

For personal use only. Not to be reproduced without permission of the editor
(permissions@pharmj.org.uk)

Inflammatory bowel disease

— drug treatment and its implications

By Anja St Clair Jones, MRPharmS

Most of the currently available drug treatments for inflammatory bowel diseases aim to reduce the inflammation in the intestine. The second part of this month's special feature gives information about the preparations used, including details about drug delivery



Reducing intestinal inflammation is the aim of most of the drug treatments for inflammatory bowel diseases

Pharmacotherapy is the foundation of inflammatory bowel diseases management. Most current treatments aim to reduce the chronic inflammation in the intestinal mucosa but cannot cure IBD, since the exact cause of the conditions is unknown.

Therapeutic management will depend on the anatomical location of the disease and therapies need to be tailored to suit patients' needs and preferences. Obtaining an accurate diagnosis is essential, because this will greatly influence disease management. Individualisation of drug regimens is crucial to promote patient adherence and to achieve the best long-term outcome. The main drug treatments currently used are:

- Aminosalicylates
- Corticosteroids
- Thiopurines
- Methotrexate
- Ciclosporin
- Anti-tumour necrosis factor (TNF) therapy

Further details about these agents are set out later. It should be noted that the need for

Anja St Clair Jones is surgical directorate pharmacist at Brighton and Sussex Universities Hospitals NHS Trust

adjunctive therapies, including pain management agents, antidiarrhoeals and nutritional supplements (such as iron and vitamin B₁₂) will need to be monitored. Opiates should be used sparingly to reduce the likelihood of the complication of a toxic megacolon (a condition where loss of muscle tone causes the colon to dilate and, in serious cases, to perforate). Non-steroidal anti-inflammatory drugs should be avoided because they might increase the production of pro-inflammatory leukotrienes by inhibiting cyclo-oxygenase enzymes and consequently promoting the lipooxygenase pathways. Antibiotics such as metronidazole have been used in IBD (based on the theory that flare-ups can be triggered by an infection) but there is no strong scientific evidence to support this therapy. However, clinical experience has found that metronidazole and/or ciprofloxacin can be useful, especially in patients who have perianal fistulae.

Drug delivery is a particularly important concept when treating IBD. When using oral therapy, minimal systemic absorption and maximal intestinal wall drug levels are desired — contrary to that required in most other conditions. Several delivery strategies have been used to achieve this, including the chemical modification of drug molecules, the use of controlled- and delayed-release formulations and the use of bioadhesive particles.

Topical agents are usually added to oral treatments because the former are unlikely to be effective alone. In general, drugs delivered as suppositories only reach the rectum up to the sigmoid flexure, whereas those delivered as a foam can reach the sigmoid colon and those delivered as an enema can reach as far as the splenic flexure and, in some patients, the ascending colon.

— Aminosalicylates

The 5-aminosalicylic acid (5-ASA) group of drugs are the treatment of choice for mild to moderate IBD.

Sulfasalazine has been used successfully for the treatment of ulcerative colitis and Crohn's disease. The azobond of sulfasalazine is cleaved by bacteria present in the colon into sulfapyridine and mesalazine. Early studies demonstrated that the 5-ASA moiety, mesalazine, is the active ingredient, whereas the sulfapyridine group acts as an inert carrier. The high incidence of adverse drug reactions (ADRs) was attributed to the sulfapyridine group, which led to the development of the 5-ASA-based compounds used today in the treatment of IBD.

The mode of action of 5-ASA is unclear, but a local effect on epithelial cells by a variety of mechanisms is proposed, including the modulation of inflammation mediators such as products of lipo-oxygenase and cyclo-

oxygenase metabolism (ie, leukotrienes, hydroxyecosatetraenoic acids and prostaglandins), platelet-activating factors, and cytokinines (ie, TNFs and interleukins).

