OCTOBER 2008

Gastroenterology IN PRIMARY CARE

JOURNAL OF THE PRIMARY CARE SOCIETY FOR GASTROENTEROLOGY

REPORT FROM THE

ANNUAL SCIENTIFIC MEETING OF THE Primary Care Society for Gastroenterology

This year's annual scientific meeting of the PCSG provided primary care practitioners (PCPs) with a chance to explore and discuss the management of a wide range of disorders and issues commonly presented within the field of gastroenterology.

The management of coeliac disease (CD) and inflammatory bowel disease (IBD), primary care educational needs and perspectives of the patient with a chronic gastrointestinal (GI) disease were discussed.

Coeliac disease management in primary care

Dr Butt (GP, Ashford) reviewed CD, which is estimated to affect around 140,000 people in the UK; however no database is available to confirm absolute numbers. The number of patients diagnosed with CD has grown substantially over the last few years, perhaps an indication of better disease awareness and diagnostic tools.

Although most patients are diagnosed using classic symptoms, e.g. diarrhoea, weight loss and fatigue; non-specific symptoms, including unexplained anaemia, irritable bowel syndrome (IBS) - like symptoms or no obvious symptoms also present. The majority of CD cases are identified in adulthood. However, children as young as 6 months have been diagnosed. A review found that certain patient groups have an increased prevalence of CD, which may assist with the identification of new cases (Table 1).

Table 1 PATIENT GROUPS WITH INCREASED PREVALENCE OF COELIAC DISEASE

Dermatitis herpeteformis	69-89%
Recurrent apthous ulcers	10-18%
Iron deficiency anaemia	2.5-5.7%
IBS	0-11.4%
First degree relative	4-22%

All patients suspected of CD should undergo blood tests (IgA endomysial antibody or IgA tissue transglutimanase, sensitivity 90%), biopsy and move to a gluten free diet. A re-challenge and future biopsy is recommended for any patient diagnosed <2 years of age. Disease status, dietary compliance, blood tests, clinical assessment (body mass index, GI symptoms and osteoporosis) and treatment must be reviewed yearly. All patients should receive the pneumococcal vaccine. Every 2-3 years, patients need to be reviewed by a dietician. Referral to a gastroenterologist should be considered if patients are responding poorly to diet and treatment, blood is evident in stools or there are new and unexplained symptoms.

Addressing the needs of IBD patients in primary care

The management of IBD is often challenging for PCPs. The issues relating to this were discussed by **Helen Griffiths** (Lead Nurse, West Midlands), **John O'Malley** (GP, The Wirral) and **Richard Driscoll** (National Association of Colitis and Crohn's Disease [NACC]).

It was broadly agreed that successful management of IBD requires a dedicated team and equal participation of the patient and their healthcare professionals (HCPs). Self-management can be particularly successful in this patient group. In primary care, sharing experiences and knowledge between the patient and PCP

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This issue...

EDITORIAL

I am taking this opportunity to write to you to introduce myself and to inform you of the changes taking place to our society. Many of these changes were discussed at the AGM and you will receive the minutes of the meeting in due course but I wanted to highlight some of the most important issues.

The most significant change is that **Richard Stevens** has stepped down as Chair of the society after nearly 10 years at the helm. Richard has guided the society through many changes and he will be sorely missed. I have assumed the Chair in his place and hope that I will be able to emulate Richard's successes. Changes to the constitution and the annual membership were discussed and passed by the membership at the meeting.

The next purpose of the editorial is to introduce myself. I have been a GP endoscopist for the past 20 years at the Norfolk and Norwich University Hospital and also at our own community endoscopy unit. I have been a committee member for 8 years and the secretary for the past 5 years. I am fully committed to promoting GP endoscopy as well as advancing the role of the GP with a special interest in gastroenterology.

The most important aim for this piece is to set out a vision for the future of the society. This will, I hope, generate comment and debate. I would like to suggest ways in which the society needs to evolve to benefit from the ever-changing medical landscape. I feel very strongly that members have a major role to play in the commissioning of primary care gastroenterological services nationally and at the PCT level. This is not just confined to endoscopy but includes the service provision of all gastroenterological conditions. For example, a number of talks at the recent ASM highlighted the abilities of many members of the society. Sohail Butt, Mark Follows and John O'Malley all demonstrated that PCSG members play a significant role in providing gastroenterological expertise in primary care.

