD (Table 4).

What are the top institutions publishing on IBD? And of course, you can do the same for institutions as well—Fig. 5 shows a league table of institutions involved with IBD, as analyzed on the Web of Science

Field: Author	Record count	% of 50000
RUTGEERTS, P	547	1.0940 %
SANDBORN, WJ	480	0.9600 %
COLOMBEL, JF	435	0.8700 %
SCHREIBER, S	377	0.7540 %
JEWELL, DP	329	0.6580 %
HANAUER, SB	307	0.6140 %
VERMEIRE, S	283	0.5660 %
TARGAN, SR	270	0.5400 %
SATSANGI, J	229	0.4580 %
LOFTUS, EV	221	0.4420 %
SCHOLMERICH, J	215	0.4300 %
LICHTENSTEIN, GR	210	0.4200 %
TREMAINE, WJ	197	0.3940 %
FAZIO, VW	174	0.3480 %
HIBI, T	172	0.3440 %
BERNSTEIN, CN	170	0.3400 %
GEBDES, K	165	0.3300 %
PALLONE, F	164	0.3280 %
KIRSNER, JB	163	0.3260 %
VAN ASSCHE, G	161	0.3220 %
[ANON]	156	0.3120 %
FORBES, A	155	0.3100 %
CORTOT, A	154	0.3080 %
MATSUMOTO, T	153	0.3060 %

Table 4 The most highly published authors in IBD – from the Web of Science.

Table 5 The best journals for IBD, from the Web of Science.

Field: Source Title	Record count	% of 50000
GASTROENTEROLOGY	5241	10.4820 %
GUT	2446	4.8920 %
AMERICAN JOURNAL OF GASTROENTEROLOGY	2396	4.7920 %
INFLAMMATORY BOWEL DISEASES	2219	4.4380 %
SCANDINAVIAN JOURNAL OF GASTROENTEROLOGY	1042	2.0840 %
DISEASES OF THE COLON & RECTUM	1003	2.0060 %
DIGESTIVE DISEASES AND SCIENCES	1001	2.0020 %
ALIMENTARY PHARMACOLOGY & THERAPEUTICS	929	1.8580 %
EUROPEAN JOURNAL OF GASTROENTEROLOGY & HEPATOLOGY	715	1.4300 %
JOURNAL OF PEDIATRIC GASTROENTEROLOGY AND NUTRITION	624	1.2480 %
JOURNAL OF CLINICAL GASTROENTEROLOGY	568	1.1360 %
WORLD JOURNAL OF GASTROENTEROLOGY	502	1.0040 %
JOURNAL OF GASTROENTEROLOGY AND HEPATOLOGY	433	0.8660 %

platform. Such an analysis is based on a frequency distribution of the affiliation field for the corresponding author. This may not be the first author, and so there is substantial opportunity for bias.

Everything you always wanted to find out about IBD—the WGO’s Ask a Librarian service

Any of the databases mentioned above can be searched for you by the Ask a Librarian (AAL) service. This is a free service for gastroenterologists, hepatologists, and endoscopists who live and work in countries that qualify for the Health InterNetwork Access to Research Initiative (HINARI)—see <http://www.who.int/hinari/eligibility/en/>. The AAL can be accessed via the World Gastroenterology Organization home page at <http://www.worldgastroenterology.org/ask-a-librarian.html>.

The medical oath of Maimonides (1138–1204) can serve as an inspiration: “Today we can discover our errors of yesterday, and tomorrow we can obtain new light on what we think ourselves sure of today.” Isn’t that pure evidence-based medicine? ■

Field: Author	Record count	% of 50000
HARVARD UNIV	932	1.8640 %
UNIV CHICAGO	688	1.3760 %
MAYO CLIN	683	1.3660 %
UNIV TORONTO	585	1.1700 %
ST MARKS HOSP	480	0.9600 %
UNIV CALIF LOS ANGELES	436	0.8720 %
UNIV PENN	433	0.8660 %
UNIV CALGARY	426	0.8520 %
MAYO CLIN & MAYO FDN	425	0.8500 %
UNIV COPENHAGEN	406	0.8120 %
CLEVELAND CLIN FDN	397	0.7940 %
MASSACHUSETTS GEN HOSP	397	0.7940 %
MCMASTER UNIV	370	0.7400 %
UNIV N CAROLINA	339	0.6780 %
UNIV AMSTERDAM	334	0.6680 %
CEDARS SINAI MED CTR	317	0.6340 %
UNIV PITTSBURGH	307	0.6140 %
KAROLINSKA INST	298	0.5960 %
JOHNS HOPKINS UNIV	293	0.5860 %
JOHN RADCLIFFE HOSP	284	0.5680 %
UNIV REGENSBURG	281	0.5620 %
UNIV ALBERTA	269	0.5380 %
UNIV MUNICH	269	0.5380 %

Table 6 Frequency distribution of institutions publishing on IBD, from the Web of Science.