The main role of 5-ASA is in maintaining remission. The dose given depends on the preparation used which, in turn, depends on the presentation of the disease. Unmodified mesalazine is rapidly absorbed from the small bowel. Therefore formulations are needed which deliver the active drug to the affected site topically and minimise absorption to increase tolerability. The preparations currently available can be classified into three main groups:

- Those that are resin coated and release drug pH-dependently
- Those that release drug in a time-controlled way
- Those that deliver drug by carrier molecules and are dependent on intestinal bacteria to cleave the azobond

Efficacy seems to depend more on adherence than the frequency of administration but preparations should be matched to the site of the disease (see Panel 1). Effective maintenance therapy with mesalazine can reduce the risk of colorectal cancer by 75 per cent in UC. However, maintenance therapy seems to be less effective in CD, but there is evidence that relapse after small bowel resection can be prevented by giving doses of more than 2g/day. To treat active disease higher doses, of 4g/day, are generally needed.¹

Since the delivery characteristics of the various formulations are different, preparations of mesalazine are not interchangeable and prescribers should indicate the proprietary name of the preparation that they intend to be used. Rectal administration is effective in distal UC and proctitis. In severe UC, oral and topical rectal formulations should be combined to give prompt symptom relief.

Side effects, such as headache, fever, pruritus and rashes are common with sulfasalazine and 5-ASA, while the less common sulphonamide-related ADRs, such as agranulocytosis (ie, insufficient number of neutrophils), Stevens-Johnson syndrome (a potentially life-threatening skin disease) and pancreatitis, are generally avoided by using mesalazine. Mesalazine is tolerated by 80 per cent of patients who are intolerant of sulfasalazine. About 16 per cent of patients taking mesalazine experience ADRs including diarrhoea, nausea, headache, abdominal pain and rashes. However, there are reports of 5-ASA induced pancytopenia (generalised reduction in blood cells), reversible hair loss, pancreatitis and lupoid phenomenon. Treatment should be stopped if blood dyscrasia occurs.

Use of 5-ASA has been associated with renal toxicity and patients should be regularly screened for renal impairment. Impaired male fertility with sulfasalazine is well documented but appears to be caused by the sulfapyridine moiety, since fertility has returned in most patients changed to 5-ASA.

— Corticosteroids

Corticosteroids are indicated in the management of the acute phase of UC and CD. Their mode of action is through the modulation of several inflammatory pathways, including interleukin and arachidonic acid metabolism. Moderate to severe relapses should be managed with oral prednisolone or budesonide or, in severe cases, with intravenous hydrocortisone. Topical preparations (foams, suppositories or enemas) can play a role in UC, either alone or in combination with oral steroids. They should generally not be used once a patient requires high-dose oral or intravenous steroid therapy. Strategies should aim to maximise local effects and limit systemic effects.

To induce remission, oral steroid therapy can be instigated. Prednisolone (40mg once

a day) seems to be sufficient, with high doses not proving to be any more effective but being associated with a higher incidence of ADRs. Patients needing admission to hospital are usually treated with intravenous hydrocortisone (100mg three to four times a day) until symptoms improve, at which point treatment can be changed to oral prednisolone. The prednisolone dose should be reduced slowly (by 10mg every two weeks approximately) until remission occurs — too rapid reduction is associated with relapses. Doses below 20mg are not generally effective in active disease states.

Corticosteroids are accepted as being unsuitable for use in maintaining remission, because of the potentially harmful side effects of long-term treatment. However, some patients with CD do seem to have fewer flare-ups if they are maintained on low dose (5mg daily) prednisolone.

Budesonide is not as effective as prednisolone, but is an alternative choice in the acute phase management of IBD in the ileum and ascending colon. It is poorly absorbed, and any drug that is absorbed is readily cleared by first-pass metabolism, thereby reducing the risk of ADRs. However, the absorbed drug has an affinity for glucocorticoid receptors 50 to 100 times that of prednisolone and so long-term treatment is not advocated.