In addition, Roger Jones's IMAGE study is likely to change the way gastrointestinal conditions are managed in primary care by promoting the role of the GP. Furthermore, Richard Driscol's National IBD Standards (a copy of which you have received) emphasises the role of the GP in managing these conditions in the community.

Recently, the DoH has approached the society for our views on future Liver Disease strategy. All these activities require the contribution of the GP specialist. There is no doubt that the members have the ability and ambition to contribute to the care of patients with gastrointestinal illnesses in primary care. I firmly believe that it is the role of the society to promote our expertise to commissioners. I would welcome members' views on how this might be achieved. I feel that the areas to concentrate on are an overview of members' interests and an indication of the willingness of the individual to assume a lead role. This will allow the society to respond to requests for advice on service provision but also ensure that services are not designed without our contribution.

I feel that within the ever-changing medical environment with its emphasis on primary care affords us an opportunity to use our expertise and enhance the role of our society.

Dr Jamie Dalrymple

Endoscopy Meeting
Wednesday 26 November 2008

East Midlands Conference Centre, University Park Campus, Nottingham NG7 2RJ

09:00 Registration and coffee

09:30 **Chairperson's welcome & introduction**, *Dr Jamie Dalrymple, GP in Norwich, Norfolk and Chair of PCSG*

09:40 **Launch of quiz**, *Dr John Galloway, GP in Kings Lynn, Norfolk and Treasurer PCSG*

10:00 **Nasal endoscopy**, *Dr Stevan Fox,*

10:45 Morning coffee

11:10 **GMC consent issues**, *Dr John O'Malley, GP in Moreton, Wirral*

12:00 **Commissioning**, *Stewart Finlay, GP in Bishop Auckland, Country Durham*

12:45 Lunch

13:40 **Quiz results**, *Dr John Galloway, GP in Kings Lynn, Norfolk and Treasurer PCSG*

14:00 **Bowel cancer screening**, *Professor John Scholefield, Professor of Surgery at Nottingham University Hospital*

14:45 Afternoon tea

15:15 **Safety and medical aspects of endoscopy**, *Roger Leicester, Trust Director of Endoscopy, St Georges Hospital, London*

16:00 Meeting close

Email secretariat@pcsg.org.uk or call 01923 712711 for a booking form.

The Society would like to acknowledge support from the following members of the Corporate Membership Scheme:

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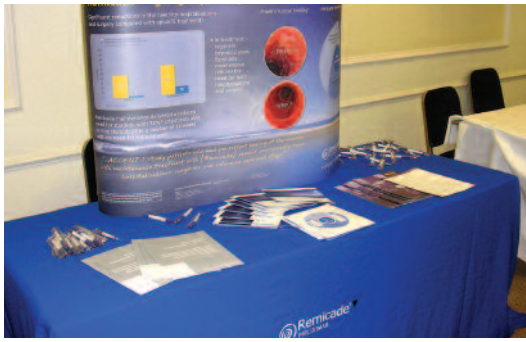
Yakult



Continued from page 1

ensures that patient expectations are met and that they feel empowered to participate in the management of their disease. As with any chronic illness, recognition of symptoms, understanding of treatment modalities and rapid management of flare ups by both the patient and PCP are crucial. This can be achieved through a patient plan of action, agreed by the patient and their PCP. The PCP, pharmacist and patient should all have a copy of this plan.

As with all chronic illnesses, annual review is important. It was suggested at the meeting that this could be done by telephone, with follow up appointments for patients with concerns, those with suspected cancer or those who have missed two telephone reviews.

The need for a dedicated multidisciplinary IBD team was agreed by all presenters. An IBD team should be clearly defined and include core staff and supporting services so that patients can be identified, treated and reviewed appropriately. It was suggested that the team should include gastroenterologists, surgeons, radiologists, dieticians, nurse specialists, counsellors and PCPs. However, there are many difficulties to be overcome in order to successfully implement this. In many primary care trusts (PCTs) not enough specialist staff are available, and IBD is frequently considered a low priority even though it has a high impact on patients lives. Additionally, there is no register of patients with IBD, making identification of patients difficult. Many patients have been diagnosed with IBD who only have suspected illness. In addition, long-term prescribing of 5-aminosalicyclic acid has been found in some patients who do not have chronic disease, making this a poor indicator of confirmed diagnosis.

