

European Crohn's & Colitis Organisation Congress

Conference Review

Making Education Easy

ECCO 2011, Dublin, Ireland

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About the Reviewer



Dr David Rowbotham

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A graduate of the University of Newcastle upon Tyne Medical School, David trained in both Gastroenterology and Hepatology in the UK at international centres of excellence in London (Kings College Hospital) and Leeds. He came to New Zealand in 1999 as Specialist Gastroenterologist at Auckland City Hospital and worked there for 5 years until 2004. During this time, he set up the first NZ service for both push enteroscopy and wireless capsule endoscopy at Auckland City Hospital, both of which have now taken hold in the country as a whole. In 2004, David travelled to the UK with his wife and worked for several years as a Consultant Gastroenterologist at a busy general hospital in South East London. In 2008, David took over the leadership role as Clinical Director for the Department of Gastroenterology & Hepatology at Auckland City Hospital.

David is also of the firm (but hopefully misguided) belief that England will win this year's Rugby World Cup.

Welcome to our review of the 6th Congress of the European Crohn's and Colitis Organisation (ECCO).

The ECCO Annual Scientific Meeting is fast becoming one of the premiere forums for the dissemination of inflammatory bowel disease (IBD) knowledge worldwide. The ECCO is taking a worldwide lead in the promotion of IBD science and clinical excellence through this meeting, and others, including task forces that examine specific issues and produce comprehensive guidelines. Dr David Rowbotham, an Auckland-based specialist in gastroenterology and hepatology, attended the ECCO 2011 congress held in Dublin, Ireland in late February. He has selected 12 of the most significant presentations and provided independent commentary for this review.

Next year, the congress will be held in Barcelona, Spain and is to be recommended for those who wish to attend a comprehensive IBD conference.

Kind regards,

Dr Chris Tofield

Medical Advisor, Research Review

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Gene expression profiling in CD8 T cells predicts disease course in Crohn's disease and ulcerative colitis

Authors: Lee JC et al

Summary: This UK study used microarray-based whole genome expression profiling of CD8 T cells from the blood of 60 patients with active Crohn's disease (CD) or ulcerative colitis (UC). These researchers had previously performed such an assessment in CD8 T cells in two other autoimmune disorders and had discovered a gene expression signature that correlates with disease course. In the current study, gene profiling was undertaken prior to therapy with a conventional "step-up" approach by clinicians who were blinded to the gene expression results. Patients were followed up for up to 700 days. Gene expression analysis revealed two distinct subgroups in each of the disease cohorts (CD and UC), and a comparison of the genes that were differently expressed in each cohort revealed that they had significant overlap ($p < 0.001$). Patients from both disease cohorts were therefore grouped together for the analysis into subgroups IBD1 and IBD2. Patients in IBD1 experienced a more aggressive disease course than those in IBD2, with a significantly ($p = 0.0025$) higher percentage of patients in the IBD1 group requiring an escalation in maintenance therapy, including the introduction of immunomodulators or surgery during the follow-up period. The study authors say that this stratification was superior to other previously described methods of predicting outcomes in CD or UC.

Comment: IBD can be a minor inconvenience or a devastating and life-threatening condition. If only we could predict the likely disease course at time of diagnosis, we could tailor therapy to suit. Well perhaps we can? This study was simple in conception. By interrogating gene expression profiles in peripheral blood CD8 T lymphocytes, and akin to other immune-mediated conditions (systemic lupus erythematosus and vasculitis), two distinct subgroups of IBD patients were identified with very different disease behaviours. This sub grouping was not possible using standard clinical or laboratory features. This is exciting work. Identification of aggressive disease behaviour and worse clinical outcome allows early aggressive intervention ("top down" therapy) in those most likely to benefit. Personalised therapy may not be so far away after all!

Oral presentation: #2

Available from: <http://tinyurl.com/4uuv6en>

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Induction and maintenance adalimumab therapy for the treatment of moderate to severe Crohn's disease in children