The principles of screening for colorectal cancer: an international guide



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Definition. Screening is the process of testing for the presence of a specific disease in apparently healthy people who have no recognized high-risk factors for that disease. Screening is distinct from surveillance, which is the testing of those who have a recognized high-risk factor.

Goal. The goal of screening is to reduce the mortality resulting from the type of cancer concerned. For it to be successful, cancer or its precursor states must be detected at a stage at which they are curable; this is best achieved in practice by detection before the onset of symptoms.

Is early detection enough to prove benefit? Early detection alone is insufficient to prove that a screening test is beneficial unless randomized controlled population trials have been conducted to prove that screening reduces the burden of disease (the disease-specific mortality) in the absence of several types of bias. Such biases include selection bias and lead-time bias—the latter being the illusion of increased survival due to earlier detection, without a real increase in longevity.

Overview of the proof of benefit. Early detection of colorectal cancer (CRC)

by screening using fecal occult blood testing (FOBT) has been proved to reduce mortality. One study has shown that a sensitive FOBT also reduces the incidence of CRC.

Just in the last month, a population trial of flexible sigmoidoscopy has confirmed that this method also reduces incidence, confirming the strategic value of polyp detection and removal in an unbiased design.

Other methods of screening, such as colonoscopy, and computed-tomographic colonography, are supported by lesser levels of evidence, which are subject to bias. Nonetheless, an improved capacity to detect preinvasive lesions, namely adenomas, can potentially reduce the incidence as well as the mortality.

Contexts of screening. Screening for colorectal cancer is usually carried out in either individualized (opportunistic) or population settings. *Population or mass screening* involves an organized and systematic approach aimed at maximum participation in screening within a population and is usually applied in universal health-care systems. Through a standardized and often impersonal approach, it seeks to engage individuals in at least some form of preventive activity—in effect, doing something is better than doing nothing.

Nature of screening. No matter what the context, screening is by its nature a process that aims to improve the likelihood that affected people receive effective diagnosis and treatment while the disease is still at a curable and usually asymptomatic stage. Screening is therefore a process involving step-wise phases: participation; performance of the screening test; if that is positive, performance of the diagnostic test; and if adenoma or cancer is found, implementation of the appropriate treatment.

In comparison with opportunistic screening, organized screening focuses much greater attention on the quality of the screening process, including follow-up of participants. Consequently, organized screening provides greater protection against many of the harmful effects of screening, including overdiagnosis, poor quality and complications of screening, and poor follow-up of those who test positive.

What justifies screening in a given population? The criteria that justify screening were defined four decades ago. In today's terms, these can be simply expressed as follows:

- The disease should be an important cause of death in the target population.
- A test must be available that is capable of detecting early-stage disease in asymptomatic people. Originally, the principles

Table 1 Screening for various populations

Criterion justifying screening	Status	Comment
Important cause of death	Country-dependent	Justified in countries/regions with incidence approaching that of Australia, USA, France, etc.
Noninvasive test for curable disease	GFOBT and FIT are proven examples	Proven in absence of bias
Invasive test for curable disease	Colonoscopy, sigmoidoscopy	These are better at detecting preinvasive lesions (adenomas)
Reduces mortality—RCT	GFOBT	FITs are now considered superior to GFOBTs
Reduces incidence—RCT	GFOBT, flexible sigmoidoscopy	
Reduces mortality—case-control studies	Sigmoidoscopy, colonoscopy	
Acceptable test for the screenee	Will vary by test and will be country/region-dependent	Rates differ substantially between these tests. Cultural factors affect acceptability
Cost-effective	Yes, if the disease is of sufficient prevalence	Studies vary. Recent studies have concluded that GFOBT/FIT are more cost-effective
High-quality diagnosis and treatment	Country-dependent	Might apply to certain subpopulations
Feasible and accessible	Country-dependent	Might apply to certain subpopulations

FIT, fecal immunochemical test for hemoglobin using a specific antibody; GFOBT, guaiac-based fecal occult blood tests such as Hemoccult; RCT, randomized controlled trial.

specified a simple, noninvasive and cheap screening test (two-step screening), although this should not necessarily preclude one-step screening using an invasive test.