The benefits of setting up an IBD service are still achievable. More refined searching to identify patients, financing to provide appropriate levels of staffing and patient involvement and education, and inclusion of patient support groups are all needed. This would ensure continuity of care, a more personalised service providing a holistic approach, easier access for patients, appointments tailored to specific patient needs and faster referrals.

The standards of care for IBD are also currently under review by the National Standards Working Group in order to address the absence of a national plan or targets for IBD patients.

A national audit has revealed that there is significant commitment and informal collaboration between HCPs. However, overuse of steroids, lack of appropriate testing and rescue therapy, and low levels of patient involvement were also identified. Many of these issues are due to lack of resources and disease awareness and resolving them will take time. Sharing good practice experiences, appropriate training as part of continuing professional development and the establishment of patient panels would all advocate improvements.

A panel discussion on the future management of IBD highlighted the need for improved relationships between primary and secondary care. This requires an acknowledgement of the need to share the workload and ensure continuity of care for IBD patients. The use of nurse specialists would be an ideal way to bridge the gap and act as the direct liaison for patients. However, the NACC has highlighted a significant shortage of IBD nurse specialists amongst the 489 UK PCTs, NHS trusts and local health authorities.

Another point raised was the overlap of developments taking place in the management of IBD patients across different PCTs. There is a real need for PCTs to communicate and work together to develop a national pathway of care. It was suggested that the PCSG should initial this discussion among its members. **Jamie Dalrymple**, Secretary of the PCSG said, "We need to evolve as a society and this should be one of our functions for the future."

Improving gastroenterology education in primary care

There is a huge burden on primary care with respect to GI disease. It is estimated that it includes 10% of the patients seen, with 80-90% of their management taking place in primary care. As a result, there is a greater need for awareness and training in this area.

Mark Follows (GP, Airedale) reviewed the educational opportunities that are now available to GPs with a special interest in gastroenterology. These include a postgraduate diploma and the Department of Health (DoH) National Endoscopy Training Programme. With this additional training, GPs can manage non-urgent referrals within primary care. However, these services need to be set up correctly and should follow the guidelines set out by the DoH. The services needs to be re-accredited every 3 years and continual training is required to ensure GPs are up to date and can provide an evolving care programme. Patient care pathways are an essential element of any integrated service between primary and secondary care.

Endoscopy training for primary care now has its own website: **www.jets.nhs.uk**. The website provides information on courses, records of personal training and, it is hoped in the near future, will be used for appraisal. There is also a push to encourage more GPs who have gained an additional qualification in gastroenterology to consider becoming a national trainer or assessor of trainers.

Patients' views on GI management

Claire Hunt (King's College London) concluded the meeting by providing insights into how patients feel about the care of their conditions, through the initial results from the soon to be published IMAGE (improving management in gastroenterology) study. This 3-year national project, funded by the Health Foundation and representing a unique collaboration between clinicians, researchers and patient organisations, is addressing four major GI disorders (coeliac disease [CD], IBS, IBD and gastro-oesophageal reflux disease) and is now in its second year.

During the first phase of the project, the patient's view of the management of their conditions within general practice was obtained through a series of focus groups. The findings from 14 focus groups involving 93 patients highlighted that whilst some patients were satisfied with the current care they received, there were a number of areas for improvement. For example, there was often no consistency in follow up appointments and clinical testing. Many patients had no access to a dietician and several CD patients reported poor experiences when collecting prescriptions for gluten-free products. IBD symptoms were often trivialised by GPs, with several patients diagnosed with psychological problems. IBD nurses were reported to be more approachable than consultants and better able to provide advice. Patients at either end of the age spectrum appeared to be treated differently, e.g. one patient at 16 years of age was told they were too young to have IBD. Many patients try alternative therapies and some find these beneficial. However, GPs sometimes came across as dismissive of these approaches.

English language evidence-based guidelines for each GI disorder were reviewed concurrently; over 180 documents were analysed and collated, and the key themes were summarised. The key themes emerging from both the focus groups and guidelines were synthesised and the quality criteria for each GI disorder were agreed at a consensus meeting held earlier this year.

These have been translated into condition-specific computerised clinical decision support system (CDSS), which it is hoped will help GPs care for the four target disorders investigated. Monitoring of the usage of the CDSS over a 12 month period will enable an evaluation of the impact of quality criteria on the quality of care provided to patients with GI disorders.