Authors: Hyams et al

Summary: The efficacy and safety of two adalimumab dosage regimens for the induction and maintenance of clinical remission in paediatric patients with moderate-to-severe CD was compared in this multicentre, randomised, double-blind study. A total of 192 patients aged 6 to 17 years with a Paediatric CD Activity Index (PCDAI) >30 at baseline (median PCDAI 40), and who had failed or been intolerant to oral corticosteroids or immunosuppressants, received induction therapy with open-label adalimumab, dosed according to baseline bodyweight (≥ 40 kg: 160mg at week 0, 80mg at week 2; <40kg: 80 mg at week 0, 40mg at week 2). Patients were stratified at week 4 according to clinical response (PCDAI decrease of ≥ 15 points from baseline) and prior infliximab (IFX) exposure, and randomised in a 1:1 fashion to double-blind maintenance therapy with either high- (n = 93) or low-dose (n = 95) adalimumab. The agent was dosed according to bodyweight at week 4 (high dose: ≥ 40 kg = 40mg every other week [eow], <40kg = 20mg eow; low-dose: ≥ 40 kg = 20mg eow, <40kg = 10mg eow), and administered for 48 weeks. An increase to blinded weekly dose adalimumab was permitted for flare or non-response after week 12. A total of 66 high-dose and 58 low-dose recipients completed the study. At week 26, clinical remission (PCDAI ≤ 10), the primary endpoint, was achieved by a higher proportion of high-dose recipients than low-dose recipients (39% vs 28%), but this comparison only reached significance in the group of patients who were IFX-naïve (57% vs 35%; $p = 0.026$). In fact, comparing the clinical remission rate of high- and low-dose adalimumab, the greatest efficacy with high-dose adalimumab was seen in the group of IFX-naïve patients who had responded to induction therapy (63% vs 38%; $p = 0.016$). With regard to safety, no new safety signals were detected.

Comment: In NZ adalimumab is funded for certain indications in CD, but there is no funded option for paediatric patients. This study looked at weight-based open-label adalimumab in paediatric patients with moderate-severe CD (PCDAI >30) who had failed or been intolerant to steroids, immunomodulators, and even IFX. Adalimumab induced clinical remission at week 26 in up to 39% of patients given the higher dose. Greatest efficacy was seen in IFX-naïve patients, but there was still up to 20% remission rate achieved in IFX-experienced patients. Adverse event rates mirrored those seen in adult patients. These results provide more evidence to support the campaign to see adalimumab funding extended to include paediatric patients with CD.

Oral presentation: #7

Available from: <http://tinyurl.com/4kvypp2>

Ciclosporin versus infliximab in acute severe ulcerative colitis refractory to intravenous steroids: a randomized study

Authors: Laharie D et al

Summary: This European randomised controlled trial (RCT) compared cyclosporin (CyA) to IFX for IV steroid-resistant, acute, severe UC. Patients with a Lichtiger score >10 after at least 5 days of IV methylprednisolone ≥ 0.8 mg/kg/day were randomised to IV CyA 2 mg/kg/day for 1 week, then oral CyA for 98 days, or IFX 5 mg/kg at weeks 0, 2 and 6. Patients exhibiting a clinical response (Lichtiger score <10 with a decrease of ≥ 3 points compared with baseline) at day 7 received azathioprine (AZA) 2.5 mg/kg/day and had their steroids decreased according to a fixed regimen. The primary endpoint was the rate of treatment failure defined by any of the following: absence of clinical response at day 7; absence of remission (Mayo score ≤ 2 without any subscore >1) without steroids at day 98; relapse between day 7 and day 98 (sustained increase of the Lichtiger score leading to a new treatment); severe adverse event leading to treatment interruption; colectomy; fatality. An interim analysis after 30 patients had been treated with IFX did not show any serious adverse events necessitating study cessation.

Comment: What to do in IV steroid-resistant UC? IV CyA has been shown to be an effective three month bridge to allow introduction of an immunomodulator. More recently IFX also has been shown to be an effective rescue therapy in this setting. This multi-centre RCT pits these two therapies head to head. Both agents achieve short-term response in 80% of patients with no difference in treatment failure rates and no difference in colectomy rates. So IFX gives another option in this setting, but is unlikely to replace CyA in routine practice in NZ as it is markedly more expensive.