The ADR profile of glucocorticosteroids includes adrenal suppression, Cushing's syndrome, acne, mood and sleep disturbances, diabetes and osteoporosis especially with prolonged use. Osteoporosis is common in IBD patients but treatment strategies are still subject to debate.²

The distribution and absorption characteristics of steroids delivered rectally vary greatly. Hydrocortisone is readily absorbed from the rectal mucosa, with high peak plasma concentrations, whereas prednisolone metabisulphate is poorly absorbed. However, the formulation used also influences absorption, with foams generally resulting in

Panel 1: Formulation and delivery details about some of the oral preparations of aminosalicylates currently available

Proprietary name	Generic drug	Formulation details	Mode of delivery	Site of delivery
Pentasa	Mesalazine	Microgranules coated with ethylcellulose membrane	Diffusion through semi-permeable membrane	Duodenum, jejunum, ileum, colon
Salofalk Mesren MR	Mesalazine	Coated with Eudragit L100 resin	pH-dependent (pH6) delayed release	Ileum, colon
Asacol MR	Mesalazine	Coated with Eudragit S resin	pH-dependent (pH7) delayed release	Terminal ileum, colon
Salazopyrin Colazide Dipentum	Sulfasalazine Balsalazide Olsalazine	Consist of a 5-ASA and SA prodrug dimer	Cleavage by azoreductase produced by intestinal bacteria	Colon

Panel 2: Common rectal preparations of corticosteroids and their properties

Preparation	Steroid	Typical dose	Peak plasma concentration achieved (nmol/L)	Peak tissue levels achieved (ng drug/g of tissue)	Licensed indication
Predsol (enema)	Prednisolone phosphate	20mg (in 100ml) at night	365 (after 2h) ³	44 ³	Rectal and distal UC
Predsol (suppository)	Prednisolone phosphate	5mg twice a day	Data unavailable	Data unavailable	Proctitis and rectal complications of CD
Predenema	Prednisolone sodium metasulphobenzoate	20mg (in 100ml) at night	45 (after 2h) ³	256 ³	UC
Predfoam	Prednisolone sodium metasulphobenzoate	20mg once or twice daily	320 (after 4h) ⁴	4,874 ⁴	Rectal and distal UC
Colifoam	Hydrocortisone acetate	125mg once or twice daily	510 (after 4h) ⁵	Data unavailable	UC, proctitis, proctosigmoiditis
Entocort enema	Budesonide	2mg (in 100ml) at night	2.5 (after 1.5h) ⁶	Data unavailable	UC involving rectal and rectosigmoid disease

Numbers in superscript refer to article references. "UC" is ulcerative colitis and "CD" is Crohn's disease

higher plasma concentrations than enemas or suppositories (see Panel 2 above).

Topical preparations are only indicated to treat UC and colitis in CD and should be chosen according to the part of the colon affected.

Thiopurines

Patients who have more than two acute attacks a year requiring steroids, and patients who relapse if their prednisolone dose is reduced below 15mg or within six weeks of stopping steroids, should be considered for treatment with thiopurines (ie, azathioprine [AZA], 6-mercaptopurine [6-MP] and thioguanine). It should be noted that none of these drugs is licensed for the treatment of IBD.

As purine antimetabolites, thiopurines inhibit RNA synthesis, but the mechanism for the therapeutic effect is largely unknown. It is proposed that they modulate T-cells, which would explain why it can take several weeks for maximum efficacy to be achieved. The ADR profile of thiopurines consists of the commonly experienced but reversible influenza-like symptoms, which tend to happen in weeks two to three of the treatment (and which do not necessitate treatment withdrawal), and the less common but severe incidences of leucopenia, hepatotoxicity and pancreatitis. 6-MP is the active metabolite of AZA and around 28 per cent of patients intolerant to AZA will tolerate 6-MP. Thioguanine has been used but has been associated with a greater risk of hepatotoxicity. Weekly full blood counts (FBCs) and liver function tests (LFTs) are recommended before starting treatment and for the first eight weeks of treatment, after which time monthly tests are advocated. Patients need to be counselled to seek medical advice if signs of infection, such as a

sore throat, occur. There is no evidence that the incidence of lymphoma increases with the use of these agents.