Laryngop

Laryngopharyngeal reflux (LPR) occurs when reflux reaches the top of the oesophagus, penetrates through the upper oesophageal sphincter and enters the back of the throat and larynx. It is often intermittent and can commonly occur in the upright position. GORD, oesophagitis, heartburn and regurgitation co-present in ~30% of cases. Patients are usually diagnosed by pH monitoring and many present with normal acid clearance.

The causes of reflux are many and varied (Table 1).

Table 1 CAUSES OF REFLUX

- Hiatus hernia
- Diet (fat, ethanol, chocolate)
- Sphincter incompetence
- Dysmotility (primary, secondary)
- Delayed gastric emptying
- Increased intra-abdominal pressure
- Gastric hypersecretion

The cause of LPR-related damage can be due to both acid and pepsin. Pepsin is active up to pH6 and even pH7[1]. Coating of the mucosa of the larynx with activated pepsin can cause significant inflammation and damage. This is associated with a number of laryngeal and pharyngeal manifestations (Table 2).

Table 2 LARYNGEAL AND PHARYNGEAL MANIFESTATIONS

LARYNGEAL

- Posterior laryngitis
- Reinke's oedema
- Laryngeal granuloma/contact ulcer
- Vocal nodules
- Laryngeal papillomatosis
- Functional dysphonia
- Paroxysmal laryngospasm
- Subglottic stenosis
- Dysplasia/carcinoma of larynx

PHARYNGEAL

- Globus pharyngeus
- Chronic sore throat
- Dysphagia
- Pharyngeal pouch

pharyngeal reflux

Pulmonary manifestations are also common (Table 3). LPR is believed to be the commonest cause of chronic cough.

Table 3 PULMONARY MANIFESTATIONS OF LPR

- Chronic cough
- Exacerbation of asthma/COPD
- Bronchiectasis
- Aspiration pneumonia
- Graft rejection after lung transplant
- Particularly associated with WEAK acid reflux

LPR may play a role in some childhood conditions, such as glue ear, sudden infant death syndrome and failure to thrive. In fact, 90% of middle ear samples in patients tested with glue ear had significantly raised levels of pepsin and this was believed to be the main cause of the condition.[2,3]

A number of miscellaneous conditions are also attributable to LPR. These include rhinosinusitis, obstructive sleep apnoea/snoring, dental erosions, halitosis and burning tongue syndrome.

A nine-item reflux symptom index (RSI) to assess the initial severity of LPR symptoms and evaluate patient response to treatment has been validated[4]. The scores can range from 0 to 45. A score of 10 is compatible with a diagnosis of reflux, 20 significant and 30 severe.

Since LPR is largely a disease of lifestyle, this should be addressed. Patients, however, are often reluctant to make lifestyle changes, even though they can have a significant impact on symptoms. Stewart et al[5] showed that lifestyle changes had such a significant effect in a proton pump inhibitor (PPI) trial that the impact of PPI therapy was diminished.

Medical treatment is usually high-dose PPI for 6 to 12 months, with an 80% response rate suggested. However, there are few randomised controlled trials to demonstrate this. The trials that are available only use small numbers of patients and have shown little evidence for this effect.[5,6,7,8,9]

Combination treatment using a PPI and an alginate may be beneficial (Chart 1).[10]

Some alginates inhibit activation of pepsinogen and reduces the binding of pepsin to substrate. Alginates will maximally inhibit pepsin activity at pH 4-5 – the pH that is typical with PPI use. Alginates also reduces cellular damage by bile acids.

To optimise treatment of patients with LPR the following treatment protocol is recommended:[11,12] (Chart 2)

Mark Watson,
ENT Consultant, South Yorkshire SHA

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3 Role of extra-Esophageal reflux in chronic otitis media with effusion. Crapko M, Kerschner JE, syring M, Johnston N. *Laryngoscope* 2007:**117**:1419-1423.
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10 McGlashan JA et al. *Europ Arch Oto-Rhino-Laryngology* 2008 May 28. [Epub ahead of print].
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12 Park W et al. *Laryngoscope* 2005; **115**: 1230-1238.