- It must be proved that detecting early-stage disease is worthwhile in that it reduces the mortality, based on unbiased studies.
- The test must be acceptable to the target population.
- The entire screening process must be judged to be cost-effective using current standards.
- High-quality diagnostic follow-up of a positive screening test, plus high-quality treatment, must be available. Without these, benefit will not follow.

- Screening must be feasible with the available resources. In the first place, this means that the test must be accessible to the population. Many countries place a high priority on equality of access to screening tests.

Table 1 shows ways in which these principles can be applied to populations around the world.

A practical approach to country-dependent criteria. *Importance of the problem.* The ranking of CRC as a cause of mortality relative to other cancers warrants major consideration. A simple way to assess this is to carry out a pilot study of FOBT or flexible sigmoidoscopic screening in a target population, to ascertain the prevalence

of the disease.

Feasibility. Countries with organized screening programs have already undertaken feasibility assessment. This includes testing the screening process in an existing health-care system. It is likely that countries with limited resources will be restricted to two-step screening, using an FIT set to deliver a manageable colonoscopy rate.

Test acceptability. This can be appropriately assessed in a pilot feasibility study. If people are unwilling to do the test, no benefit will be seen at the population level.

Conclusions. Several important local considerations need to be assessed. These include the ranking of CRC as a problem, the acceptability of the test to the target population, the feasibility of the program within the existing health-care system, and the capacity to deliver high-quality treatment and follow-up after a positive screening test. Pilot programs should be implemented in a progressive fashion to assess each of these factors in relation to local circumstances. ■

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The purpose and importance of the first World IBD Day

In an effort to raise awareness of inflammatory bowel disease throughout 2010, the WGO is collaborating with IBD patient groups and fully supports the World IBD Day 2010 initiative.



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For the first time, patient organizations concerned with Crohn's disease and colitis in countries across four continents (United States, Canada, Brazil, Europe and Australia) have set May 19, 2010 as "World IBD Awareness Day," as part of a new campaign to increase public understanding of Crohn's disease and ulcerative colitis (collectively known as IBD) and their often devastating impact on lives all over the world. The campaign also aims to increase awareness and confidence among IBD patients in knowing that they are not alone in their battle with these disabling diseases.

IBD affects millions of people around the world, generally in the prime of life, and with an increasing incidence among children and adolescents. In the United States alone, 1.4 million Americans are living with IBD, with a similar number in Europe, and as many as 5 million are affected worldwide. In spite of these growing numbers, Crohn's disease and ulcerative colitis are often misunderstood and are not among the diseases and causes that people can easily identify, such as cancer or diabetes.

The goal of the inaugural World IBD Day event is to generate global

awareness of these increasingly common, serious, and incurable digestive diseases. The patient organizations working in the field include the Crohn's & Colitis Foundation of America (CCFA), the Crohn's and Colitis Foundation of Canada (CCFC), Crohn's and Colitis Australia, the Crohn's and Colitis Association of Brazil (ABCD), and the European Federation of Crohn's & Ulcerative Colitis Associations (EFCCA). The World Gastroenterology Organization (WGO) has also joined forces with these organizations, bringing together patients and medical organizations in order to maximize the reach of the campaign's message.

It is crystal clear that the challenges of IBD do not stop at any one border. Through the World IBD Day campaign, the Crohn's and colitis patient organizations hope to find synergies and opportunities to ease the difficulties that IBD patients and families experience as a result of the lack understanding that exists today among the general population. The patient organizations also hope that the awareness that the campaign generates through social media and other forms of outreach will help advance the

research needed to find cures.

World IBD Day is a great symbol for what can be accomplished in a global setting. Whether people visit the new web site (www.worldibdday.com) or post a supportive "tweet for IBD awareness" on Twitter (www.twitter.com/worldibdday), anyone can get involved and make a difference for IBD patients all over the globe. Educational materials and other resources can also be accessed online.