Oral presentation: #12

Available from: <http://tinyurl.com/4fchhud>

Infliximab, azathioprine, or infliximab + azathioprine for treatment of moderate to severe ulcerative colitis: The UC SUCCESS trial

Authors: Panaccione R et al

Summary: The efficacy of IFX/AZA, IFX alone and AZA alone was compared in this 16-week, randomised, double-blind, controlled trial involving 239 biologic-naïve patients with moderate-to-severe UC (Mayo score ≥ 6) who were failing corticosteroids and who had either stopped AZA for ≥ 3 months, or who were AZA naïve. Patients received IFX 5 mg/kg plus AZA 2.5 mg/kg, IFX 5 mg/kg plus placebo, or AZA 2.5 mg/kg plus placebo. In the AZA arm of the study, non-responders at week 8 (Mayo score reduction <1 point) were eligible for IFX 5 mg/kg at weeks 8, 10 and 14. Analysis was performed on 231 patients (median age 37 years). At week 16, the proportion of patients experiencing steroid-free remission (total Mayo score ≤ 2) was significantly higher in the IFX/AZA arm compared with the IFX or AZA monotherapy arms (40% vs 22% and 24%, respectively; $p < 0.05$). Clinical response (decrease in Mayo score of ≥ 3 points and at least 30% lower than baseline Mayo score) and mucosal healing (Mayo endoscopy subscore of 0 or 1) at week 16 was evident in significantly ($p < 0.05$) more IFX/AZA and IFX recipients compared with AZA recipients (IFX/AZA 77% and 63%; IFX 69% and 55%; AZA 50% and 37%, respectively).

Comment: SONIC showed that dual therapy with IFX and AZA is most efficacious in biologic-naïve CD. But what about UC? This multicentre study (UC SUCCESS) looks at the same question in moderate-to-severe UC (combination IFX/AZA vs IFX alone vs AZA alone), although the duration of the study is much shorter at only 16 weeks. Combination IFX/AZA therapy was superior in inducing steroid-free remission compared to either IFX or AZA monotherapy. IFX monotherapy achieved greater clinical response and mucosal healing compared to AZA, but this result may have as much to do with the short study timeframe as anything else. Longer follow-up data from these patients at 52 weeks (*a la* SONIC) will be interesting.

Oral presentation: #13

Available from: <http://tinyurl.com/4p579bf>

Risk of squamous and basal cell carcinomas in patients with inflammatory bowel disorders exposed to thiopurines: the Cesame National Cohort Study

Authors: Peyrin-Biroulet L et al

Summary: The potential role of thiopurines in promoting skin carcinomas was assessed in this prospective cohort study involving 19 486 patients with IBD (39.7% UC, 60.3% CD) who were enrolled in a nationwide French cohort. Median follow-up was 35 months (interquartile range 29-40). At baseline, 10 810 (55.5%) patients were thiopurine-naïve, 5867 (30.1%) patients were receiving thiopurines and 2809 (14.4%) patients had discontinued such agents. In total, 20 basal and 12 squamous cell carcinomas were recorded. The incidence of skin carcinomas before the age of 50 years in past and present thiopurine recipients was 0.50 and 0.59 per 10 000 patient-years. In those who were thiopurine-naïve, the incidence was nil. Analysis in patients aged 50-65 years, and over 65 years, showed similar findings. Independent risk factors for skin carcinomas were ongoing treatment with thiopurines (OR 7.3; 95% CI 2.7-19.8), past exposure to thiopurines (OR 5.1; 95% CI 1.7-15.3) and age (OR 1.1; 95% CI 1.04-1.09). When basal and squamous cell carcinomas were considered separately, the findings were similar.

Comment: Here in NZ, a lack of ozone means that we have been preaching about sun-protection for years, particularly to our patients receiving thiopurines. Seems like the Europeans are finally getting the message too. This large French cohort study confirmed significant increase in the incidence of non-melanomatous skin cancer. This held true even if the thiopurine had been discontinued, and was certainly present by the age of 50. Hence, sun-smart advice is relevant and important for all our patients whatever their age, and even if they no longer take thiopurines.

Oral presentation: #15

Available from: <http://tinyurl.com/4nuwgmd>



Long term evolution and impact of immunomodulator co-treatment and withdrawal on infliximab trough levels in 223 patients with Crohn's disease