Chart 1
BENEFITS OF COMBINING PPI WITH A LIQUID ALGINATE SUSPENSION
QLI for Standard esomeprazole patients vs Gaviscon Advance + esomeprazole

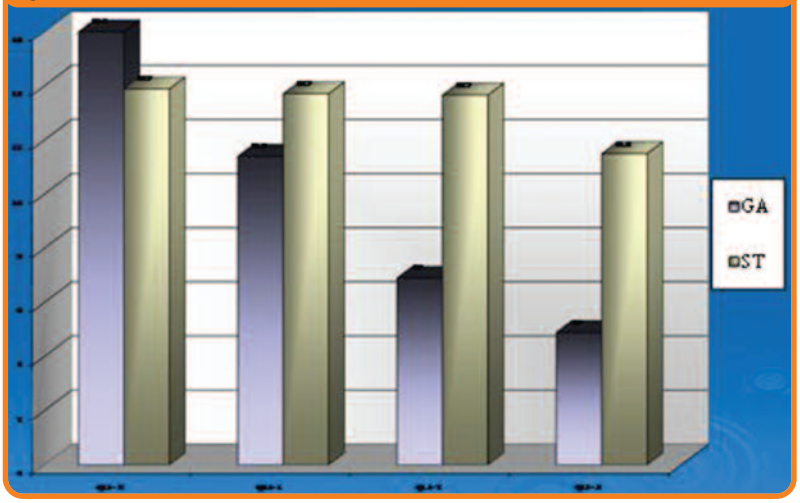
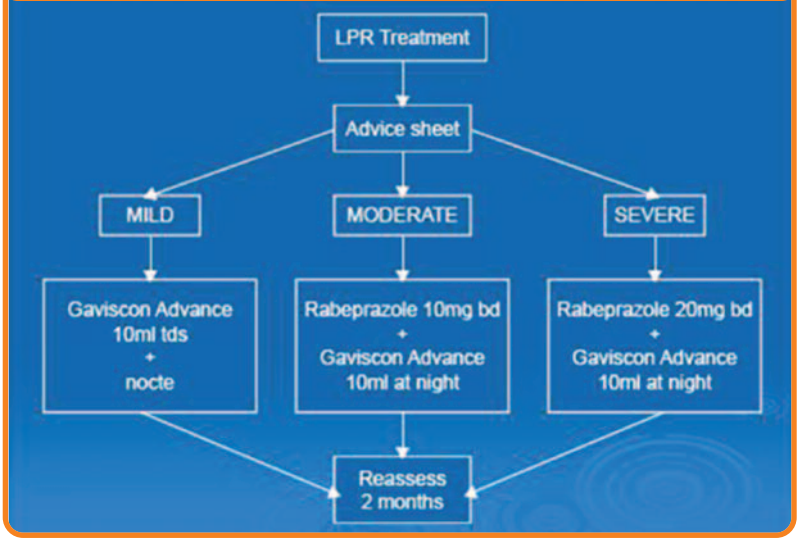