For more information on how you can help support World IBD Day, contact:

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International meetings in Asia and Latin America

THE LARGEST GASTROENTEROLOGY MEETING IN LATIN AMERICA: THE 32ND PAN-AMERICAN CONGRESS ON DIGESTIVE DISEASES: GASTRO-GUAYAQUIL 2010

September 30–October 4, 2010



Carlos Ledesma
President, AIGE

The 32nd Pan-American Congress on Digestive Diseases (Gastro-Guayaquil 2010) will take place from September 30 to October 4, 2010. The event is the largest and most prestigious gastroenterology meeting in Latin America, attracting more than 3000 physicians, and will be held at the Convention Center in Guayaquil, Ecuador.

Speakers from all over the Western world will be contributing to the Scientific Program in the field of Gastroenterology and Digestive Endoscopy. The program has been developed by the Scientific Committee to cover the continuing advances being made in the Science of

Gastroenterology, Digestive Endoscopy, Hepatology, and Digestive Surgery.

The 32nd Pan-American Congress on Digestive Diseases will be offering interdisciplinary symposia on new approaches to diagnosis and treatment, with a special emphasis on innovative technical advances in the noninvasive management of gastrointestinal and hepatic disorders. Participants will also be able to take part in two full-day Postgraduate Courses in Gastroenterology and Endoscopy and a simultaneous satellite transmission from the 2010 Boston International Live Endoscopy Course.

The conference is being organized jointly by the *Asociación Interamericana de Gastroenterología (AIGE)*, the *Sociedad Interamericana de Endoscopia Digestiva (SIED)*, the

Asociación Latinoamericana para el Estudio del Hígado (ALEH), the Latin-American Association for the Study of the Liver), the World Gastroenterology Organization (WGO), and the World Organization of Digestive Endoscopy (OMED).



THE ASIAN-PACIFIC DIGESTIVE WEEK 2010 COMES TO MALAYSIA

September 19–22, 2010



K.L. Goh
President, APDW 2010

The Malaysian Society of Gastroenterology and Hepatology (MSGH) is hosting the Asian-Pacific Digestive Week (APDW) for the first time from September 19 to September 22, 2010,

focusing on “Gastroenterology in the Asian-Pacific Region: Excellence in the New Decade.” The meeting will be held in the Kuala Lumpur Convention Center, and more than 3000 participants are expected.

The APDW 2010 is the tenth such conference since the successful

inaugural meeting in 2001 in Sydney under the presidency of Professor Geoff Farrell. The APDW is modeled on the Digestive Disease Week (DDW) in the United States and the United European Gastroenterology Week (UEGW). The meeting brings together annually the scientific meetings of four Asian-Pacific

societies: the Asian–Pacific Association of Gastroenterology (APAGE), the Asian–Pacific Society of Digestive Endoscopy (APSDE), the Asian–Pacific Association for the Study of the Liver (APASL), and the International Society of Digestive Surgery (ISDS).

The conference will be preceded by two workshops:

- The APDW 2010 Postgraduate Course, chaired by Professors Guido Tytgat and Shu-Dong Xiao, focusing on digestive cancer (co-sponsored by APAGE, IDCA, and EAGE)
- The OMED/APSDE Endoscopy Directors' Workshop, chaired by Professors Anthony Axon and William Chao

The main scientific meeting will have five concurrent sessions. Highlights of the conference will include the two named lectures organized by the Journal of Gastroenterology and Hepatology Foundation (JGHF):

- The Okuda Lecture, given this year by Professor Anna Lok (Ann Arbor, USA): “Does antiviral therapy for hepatitis B and C prevent hepatocellular cancer?”
- The Marshall and Warren Lecture, given this year by Professor Neville Yeomans (Melbourne, Australia) on “Aspirin: old drug, new uses and challenges?”



www.apdw2010.org.my



The Chinese Society of Gastroenterology (CSGE) meeting in Guangzhou in December 2009 was attended by approximately 3000 GIs. The WGO looks forward to cooperating with the CSGE in Shanghai in 2013, in a combined meeting with APAGE (Asian Pacific Digestive Week Foundation/APDWF), OMED, and a Federation of four of the Chinese gastroenterology societies.