Authors: Drobne D et al

Summary: This Belgian study examined the influence of immunomodulator (IMM) withdrawal on clinical outcome and IFX trough levels in 223 patients receiving IFX maintenance therapy for CD. The study aimed to determine the usefulness of IFX trough levels when deciding to withdraw IMM therapy. A total of 158 patients were receiving combination therapy with IFX and IMMs while 65 received IFX monotherapy. IMM therapy was discontinued in 117/158 patients after ≥ 6 months (median 13 months) of combination therapy. A mean of 4.7 IFX trough level measurements were undertaken for each patient and for those patients receiving combination therapy, measurements were taken before, at the time of and after withdrawal of IMMs. Significantly higher IFX trough levels overall were seen in those patients treated with combination therapy compared to those receiving IFX alone (3.4 $\mu\text{g/mL}$ vs 2.5 $\mu\text{g/mL}$; $p < 0.001$). IFX trough and CRP levels remained stable in the majority of patients after IMM withdrawal. However, when adjustment was made for dose optimisation due to flares, IFX trough levels were found to decrease in a proportion of patients, necessitating dose increase or interval shortenings. Undetectable IFX trough levels were seen in 16% of patients shortly after IMM withdrawal, however, 50% of those patients had undetectable IFX trough levels already at the time of withdrawal. The majority (90%) of those patients experienced a flare, while significantly ($p < 0.001$) fewer patients (30%) with detectable IFX trough levels experienced such an event. Generally, patients who lost response to IFX more often had low or undetectable IFX trough levels (72% and 49%, respectively) compared with patients who did not (48% and 19%, respectively). The authors concluded that measuring IFX trough levels before stopping IMM therapy appears to be a reasonable option to aid in identifying those who are more likely to experience a disease flare.

Comment: Combination therapy (anti-TNF and IMM) works best in CD, but it remains unclear in which patients we should stop therapy, when should we stop, and which agent we should stop? This Belgian study looked specifically at IMM withdrawal in patients continuing on maintenance IFX. Belgium is far removed from NZ in terms of management of CD ("dose optimisation" is not a familiar phrase over here). Nevertheless it is interesting to see that patients with undetectable IFX trough levels at discontinuation of IMM therapy are the most likely to flare. We don't know whether these patients would have flared anyway even without discontinuation of IMMs; so whether this study helps us in the long term remains to be seen.

Oral presentation: #17

Available from: <http://tinyurl.com/4ufqg2f>

Risk factors for inflammatory bowel disease associated colorectal carcinoma

Authors: Lutgens M

Summary: This retrospective case-control study aimed to identify risk factors for colorectal cancer (CRC) in patients with IBD. Using a nationwide pathology database, 118 reported cases of IBD-associated CRC in the Netherlands between 1990 and 2006 were identified. A total of 206 matched IBD controls were also identified from the database. The mean follow up was 4059 days. Independent risk factors for developing CRC in patients with CD were disease extent $>50\%$ of the colon (HR 4.7; 95% CI 1.6-14.1) and 5-ASA medication >3 months (HR 0.4; 95% CI 0.2-0.9). Independent risk factors for developing CRC in patients with UC were disease extent $>50\%$ of the colon (HR 3.0; 95% CI 1.4-6.5), colonic stenosis (HR 5.2; 95% CI 2.6-10.3), pseudopolyps (HR 2.5; 95% CI 1.2-5.2) and 5-ASA medication >3 months (HR 0.4; 95% CI 0.2-0.8). Primary sclerosing cholangitis was associated with an increased, but non-significant risk (CD: HR 1.8; 95% CI 0.2-14.8; UC: HR 1.7; 0.6-4.3).

Comment: IBD of >10 -15 years duration is associated with an increased risk of GI malignancy, particularly colorectal cancer (CRC). Expert opinion on the value of colonoscopic surveillance for this population is dichotomised, so further clues to help refine risk stratification for CRC in IBD would be helpful. The results from this Dutch study would support the theory that it is "inflammatory burden" that is associated with increased CRC risk, not simply duration of disease. 5-ASAs again appear protective. Interestingly there was no increased CRC risk from primary sclerosing cholangitis, presumably simply due to lack of power. An overhaul and rationalisation of international guidelines for the frequency of surveillance colonoscopy in IBD is overdue.

Oral presentation: #20

Available from: <http://tinyurl.com/4bh9v4w>

Normalization of mucosal TNF-alpha as a criterion when to stop treatment with anti TNF in UC patients? A preliminary report

Authors: Olsen T

Summary: The potential for colonic mucosal TNF- α mRNA levels as a predictive marker for relapse after successful induction therapy with IFX (0, 2 and 6 weeks) in 18 patients with moderate-to-severe UC was assessed in this study. Patients had colon biopsies taken and real-time PCR was used to assess mucosal TNF- α mRNA levels. Patients used concomitant 5-ASA and AZA, and were monitored regularly for relapse, defined using endoscopic and clinical disease activity indices. During 12 months of follow up, 10 patients (62%) experienced a relapse with a mean mucosal TNF- α mRNA level of 17 710 copies/ μg mRNA. The corresponding level in the group of patients who were free from relapse was 8811 copies/ μg mRNA, but the difference between the two groups did not reach significance.