The potential for serious drug interactions is well documented. Allopurinol inhibits the principal pathway for detoxification of AZA and 6-MP. Patients receiving azathioprine and allopurinol concomitantly should have their dose of azathioprine reduced to approximately a third to a quarter of the usual dose.

Thiopurines are effective both in active disease states and in the maintenance of remission in CD and UC. They should be used at doses of 2–2.5mg/kg/day for azathioprine and 1–1.5mg/kg/day for 6-MP in CD and UC. Advice to take these drugs with food and to divide the dose will reduce initial nausea. The maximum dose is determined by the incidence of leucopenia, which is dependent on the thiopurine methyl transferase (TPMT) activity. Evidence is still limited about the importance of TPMT genotyping of patients in determining the initial thiopurine dose.

Two Cochrane reviews of the efficacy of thiopurines to induce remission suggest an NNT (number needed to treat) of five and an NNH (number needed to harm) of 14⁷ and to maintain remission an NNT of 7 and an NNH of 19.⁸

Methotrexate

Methotrexate is unlicensed for use in IBD and should only be used under the supervision of a gastroenterologist. It is effective in inducing remission and preventing relapses in CD patients refractory to AZA and 6-MP. The mechanism of action is not clear because the cytotoxic action does not explain the anti-inflammatory effect. Doses of less than 15mg/week are generally ineffective and 25mg/week is standard in CD. Oral administration is preferred but parenteral

administration may be more effective. The co-prescribing of folic acid 5mg daily for six days a week may alleviate gastrointestinal ADRs, such as nausea and vomiting, diarrhoea and stomatitis. The serious ADRs of hepatotoxicity and pneumonitis require the treatment to be stopped. Monitoring of FBCs and LFTs is required weekly in the first four weeks, and then monthly.

Ciclosporin

Ciclosporin is generally used as a last attempt to prevent surgery in refractory UC. It is not effective in CD. Like methotrexate, it is unlicensed for use in IBD and should only be used under the supervision of a gastroenterologist.

The blocking of lymphocyte activation is proposed as its mechanism of action. Its use is controversial because of its toxicity and long-term failure rate and it should only be used as a bridge to instigate thiopurine therapy successfully. Generally a dose of 2–4mg/kg/day (in two or three divided doses) is recommended, depending on severity of the flare up.

Minor ADRs, such as malaise, tremor, headaches, abnormal LFTs, hirsutism and gingival hyperplasia are common. Serious ADRs include renal impairment, infections and neurotoxicity. In patients with low cholesterol and magnesium, the risk of seizures is increased. It is recommended that FBCs, renal function, LFTs, blood pressure and, in addition, cholesterol and magnesium levels are monitored before therapy is started. Ciclosporin levels should be monitored weekly for the first month and then monthly, aiming for plasma concentrations of around 100–200ng/ml. Approximately 50 per cent of patients whose condition is serious enough to require ciclosporin therapy will still need to undergo surgery within a year of treatment.⁹

— Anti-TNF therapies

Infliximab is currently the only licensed biological therapy for CD. It is a genetically engineered murine-human chimeric monoclonal antibody. The mode of action is not fully understood but includes the neutralisation of soluble and transmembrane tumour necrosis factors.

National Institute for Health and Clinical Excellence guidance recommends infliximab for severe active CD refractory to immunomodulatory drugs. A single infusion of 5mg/kg is recommended for active CD and, if needed, a maintenance dose of 5mg/kg every eight weeks (from the ACCENT-1 study).¹⁰ For patients with fistulating CD, a single dose of 5mg/kg at weeks 0, 2 and 6 is effective, again with maintenance infusions every eight weeks (from the ACCENT-2 study).¹¹

Two studies published in December 2005 (ACT-1 and ACT-2)¹² look at the efficacy of infliximab in UC. Based on the favourable outcome of these studies, infliximab is now licensed for UC.