Dr. Yanfei Liu, Deputy Secretary General, Chinese Medical Association, Dr. Nanshan Zhong, President, Chinese Medical Association, Dr. Richard Kozarek, President, WGO, Dr. Daiming Fan, President, Chinese Society of Gastroenterology, Dr. Guoming Qi, Vice President, Chinese Medical Association, Dr. Lan Lan, Deputy Secretary General, Guangdong Medical Association

WGO cascades in Sudan



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Cascades are methods used to present treatment recommendation that can be adapted to the resource and infrastructure available in each country. WGO global guidelines aim to make international guidelines usable for physicians working in developing countries with poor resources.

The main reason for the “cascades” used in the WGO Global Guidelines—which present treatment recommendations that can be adapted to the resources and infrastructure available in each country—is that they make the international guidelines usable for physicians working in developing countries with poor resources.

The first question to be asked is: do physicians in low-resource countries actually want guidelines? When we asked 500 physicians working in Khartoum State, Sudan—mostly registrars and medical officers—whether they needed guidelines, 95% said they did. An Internet search shows that there are many medical guidelines that are used in resource-poor countries or middle-income countries, such as Colombia and Thailand.

Value of guidelines for physicians. Global guidelines are important for physicians working in developing countries, as they often have only limited time to keep up with the

advances being made in medicine. Most of them work in the morning in government hospitals and in the evening in private clinics. The study materials available to them are also limited. Guidelines are therefore important in providing continuing professional education and in encouraging medical doctors to abandon outdated practices. Many physicians still have knowledge that was acquired during their training periods many years ago.

A very important aspect of medical practice in developing countries is that physicians often have different training backgrounds. In our gastroenterology unit at Ibn Sina Hospital in Khartoum, we have had physicians whose postgraduate training was in the Sudan, United Kingdom, Germany, Russia, Ireland, Austria, and South Africa. Global guidelines can provide these doctors with a common language and can help prevent variations in practice.

The guidelines can also improve physicians’ status and their pride in their own work, as they can boast and say: “I am using the global guidelines written and recommended by the World Gastroenterology Organization.”

The global guidelines can also avoid information overload for physicians, as many societies and companies produce guidelines that are freely

available on the Internet, and some of the recommendations given in them are conflicting. Global guidelines can rationalize drug use in developing countries and prevent unnecessary use of drugs—e.g., intravenous vitamins for viral hepatitis, multivitamins for diabetics, and unnecessary use of antibiotics.

Value of guidelines in relation to government and other organizations.

Guidelines based on the cascade system can enable physicians to obtain funds from the government or even private donors and can strengthen the position of physicians in dealing with administrators. The practitioner is able to state that the treatment is approved by the international community.

Developing countries are also often unable to produce their own guidelines for medical practice. Guidelines are expensive to produce and require expertise in many fields, such as epidemiology, biostatistics, health-care research, and clinical medicine, as well as the need for writing and editing. There is often a lack of such experts in developing countries, and it may therefore be beyond their capacity to produce such guidelines on their own.

Value of guidelines in relation to patients. Patients in developing countries often lack education and are unable to take part in the management of their health problems. Physicians are the sole decision-makers and therefore have to be equipped with good practical and up-to-date medical

knowledge—otherwise they will be forced to use guidelines derived from interest groups such as the pharmaceutical companies.

Global guidelines are also now becoming more relevant internationally, as diseases once thought to be absent in developing countries are beginning to emerge there as well—e.g., reflux esophagitis is now more common in Sudan than duodenal ulcer. Cholecystectomy is now the commonest abdominal operation in Khartoum. Diseases such as inflammatory bowel disease, celiac disease, obesity, and cancer are catching up very rapidly with rates in the developed countries. The emergence of many generic and less expensive drugs will eventually make

it possible for them to be used in developing countries.

Conclusions. The cascade system has now been in existence for 10 years, and I think it has proved its value, as the WGO web site shows a high rate of visits from developing countries. A good example of the use of cascades in practice is the management of bleeding esophageal varices in Sudan in patients with schistosomiasis, for whom simple and cheap alternatives are frequently used effectively.¹ I would conclude by quoting Pang et al.:² “Applying what we already know will have a bigger impact on health and disease than any drug or technology likely to be introduced in the new decade.” ■

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