Survival for colorectal cancer

EDITOR,—The EUROCARE study of Gatta et al [Gut 2000;47:533–8] provides important information on survival for colorectal cancer in different parts of Europe. In particular, it relates survival to stage, a feature which was absent from earlier studies.1,2 The study also looks at procedures used in determining stage which may be important factors in accurate staging—for example, higher numbers of lymph nodes sampled will be more likely to detect metastases and consequent upstaging of tumours.3

However, the usefulness of this study is reduced by inaccuracies in the descriptions of staging and choice of staging system. The authors have chosen to use Dukes’ staging system which has been in use for at least 50 years and has been well characterised by many studies with long term follow up.4 Unfortunately, they have given incorrect labels to some of the stages in this system. In tables 2 and 3, Dukes’ stages A and B are described as being “confined to the bowel wall” which is incorrect. Dukes’ stage A is invasion into, but not through, the bowel wall whereas Dukes’ stage B is invasion through the bowel wall but without lymph node metastases.5 Dukes’ stages A and B combined could have been correctly labelled as no lymph node metastases. In the methods section the authors state that where only the TNM stage was available, TNM categories were converted to Dukes’ stages and where stage was not explicitly stated this was reconstructed from information records (in 45% of cases in the study). It is hoped that the correct descriptions of Dukes’ staging were used for this encoding process.

While it is understandable that the authors have chosen to use a well established staging system, the Dukes’ system does have a major flaw which is improved by the TNM system.6 The Dukes’ B stage contains tumours which may be in the pT3 stage (tumour invades through the muscularis propria into the subserosa or into non-peritonealised pericolic or perirectal tissues) or pT4 stage (tumour directly invades other organs or structures and/or perforates visceral peritoneum) both with a pN0 nodal stage. The prognosis for pT3 pN0 tumours is relatively good whereas the prognosis for pT4 pN0 tumours is poor with a high risk of local recurrence.7 8 A final histopathology observation on this study protocol is the threshold set for the number of lymph nodes examined. The authors state that “it is generally considered that at least 12 lymph nodes should be examined for accurate staging” but this is not a universally accepted threshold. The recommendation from the Royal College of Pathologists in the UK is that all lymph nodes identified in the resection specimen should be examined histologically but does not specify an arbitrary minimum number9 10 as pointed out by Dr Cross, Dukes’ B includes tumours perforating the visceral peritoneum and directly invading other organs (T4), and our category “Dukes’ A and B” encompasses pT1–T4 N0 M0. A better definition of this category would have been “absence of lymph node and distant metastasis.” The stage definitions of Hermanek and Sобin’ were in fact used for the encoding process, as stated in the article. Furthermore, they were specified a priori in the study protocol as the reference standard, so as to avoid problems of comparison between registries.

CHOICE OF STAGING SYSTEM

We used Dukes’ instead of TNM because in 1990 Dukes’ was the most widely used staging procedure in all the registries. The other reason we decided to use Dukes’ was that from the information available to us (patients’ clinical and pathological notes), it was not possible to clearly separate pT3pN0 from pT4pN0 cases (all Dukes’ B) which a Dr Cross pointed out, differ markedly in prognosis. Furthermore, it was often impossible to distinguish pT2 from pT3 cases, and for this reason we considered Dukes’ A and B together. The next step was to determine whether the study implied the use of information from numerous hospitals and pathology laboratories, which had various ways of recording information. Greater standardisation of stage reporting in pathology notes would be highly desirable.

We are currently engaged in a new high resolution EUROCARE study on colorectal cancers diagnosed in 1997 when the TNM system was more widely used than in 1990. However, the distinction between pT categories is still not adequately made in a considerable number of cases.

NUMBER OF LYMPH NODES EXAMINED

Our statement that “at least 12 lymph nodes should be examined for accurate staging” derives from the International Documentation System, referenced by Fielding and colleagues’ in their article in 1991. The recommendation that, “Before deeming a radical resection to be without lymph node metastasis, it is recommended that at least 12 lymph nodes be examined.” Furthermore, the 1993 TNM supplement’ states: “histological examination of a regional lymphadenectomy specimen . . . will ordinarily include 12 regional lymph nodes,” and this number is considered “adequate for staging.”