Chart 2
TREATMENT PROTOCOL



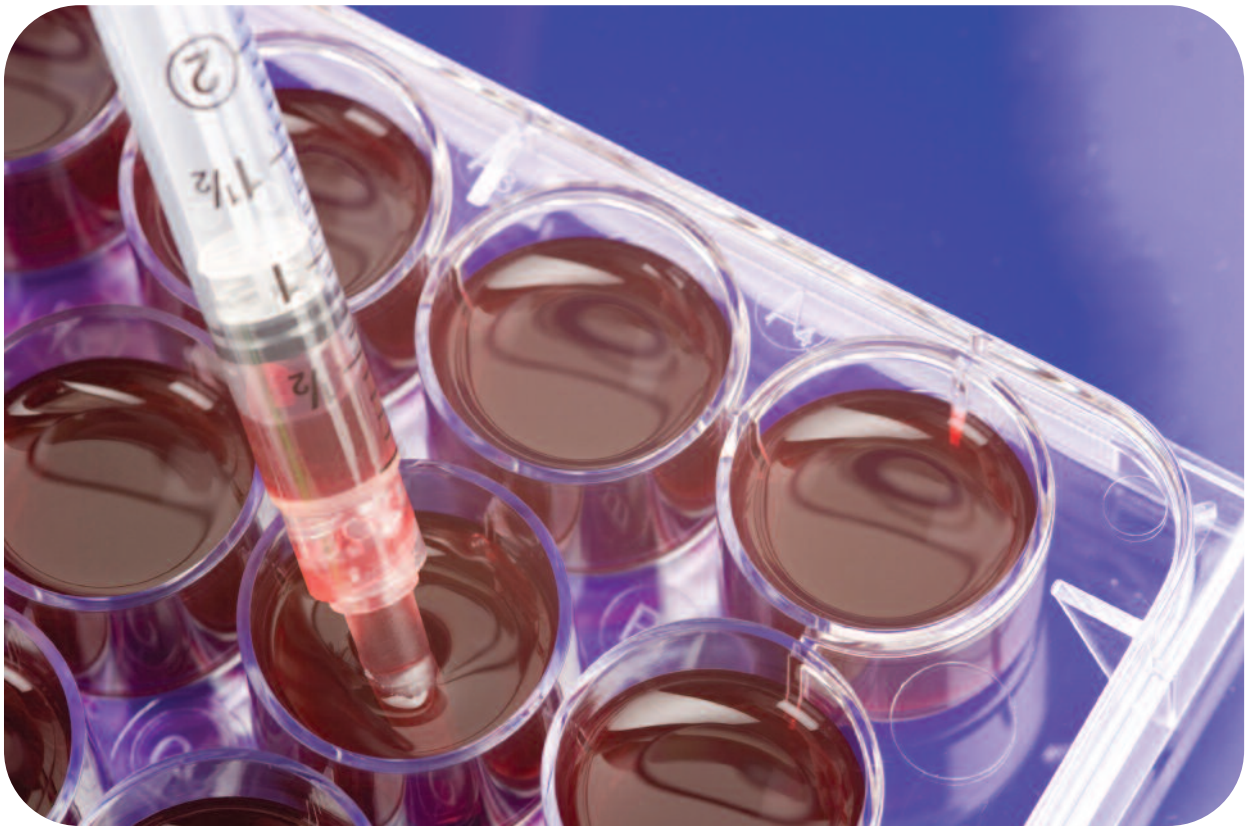
COLON CANCER AND THE STEM CELL NICHE IN THE INTESTINAL CRYPT

Nicholas Wright is not only warden of St Bartholomew's and the London School of Medicine, but one of the UK's most distinguished researchers into cancer pathogenesis. His State-of-the-Art lecture at DDW had a room full of 300 people on the edge of their seats and when I left I thought I knew everything there was to know about stem cells, field cancerisation and crypt fission. I am not sure that I do now.

Wright told a fascinating story of his group's discovery of the importance of stem cells sitting at the bottom of colonic intestinal crypts and mutating monoclonally to replace the 'normal' cells in the crypt, with crypt fission working as the mechanism to spread the monoclonal mutation into other areas of the colon. It appears that the same process may well work for the development of inflammatory changes in inflammatory bowel disease, although in Barrett's oesophagus multiple clonal sites with clonal competition appears an equally appealing theoretical possibility. Wright's group appears to have written the script for research into the development of gastrointestinal cancers for the next five years - they have 'peered into the stem cell niche' and have emerged with a comprehensive theory of clonal spread of mutations in the large bowel and the replacement in the stomach of gastric glands, which are repopulated by a single stem cell line.

However, Wright was candid enough to describe a number of serendipitous histopathological events on the way to this understanding, including the discovery of a male patient, whose colon was preserved in paraffin at St Mark's, who happened not only to have an XY/XO genotype but also had familial adenopolyposis - a coincidence with a probability of 1:140,000,000. Another bizarre syndrome - MRERRF (myoclonic epilepsy with ragged red fibres!!) identified by a neurological colleague in Newcastle also provided a further key genetic substrate for his research. I couldn't help being impressed by the intellectual sweep of this State-of-the-Art lecture, but was also struck by the fact that, alongside the PCR and complex genotyping, accurate and elegant staining, microscopy and histopathological interpretation are still key ingredients of our new understanding of cancer pathogenesis.

Professor Roger Jones



BLUE GENES?

IBS, FUNCTIONAL GUT DISORDERS AND GENETICS

Genetic biomarkers offer important opportunities to improve the precision of diagnosis, to provide insights into pathophysiology, to identify new therapeutic targets and to individualise treatment. Although irritable bowel syndrome (IBS) is not regarded as a strongly familial disorder, it is gradually becoming clear that a number of the pathological mechanisms underlying IBS may have their basis in multiple, common genetic polymorphisms.

Much of this work is being conducted at the University of North Carolina, where Dr William Maixner directs the Centre for Neurosensory Disorders. He emphasised the complexity of the phenotypes involved in chronic pain syndromes, including IBS, temporo-mandibular joint dysfunction (TMJD), pelvic pain etc, pointing out that patients with one of these disorders have an 80% chance of having two of the others. His conceptual model for these disorders, including IBS, includes a combination of high psychological distress with a high state of pain amplification, with underlying genetic abnormalities being triggered by environmental factors such as injury and infection.