All patients should receive concomitant immunomodulator therapy if tolerated. This will reduce the risk of developing antibodies against infliximab and increase the time between maintenance doses. Infliximab increases the incidence of infections and all patients should have a chest X-ray to exclude past or present tuberculosis infections before starting infliximab therapy. Patients should be monitored for signs of infections during treatment, and for several months after it has stopped. There is a four- to five-fold risk of tuberculosis associated with this therapy and an active infection, such as an abscess, is an absolute contraindication to treatment. There does not seem to be an increased risk of lymphoma.

Acute infusion reactions related to histamine are common and it is advised to give patients paracetamol and/or antihistamines 20min before the infusion. Full anaphylactic reactions are rare but the 2h infusion must be given in a hospital with resuscitation facilities available. Common ADRs are headache, dizziness, nausea, rash, raised LFTs, abdominal pain, fatigue and dry skin. Patients retreated after a break may experience a delayed hypersensitivity reaction and need to be appropriately counselled because reactions can occur within 12 days of administration.

— Safety in pregnancy

The conception potential of women with CD and UC is similar to that of the general population. In general, pharmacological therapy used for CD during pregnancy is similar to that used in non-pregnant patients. It is essential to the health of the mother and the fetus that disease control is optimised both before conception and during preg-

nancy. Monitoring and treating anaemia associated with folate and/or vitamin B₁₂ deficiencies is essential.

Drugs used in IBD and considered to be safe in pregnancy are:

- Mesalazine
- Sulfasalazine
- Prednisolone
- Methylprednisolone
- Hydrocortisone
- Loperamide
- Ampicillin
- Cephalosporins
- Metronidazole (second and third trimester only)

Those considered probably to be safe in pregnancy are:

- Azathioprine
- 6-Mercaptopurine
- Budesonide
- Ciprofloxacin (second and third trimester only)

For infliximab and olsalazine, insufficient data is currently available to decide on their safety status in pregnancy. Methotrexate is contra-indicated in pregnancy.

It should be noted that evidence for the safety of IBD treatments in pregnancy is to be considered with caution because data are mainly based on case reports.

— Probiotics

Probiotics are products containing viable micro-organisms. Interest in probiotics is currently high and there are many products to choose from in an unregulated market. Evidence for their use is of questionable quality. Studies generally have small subject numbers and are difficult to compare because of differences in the strains, the doses and the formulations used. There are no *in vitro* biomarkers for probiotic performance *in vivo*.

More research is therefore needed on a variety of issues, including:

- Best strain selection for different indications
- Product quality and shelf life
- Survival and gut transit times of the different strains
- Best dose
- Best delivery vehicle
- Ways to monitor efficacy

— Worm therapy

Industrialised western societies have a higher incidence of IBD than developing countries. It is speculated that western societies have become too clean and that, epidemiologically, the prevalence of IBD is inversely proportional to the prevalence of

helminthic infections, which are common in less clean and more crowded societies (the "hygiene hypothesis").

Hence, the non-invasive and non-infective pig whipworm helminth (*Trichuris suis*) has been suggested as treatment for IBD. It is genetically related to human whipworm and its ova can colonise a human host for several weeks. Ova are then shed in the stool but are not able to reinfect because they are not embryonated. The ova are harvested from pathogen-free pigs and patients swallow 2,500 eggs weekly for three weeks, after which time the symptoms are expected to improve.¹⁴⁻¹⁶ However, the statement that *Trichuris suis* is non-invasive has been questioned.¹⁷

— Leukapheresis for IBD

NICE guidance is available on the use of leukapheresis.¹⁸ The procedure involves the removal of leukocytes from the blood, either by passing blood through an adsorptive system or by centrifugation. In each system, venous blood is removed in a continuous flow, anticoagulated, processed to deplete the leukocytes, and returned to the circulation. A leukapheresis session takes approximately 1-2 hours. The procedure is usually carried out once or twice a week, for about 5-10 sessions.