Our analysis used four categories for number of nodes examined: 0, 1–5, 6–11, and ≥12; choosing other division points did not change the rank of the registries. However, it is important to note that in the hospital study taken as an example by Dr Cross’, 12 or more lymph nodes were examined in 50% of cases—well above the percentages reported by our study (range 2–31%). This suggests that in 1990 the extent of resection or the thoroughness of the pathological examination (or both) would not be considered adequate by today’s standards.

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Helicobacter pylori, Lewis antigens, and inflammation

**Editor,—**In a recent issue of Gut, Zheng et al reported that acute ulcer disease was associated with increased expression of Lewis (Le) antigens but not 

*Helicobacter pylori* strains isolated from Chinese patients infected with *Helicobacter pylori* (Gut 2000;47:18–22). These data raise an interesting question of further complexity and potentially add to our understanding of the role of *H. pylori* infection in the individual and its relationship with the expression of Lewis antigens in ulcer disease. The nature of the infection is uncertain, however, as the relationship between *H. pylori* strain and Lewis antigen expression is not clear, and the role of the Lewis antigen in the pathogenesis of ulcer disease is also uncertain.

To determine whether the expression of Lewis antigens in *H. pylori* strain infection is related to the host's Lewis phenotype, we compared the expression of Lewis antigens in *H. pylori* strains isolated from Chinese patients infected with *H. pylori* with those from our population. We have reviewed our raw data for the general population and for ulcer disease and the relationship between *H. pylori* strain and Lewis antigen expression in ulcer disease is related to the host's Lewis phenotype. We have also reviewed our raw data for the general population and for ulcer disease and the relationship between *H. pylori* strain and Lewis antigen expression in ulcer disease is related to the host's Lewis phenotype.

The results of our study indicate that the expression of Lewis antigens in *H. pylori* strain infection is related to the host's Lewis phenotype. We have also reviewed our raw data for the general population and for ulcer disease and the relationship between *H. pylori* strain and Lewis antigen expression in ulcer disease is related to the host's Lewis phenotype. We have also reviewed our raw data for the general population and for ulcer disease and the relationship between *H. pylori* strain and Lewis antigen expression in ulcer disease is related to the host's Lewis phenotype.

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infection revealed that *H* *pylori* cally existed on and between the surface in Carnoy fixed stomachs with *H* *pylori*.

13 In figure 1, *H* *pylori* type and the gland mucous cell type (fig 2). Types of mucins: the surface mucous cell structure consisting of laminating layers of mucins; the surface mucous cell type (blue) and the gland mucous cell type (brown) (dual staining of GOTS-PCS). (C) Carnoy fixed gastric mucosa without *Helicobacter pylori* infection. Immunostaining with anti-MUC5AC (45M1) labels gastric surface mucous cells. The reactivity of MUC5AC and MUC6 proteins—tissue sections.

Dual staining of GOTS-PCS of Carnoy fixed gastric mucosa without *H pylori* infection showed that SMGL had a characteristic structure consisting of laminating layers of two types of mucins: the surface mucous cell type (blue) and the gland mucous cell type (brown) (dual staining of GOTS-PCS). (B) Immunostaining with anti-MUC5AC (45M1) labels gastric surface mucous cells. The reactivity of anti-MUC5AC (45M1) is identical to that of GOTS (see (A)) (immunoperoxidase method with anti-MUC5AC (45M1)). (C) Carnoy fixed gastric mucosa without *Helicobacter pylori* infection. The surface mucous gel layer had a characteristic structure consisting of laminating layers of two types of mucins: the surface mucous cell type (blue) and the gland mucous cell type (brown) (dual staining of GOTS-PCS). (D, E) Carnoy fixed gastric mucosa with *H pylori* infection. (D) *H pylori* is stained red by an immuno-alkaline phosphatase method with anti-*H pylori* antibody. Numerous *H pylori* exist in the surface mucous gel layer and on and between the surface mucous cells. In the surface mucous gel layer, *H pylori* preferentially colonise in the layer of surface mucous cell type mucins (dual staining of GOTS-PCS followed by immunostaining for *H pylori*). (E) *H pylori* is stained red by an immuno-alkaline phosphatase method with anti-*H pylori* antibody. Numerous *H pylori* exist on and between the surface mucous cells but not on pyloric gland cells (dual staining of GOTS-PCS followed by immunostaining for *H pylori*).