His group are now working on candidate gene studies to explain some of the abnormalities found in chronic pain syndromes. They have, for example, identified three haplotypes, reflecting different degrees of pain sensitivity, which predict the development of TMJD and also correlate with greater or lesser pain experiences during the menstrual cycle. Patients with different numbers of copies of the pain and sensitivity haplotypes also turn out to have different psychological profiles.

Dr William Whitehead, a gastroenterologist working closely with Dr Maixner at UNC, emphasised the importance of gene-gene and gene-environment interactions and provided an overview of the large range of genetic abnormalities that are associated with IBS. Rather than using a candidate gene approach, Whitehead's group have recently embarked on genome-wide assays, using a 'Pain Chip', devised by Maixner, which is able to examine 3300 SNPs in 320 genes. The data emerging are analysed using the charmingly-named PLINK computer programme.

In their recent work Whitehead's group have identifed five groups of SNPs relating to inflammation, adrenergic processing, genetic coding for pain sensitivity, motility and secretion and genes associated with affective disorders. Genes coding for cytokines, prostaglandins, cannabinoid receptors and sodium channel regulators, as well as genes which may have some protective effect, are gradually being established as having significant associations with IBS and its sub-types.

Emeran Mayer, working at UCLA, has taken genetics further, linking it with brain imaging. He emphasised the importance of 'deconstructing' common clinical syndromes into 'biological endophenotypes'. In other words we need to get below the syndrome and its constituent symptoms, through neuropsychiatric features and underlying neuromechanisms into the cell signalling involved, the proteins which mediate this and, ultimately, to the genes regulating these proteins. Emeran gave a beautiful illustration of this hierarchical, 'unpeeling' approach by beginning with the observation that IBS patients have an exaggerated startle response, enhanced by previous experiences of pain, which can be shown using functional brain scanning to involve abnormal activity in the brain areas of the amygdala and nucleus caeruleus. It is possible to go on to identify the 'emotional arousal circuitry' involved, to identify abnormal feedback within this circuitry and to identify the role of 5-HTTLPR and SERT activity, at the genetic level. This elegant explanation of vulnerability to an environmental stress has been further strengthened by obtaining corroborative data on structural and functional abnormalities in otherwise normal volunteers.

Finally, Michael Camilleri, head of functional bowel research at the Mayo Clinic, took us on a whistlestop tour of pharmacogenetics and IBS and other gastrointestinal problems. Phase I metabolism is crucially dependent on cytochrome P450 pathways, and accumulating evidence implicates abnormalities in CYP450 2C19 and CYP450 2D6 genes. The 2C19 variant, common in patients in south east Asia, impairs metabolism of protein pump inhibitors, increasing their biological activity, and also affects the metabolism of antidepressants. The 2D6 polymorphism, which has been found to be exaggerated in certain African races, is linked to changes in the metabolism of a number of agents, including the protein pump inhibitors, certain analgesics, antidepressants and antinauseants. Metaboliser status varies significantly in white populations and its clinical relevance is currently being investigated. Other gut-active agents affected by genetic polymorphisms of this kind include the response to the anti-obesity drug sibutramine, clonidine and, interestingly, alosetron - the early alosetron trials have been re-analysed and it appears that a defect in SERT activity and in 5HT re-uptake transporter activity are linked to differential rates of metabolism of alosetron.

This is fascinating, cutting edge stuff, although in the discussion it was difficult to get the panel to commit themselves as to whether they think there are likely to be underlying genetic abnormalities capable of explaining the characteristic features of a range of otherwise unexplained physical symptoms, or whether the genetic research pathway is leading us down a means of better-specifying the clinical syndromes concerned - Meyer's endophenotypes. I think that Maixner's view is that we are likely to find more similarities than differences across the spectrum of medically unexplained symptoms while Camilleri, whose work has largely focused on peripheral (cf. brain) abnormalities in the functional bowel disorders, was more persuaded that this research will sharpen our definition of currently heterogeneous and poorly-defined syndromes.