— Conclusion

Therapeutic choices need to be tailored to the patient and the clinical presentation of the disease. It is essential that clinical staff clearly understand the different formulations, so they are able to integrate them successfully into their treatment plans. Therapeutic strategies may well change rapidly over the next few years. New biological agents look set to come onto the market (summarised in Panel 3, p166), which are likely to have a significant impact on the health economy.

Although, pharmacotherapy is the main stem of IBD management, a holistic approach is necessary, particularly because IBD affects patients fairly early on in their

Hospital Pharmacist online

Hospital Pharmacist is available online at www.pjonline.com/hp/index.html. The website contains the current issue and an archive of back issues from January 2000 onwards. There are also links to the regular features in *Hospital Pharmacist* (eg, Life-long Learning, meeting reports, comments, careers, focus on technicians). The site also contains advice to contributors to *Hospital Pharmacist*, information about the annual *Hospital Pharmacist* conference, a link to *The Pharmaceutical Journal* careers page and information on subscribing to the journal.

Panel 3: Some of the agents in development for treating IBD

Agent	Mode of action	Progress of development
Adalimumab	Fully human antibody against TNF-alpha	Expected licensing late 2006
Certolizumab	Pegylated monoclonal antibody fragment TNF-alpha	Submission to FDA made in US and provisional NICE document in preparation
Rituximab	Monoclonal antibody against CD-20	Phase III clinical trial in progress
Basiliximab	Chimeric antibody against IL-2	Phase II clinical trial in progress
Sargramostin	GM-CSF	Phase II clinical trial in progress
Fontolizumab	Interferon-gamma antibody	Phase II clinical trial in progress
Interferon-beta	Cytokine inhibitor	Phase II clinical trial in progress
Visilizumab	Humanised monoclonal antibody against CD3	Phase II clinical trial in progress
Nitazoxamide	Anti-infective	Phase II clinical trial in progress
Rivenprost	Prostaglandin E ₂	Phase II clinical trial in progress
STA-5326	Oral IL-12 inhibitor	Phase II clinical trial in progress
Filgrastim	G-CSF	Phase I clinical trial in progress

GM-CSF means granulocyte-macrophage colony-stimulating factor, G-CSF means granulocyte colony-stimulating factor, CD-3 is a type of T-cell surface glycoprotein, IL means interleukin, FDA means Food and Drug Administration and "NICE" means National Institute for Health and Clinical Excellence

lives. This includes offering patients information about nutrition and lifestyle choices, as well as discussing pharmacological and surgical options with them. If necessary, advice about pregnancy issues and genetic counselling should be offered. Psychological support, including details about patient support groups is often also welcomed (see Panel 4). The British Society of Gastroenterology's guidelines for the management of inflammatory bowel disease in adults advocate a multidisciplinary approach to care, outlining service delivery expectations.¹

References

- Carter M, Lobo A, Travis S. Guidelines for the management of inflammatory bowel disease in adults. *Gut* 2004;53(Suppl V):v1-16.
- Scott E, Gaywood I, Scott B. Guidelines for osteoporosis in coeliac disease and inflammatory bowel disease. London: British Society of Gastroenterology; Feb 2000.
- McIntyre PB, Macrae FA, Berghouse L, English J, Lennard-Jones JE. Therapeutic benefits from a poorly absorbed prednisolone enema in distal colitis. *Gut* 1985;26:822-4.
- Cann PA, Holdsworth CD. Systemic absorption from hydrocortisone foam enema in ulcerative colitis. *Lancet* 1987;1:922-3.
- Rodrigues C, Lennard-Jones JE, English J, Parsons DG. Systemic absorption from prednisolone rectal foam in ulcerative colitis. *Lancet* 1987;1:1497.
- Nyman-Pantelidis M, Nilsson A, Wagner ZA. Pharmacokinetics and retrograde colonic spread of budesonide enemas in patients with distal ulcerative colitis. *Alimentary Pharmacology and Therapeutics* 1994;8:617-22.
- Sandborn W, Sutherland L, Pearson D, May G, Modigliani R, Prantera C. Azathioprine or 6-

mercaptopurine for induction of remission in Crohn's disease. *The Cochrane Database of Systematic Reviews* 2006;(1):CD000545.