The SMGL appears to be the major site of *H pylori* infection. *H pylori* preferentially colonise in the layer of surface mucous cell type mucins (dual staining of GOTS-PCS). (D) *H pylori* is stained red by an immuno-alkaline phosphatase method with anti-*H pylori* antibody. Numerous *H pylori* exist in the surface mucous gel layer and on and between the surface mucous cells. In the surface mucous gel layer, *H pylori* preferentially colonise in the layer of surface mucous cell type mucins (dual staining of GOTS-PCS followed by immunostaining for *H pylori*).

Advantage of fixing the gastric surface mucous gel layer (SMGL) in paraffin embedded tissue sections. Dual staining of GOTS-PCS of Carnoy fixed gastric mucosa without *H pylori* infection showed that SMGL had a characteristic structure consisting of laminating layers of two types of mucins: the surface mucous cell type and the gland mucous cell type (fig 1C). Dual staining of GOTS-PCS in conjunction with immunostaining for *H pylori* in Carnoy fixed stomachs with *H pylori* infection revealed that *H pylori* characteristically existed on and between the surface mucous cells and in the SMGL (fig 1D). In the SMGL, this organism was most often associated with the layer of surface mucous cell type mucins (fig 1D, E). Receptors responsible for adherence of *H pylori* might exist only on the plasma membrane of surface mucous cells. In the SMGL, these receptors appear to be mixed with the surface mucous cell type mucins. The SMGL inhabited by *H pylori* revealed marked derangement of the multilaminating structure with fragmentation of the mucin layers and formation of vacuoles (fig 1D, E). After eradication of *H pylori*, the SMGL regained the laminating structure. Alteration of the SMGL found in *H pylori* infected stomachs suggests destruction of the SMGL by bacterial lipase and protease from *H pylori*.

After eradication of *H pylori*, the SMGL regained the laminating structure. Alteration of the SMGL found in *H pylori* infected stomachs suggests destruction of the SMGL by bacterial lipase and protease from *H pylori*.
molecules. Glycosylation is a complex process that may easily become disturbed in Helicobacter pylori infection, for example. Thus as glycosylation may change under pathological conditions, I would urge the authors and other workers in the field to concentrate on the invariant part of the mucins; the non-O-glycosylated parts of the protein, as discussed by us previously.¹

One of the issues addressed by Ota et al is the correlation of Helicobacter pylori infection with disturbance of the laminated gastric mucus layer, as demonstrated in fig 1C versus fig 1D. In common with the authors, it is tempting to speculate that Helicobacter pylori has a direct role in this disturbance. Similar to most bacteria, Helicobacter pylori produces enzymes that in principle can degrade mucins, such as proteases and “mucinases”, as referred to by the authors. I personally doubt whether Helicobacter pylori is directly responsible for disturbance of the mucus layer for two reasons. Firstly, as the bacterium resides in the mucus layer, what would be the benefit of destroying its own milieu? Secondly, there are data by many authors to indicate that the tissue dynamics of the gastric mucosa is altered by Helicobacter pylori infection. Accompanying the inflammatory response, epithelial turnover is enhanced, and there are profound shifts in cell populations. Obviously, this type of discussion calls for a more dynamic approach to this problem. However, in countries such as Japan where corpus gastritis and mucosal atrophy are common among patients infected with Helicobacter pylori, the situation is not as simple. We performed a similar study by measuring basal acid output (BAO) and maximum acid output (MAO) using pentagastrin administration before and six months after eradication in Japanese patients with GU (n=21) and DU (n=22). In both patients with GU and DU, serum gastrin levels were significantly higher than those of control subjects (p<0.01) before eradication; they decreased significantly after eradication (GU: 129.7 (19.2) to 94.6 (15.0) pg/ml; DU: 116 (19.8) to 90.3 (16.5) pg/ml; p<0.05). These data are similar to those of Iijima et al (Gut 2000;46:20–6). In contrast with their data, we found an interesting fact: acid secretion of the Japanese may be responsible for the controversy may be that a considerable number of patients with DU had corpus gastritis, which may somehow cause hypo-acidity. We previously reported that gastric acid secretion in Japanese subjects is lower than that in Europeans or North Americans, irrespective of Helicobacter pylori infection. As Helicobacter pylori is induced to report a decrease in corpus gastritis more easily in subjects with decreased acid secretion,¹ we suggested that low gastric acid secretion of the Japanese may be responsible for the higher incidence of corpus gastritis and atrophy in Japanese subjects with Helicobacter pylori infection. This appears to be the case, even for patients with DU in Japan, and it may be the reason why acid secretion was not significantly reduced after Helicobacter pylori eradication in our DU patients.