Professor Roger Jones

JOURNAL OF THE PRIMARY CARE SOCIETY FOR GASTROENTEROLOGY

Event Diary

21 October 2008
Yakult UK Symposium – Probiotic relevance. Putting theory into practice
76 Portland Place,
London W1B 1NT
Contact: 020 8842 7600
yakultsymposium@yakult.co.uk
www.yakultsymposium.co.uk

7 November 2008
Gastroenterology Symposium
Royal College of Physicians of Edinburgh
Contact: c.gray@rcpe.ac.uk
www.rcpe.ac.uk/education/events/gastro-nov-08.php

26 November 2008
Endoscopy Meeting
East Midlands Conference Centre,
Nottingham
Contact:
secretariat@pcsg.org.uk

23-26 March 2009
BSG Annual Meeting
Scottish Exhibition & Conference Centre,
Glasgow
Contact: 020 7935 3150 for information

ISSN 1752-8763

KEY FACTS 1 Inflammatory Bowel Disease

Topic/Definition
- **IBD:** Inflammatory Bowel Disease

Prevalence/Incidence
- **Ulcerative Colitis** (UC)Prevalence 100-200/100,000
- **Crohn's Disease** (CD) 50-100/100,000
- Peak Incidence 10-40 years but can occur at any age.

Common symptoms
- **UC.** Bloody diarrhoea, tenesmus, abdominal pain, urgency
- **CD.** Abdominal pain, diarrhoea and weight loss. Malaise, anorexia and weight loss also common and occur more often in CD than UC. Intestinal obstruction common.
- Both Crohn's Colitis and Ulcerative Colitis increase risk of colonic cancer

Investigations
- If suspected, fbc, esr, crp, full profile, faecal cultures including tests for C. Difficile.
- If available in practice, rigid sigmoidoscopy with biopsy of inflammation can be very useful. Even without this a pr exam is mandatory.
- Refer urgently and don't delay with radiological investigations.
- Do not start 5ASA treatment before referral.

Do and don'ts of treatment
- Encourage non smoking especially in CD.
- Education is very important. Encourage membership of NACC and liaison with gastrointestinal nurse.
- Long term illness so give 3-6 monthly scripts.
- Use brand named 5ASAs rather than generic. Changes in type can result in relapses.
- Milder relapses of UC can be treated with increased dose of 5ASAs, oral steroids (high dose with gradual reduction) and in the case of distal colitis rectal steroid preparations.

Additional
- Complex and distressing condition at times so GP essential as a point of continuous care and support, facilitating rapid referral to clinic during exacerbation.
- May need help with social services and insurance companies.

Review articles
Guidelines for the management of inflammatory bowel disease in adults
M J Carter[1], A J Lobo[2] and S P L Travis[3] on behalf of the IBD Section of the British Society of Gastroenterology
GUT 2004:v1-v16

Dr John O'Malley

KEY FACTS 2 Rectal Bleeding in Adults

Topic/ Definition
RECTAL BLEEDING: Blood found on wiping, coating the stools or mixed in the stools

Prevalence/ Incidence
The prevalence of reported rectal bleeding in the previous twelve months is approximately 1 in 6 in the UK adult population[1]

Common symptoms
- **Bright red** Clearly separate from stools indicates anal origin as in anal fissure, haemorrhoids, anal carcinoma. Coating of surface of stools can indicate anal or more proximal origin as in rectal or sigmoid colon polyp, cancer or diverticular disease
- **Dark red** Usually mixed with stools and indicates higher lesion as in left or even transverse colon pathology as in cancer or inflammatory bowel disease
- **Black or plum coloured (malena)** Indicates bleeding from upper GI tract and usually requires immediate admission
- **Occult bleeding** May be present with iron deficiency anaemia

Investigations
- Ano-rectal examination, proctoscopy and rigid sigmoidoscopy
- Flexible sigmoidoscopy and or colonoscopy when stool is coated or mixed with blood and in all cases over the age of 40[2]. Faeces for Occult blood (FOB*[3]) when iron deficiency anaemia present. Check Full Blood count, ferritin levels in all cases.

Do and don'ts of treatment
- **DO** take careful history (including family history) and examine ano-rectal area in all patients
- **DO** offer specialist access to all patients over the age of 40 presenting with rectal bleeding[3]
- **DON'T** delay diagnosis or ignore rectal bleeding symptom at any age

Review articles

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Produced by the Primary Care Society for Gastroenterology, Gable House, 40 High Street, Rickmansworth, Herts WD3 1ER
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