Comment: This Norwegian group have reported previously that TNF- α mRNA in colonic mucosa in UC correlates proportionately with the degree of inflammation, and inversely with the clinical outcome after IFX induction therapy. Could these principals guide us as to when we could safely withdraw IFX in UC patients? Colonic mucosal TNF- α mRNA levels tended to be higher in those who clinically relapsed after an initial clinical and endoscopic remission compared to those who did not relapse, but this trend failed to reach significance. Nevertheless this is an interesting study that demands further research in terms of numbers of patients, and to see if these findings are also applicable to CD.

Poster presentation: #P029

Available from: <http://tinyurl.com/4lrylmc>

Pulmonary function tests in patients with ulcerative colitis

Authors: Lazebnik L et al

Summary: These Russian researchers explored the relationship between UC and pulmonary dysfunction in their study involving 164 patients with UC (median age 44.6 years) and 60 healthy matched controls. Among patients with UC, 73 were smokers or ex-smokers. Lung function abnormalities were identified in a significantly higher proportion of patients with UC than healthy controls (43.9% vs 8.3% ($p < 0.001$)). Among patients with UC exhibiting pulmonary dysfunction, 57 had a predominantly obstructive pattern of small airways, five had a restrictive pattern and 10 had a restrictive-obstructive pattern. Pulmonary dysfunction was found more frequently in patients with a >10 -year history of UC ($n = 28$), compared with those with a <10 -year history of UC ($n = 98$); 89.3% vs 31.6% ($p < 0.005$). Patients with distal colitis ($n = 47$) had a significantly ($p < 0.05$) lower incidence of lung function abnormalities (25.5%) than those with subtotal ($n = 53$) and total ($n = 62$) colitis (50.9% and 51.6% respectively). Altered pulmonary function was observed in 18/25 (72%) patients with a chronic continuous form of UC, in 16/38 (42.1%) patients with a first attack of UC ($p < 0.05$) and in 38/101 (37.6%) patients with a chronic relapsing form of UC ($p < 0.005$). Smokers and ex-smokers exhibited an insignificantly higher rate of pulmonary function disorders compared with non-smokers (50.7% vs 38.5%; NS).

Comment: Extraintestinal manifestations occur in up to 40% of IBD patients, but bronchopulmonary manifestations related to IBD are not top of most clinicians' concerns. This Russian study looked at patients with UC and compared them to well matched healthy volunteers, including smoking history. Abnormal pulmonary function was significantly more common in the UC cohort with, astonishingly, nearly half of these patients showing abnormal spirometry (obstructive abnormalities more common than restrictive). These findings were independent of current or previous smoking history, and were present even in newly diagnosed patients (excluding a drug side-effect). So should pulmonary function testing be part of the standard initial assessment of our newly diagnosed UC patients? Should we be checking routine regular spirometry? It is non-invasive, safe, cheap, and easy ... so why not?

Poster presentation: #P056

Available from: <http://tinyurl.com/463148u>



Current European practice diagnosis and treatment of IBD-associated anaemia

Authors: Stein J et al

Summary: Iron deficiency is a major cause of anaemia in patients with IBD. Current (2007) international guidelines recommend IV iron as the preferred route of administration. In this study, current European practice for the diagnosis and treatment of iron-deficiency anaemia in IBD was evaluated. Between June and September 2009, a total of 236 gastroenterologists from the UK, France, Spain, Germany and Switzerland completed questionnaires on patient demographics, initial haemoglobin levels and iron parameters, and reported therapies for the five most recent IBD patients treated for anaemia within 6 months. Data was reported on a total of 1173 patients with IBD-associated anaemia. At diagnosis, a median of 55% (range across countries 26-63%) of patients presented with severe anaemia ($Hb \leq 10$ g/dL) and a median of 12% (range 2%-15%) with an Hb level ≤ 8 g/dL. Absolute iron deficiency (ferritin ≤ 30 ng/mL) was found in 81% of patients (range 66%-89%). A median of 65% of patients (range 53%-78%) received iron, delivered mainly as monotherapy. In Switzerland, 56% of patients treated with iron received the IV form and only 6% received a blood transfusion for anaemia. However, in the other countries investigated only 18%-30% of those who received iron received it intravenously and 9%-19% underwent blood transfusions.