- Pearson DC, May GR, Fick G, Sutherland LR. Azathioprine for maintenance of remission in Crohn's disease. *The Cochrane Database of Systematic Reviews* 2006;(1):CD000067.
- Podolsky D. Inflammatory bowel disease. *New England Journal of Medicine* 2002;347:417-29.

- Hanauer SB, Feagan BG, Lichtenstein GR, Mayer FM, Schreiber S, Colombel JF et al. Maintenance infliximab for Crohn's disease: the ACCENT 1 randomised trial. *Lancet* 2002;340:1398-1405.
- Sands B, Anderson F, Bernstein C, Lloyd FM, Schreiber S, Colombel JF et al. Infliximab maintenance therapy for fistulizing Crohn's disease. *New England Journal of Medicine* 2004;350:876-85.
- Rutgeerts P, Sandborn WJ, Feagan BG, Reinisch W, Olson A, Johanns J et al. Infliximab for induction and maintenance therapy for ulcerative colitis. *New England Journal of Medicine* 2005;353:2462-76.
- Mottet C, Juillerat P, Gonvers J, Froehlich F, Burnard B, Vader JP et al. Pregnancy and Crohn's disease. *Digestion* 2005;71:45-61.
- Summers RW, Elliott DE, Urban JF Jr, Thompson R, Weinstock JV. Trichuris suis therapy in Crohn's disease. *Gut* 2005;54:87-90.
- Summers R, Weinstock D, Urban J, Thompson RA, Weinstock JV. Trichuris suis therapy for active ulcerative colitis: A randomized controlled trial. *Gastroenterology* 2005;128:825-32.
- Better medicine through biology. Available at www.ovamed.de/english/home/home.html (accessed 25 April 2006).
- Van Kruiningen H, West A. Potential danger in the medical use of Trichuris suis for the treatment of inflammatory bowel disease. *Inflammatory Bowel Disorders* 2005;11:515.
- Leukapheresis for inflammatory bowel disease. Available at www.nice.org.uk/pdf/ip/IPG126guidance.pdf (accessed 25 April 2006).
- Hanauer S, Present D. The state of the art in the management of inflammatory bowel disease. *Review of Gastroenterology Disorders* 2003;39:8-21.

Panel 4: A patient's view of the importance of an holistic approach in the management of inflammatory bowel disease (IBD)

Most people with ulcerative colitis or Crohn's disease have their condition diagnosed in their late teens or early twenties. This is not the best time to take on board the implications of living with an unpredictable chronic illness.

In my case, I was not offered enough information about my condition and quite happily lived in denial for years, stopping my medicines when I was in remission and being thoroughly despondent when I relapsed.

The experience of those in our patients' group in Brighton (contactable by e-mailing: ibdactiongroup.yahoo.co.uk) is that people with IBD react to their illness in many different ways. However, a common feeling is one of isolation and of frustration with the health care system.

In my experience, most people with IBD benefit from access to good-quality information about treatment options. However, a lot of the information available, particularly over the internet, is either too technical, confusing or even potentially dangerous.

Having the opportunity to receive information from local health care professionals is vital. At our group, we have found that encouraging patients to prepare questions in advance of appointments is helpful, as is organising and attending more informal forums such as open days and awareness events.

Understanding our treatment is the first step, but a bigger task is living day-to-day with a long-term condition. Sharing tips and experiences with others helps improve self-management skills and acknowledges the difficulties of living with IBD. We find that health care professionals often fail to consider this wider context.

In Brighton, patients have a number of forums to meet, including an e-mail discussion group, regular drop-in sessions, a hospital visiting service and an annual day conference. In addition, the NHS now offers a six-week self-management course for people living with any long-term health condition as part of the Expert Patient Programme.