Comment: With all the excitement surrounding "deep remission", biologicals, and risks of malignancy from both the disease and the treatments in IBD, it is easy to forget the basics ... the simple stuff that can make just as big a difference to our patients' quality of life. International guidelines from 2007 have advocated the use of IV iron for the management of IBD-associated anaemia. This study demonstrates that, in Europe at least, adherence to these recommendations is poor (except for the diligent Swiss!). Such an audit has not been performed in NZ, but I suspect the results would be similar. So what prevents us adhering to such guidelines, especially since we know that tolerance of (and hence adherence to) oral iron therapy is poor? Answers on a postcard please ...

Poster presentation: #P080

Available from: <http://tinyurl.com/4ez7q52>

Anti-TNF therapy and pregnancy in inflammatory bowel disease: a prospective cohort study from the GETAID

Authors: Seirafi M et al

Summary: The impact of anti-TNF therapy on foetal development and pregnancy outcome in IBD was assessed in this prospective ongoing European study. At the time of analysis, there had been 47 live births from 49 pregnancies in patients who were receiving anti-TNF therapy (IFX, adalimumab or certolizumab) during pregnancy, or who had become pregnant within 3 months of the withdrawal of such therapy. A small proportion of patients had received concomitant AZA. Anti-TNFs were continued until delivery in 14/49 (29%) patients and preventively interrupted in 31/49 (63%) patients at the end of the 6th gestational month. A total of 33/49 (67%) pregnancies were uneventful, while 16/49 (32%) pregnancies were associated with adverse events: 3 (6%) deaths (2 deaths in utero, 1 extreme prematurity), 8 (17%) premature deliveries (<37 weeks gestation) of children born alive, 1 HELLP syndrome, 2 gestational cholestasis, 1 colectomy for severe UC, 1 maternal infection. In the normal pregnancy group, 27% were experiencing a relapse of their UC at the time of conception, vs 31% among the complicated pregnancies group. Of the 46 infants alive, 7 (15%) presented 10 neonatal complications; 6 (13%) foetal hypotrophy (birth weight <2500 g), 3 respiratory distress syndromes and 1 foetal infection. The study authors say that while their results indicate that one third of pregnancies exposed to anti-TNF are complicated, the numbers do not differ from those reported in the general IBD population, and indicate an absence of excess risk linked to anti-TNF therapy.

Comment: What should we recommend to our female IBD patients regarding the safety of biological therapy in pregnancy? Data are limited but there appears to be no specific adverse outcome to either mother or baby. After all, it is often the use and success of these agents that allow the possibility of conception and pregnancy in the first place. This abstract from the prolific French IBD research group (the GETAID) reports on a prospective cohort study of pregnancies that occurred during anti-TNF therapy or within 3 months of treatment withdrawal. Thus far, at least, anti-TNF agents appear to carry no excess risk to pregnancy outcome and foetal viability over and above IBD itself.

Poster presentation: #P140

Available from: <http://tinyurl.com/4ofsdm2>

Evaluation of efficacy and safety of induction therapy with curcumin enema in ulcerative colitis patients with mild to moderate proctitis

Authors: Ahuja V et al

Summary: This randomised, double-blind, single centre pilot trial involving 43 patients with UC and mild or moderate proctitis (UCDAI score > 3) compared the efficacy and safety of curcumin enema plus oral 5-ASA (n = 22) with that of placebo enema plus oral 5-ASA. The baseline mean UCDAI scores were 6.05 and 6.1 in the curcumin and placebo groups. Both treatments were well tolerated and a total of 29 patients, 14 curcumin recipients and 15 placebo recipients, completed therapy. The primary end-point of a reduction in UCDAI score of >3 points at week 8 was achieved by significantly more curcumin recipients than placebo recipients (92.8% vs 53.3%; p = 0.017). Remission rates at week 4 were 64.2% for curcumin recipients vs 20% for placebo recipients (p = 0.016), and at 8 weeks were 78.5% vs 40%, respectively (p = 0.03).

Comment: Curcumin, a natural compound used as a food additive, has been shown to have anti-inflammatory and antioxidant properties in cell culture and animal studies. In open label studies in UC and CD, curcumin enemas were associated with improved clinical and laboratory parameters. This study demonstrates that, in UC patients with proctitis at least, curcumin enemas with oral 5-ASA achieved significantly higher clinical response and remission rates compared to oral 5-ASA and placebo enemas. There have been no studies to date comparing curcumin enemas with topical 5-ASA, but these results are encouraging. Who said all food additives are bad?

Poster presentation: #P289

Available from: <http://tinyurl.com/4qpedy3>