Data on changes in gastric stimulated acid secretion after eradication in patients with DU are controversial,¹ although most reports show a decrease in acid secretion.¹ As Iijima et al indicated, the most likely reason for the controversy may be that a considerable number of patients with DU had corpus gastritis, which may somehow cause hypo-acidity. We previously reported that gastric acid secretion in Japanese subjects is lower than that in Europeans or North Americans, irrespective of Helicobacter pylori infection. As Helicobacter pylori is induced to report a decrease in corpus gastritis more easily in subjects with decreased acid secretion,¹ we suggested that low gastric acid secretion of the Japanese may be responsible for the higher incidence of corpus gastritis and atrophy in Japanese subjects with Helicobacter pylori infection. This appears to be the case, even for patients with DU in Japan, and it may be the reason why acid secretion was not significantly reduced after Helicobacter pylori eradication in our DU patients.

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Helicobacter pylori infection and acid secretion in patients with duodenal ulcer in Japan

EDITOR—Helicobacter pylori infection affects gastric acid secretion of the host in various ways. For corpus gastritis and subsequent development of mucosal atrophy induced by Helicobacter pylori infection result in decreased acid secretion.¹ In contrast, several investigators have reported elevated acid secretion in patients with duodenal ulcer

Figure 1 Effects of Helicobacter pylori eradication on maximum acid output (MAO) in Japanese patients with duodenal (DU) and gastric (GU) ulcers. MAO in response to pentagastrin (6 μg) was measured before and six months after Helicobacter pylori eradication. *P<0.05 vs before eradication.

Figure 2 Correlation between decrease in serum gastrin concentration (−Δgastrin) and increase in maximum acid output (ΔMAO) in response to pentagastrin (6 μg) by Helicobacter pylori eradication in Japanese patients with duodenal (DU) and gastric (GU) ulcers. Groups A, B, and C show DU patients in whom MAO decreased (more than 1 mEq/h), did not change, and increased (more than 1 mEq/h) after eradication, respectively.


It has been a matter of debate whether gastric acid hypersecretion observed in patients with DU is a result of H pylori infection or if the infection accelerates development of DU in subjects who originally had acid hypersecretion. Our data showing that acid secretion was recovered in association with the decrease in serum gastrin levels in some DU patients and that MAO levels in patients with DU after eradication are still higher than those of normal subjects without H pylori infection may support the latter idea. Moreover, this idea may explain the fact that there are more patients with GU than DU among Japanese subjects, who exhibit lower acid secretion than Western patients.

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BOOK REVIEWS


I was taught as a medical student that the major function of the colon was that of a storage organ. It was then presumed that once the colon have evolved and the complexities of colonic function have been better understood, as described by Gibson and Roberfroid’s (editors) multiauthored book Colonic Microbiota, Nutrition and Health.

Although the editors state that the purpose of the book is to overview current knowledge of the activities and functions of the gut microflora, this may be beyond these major boundaries and takes us on an ecological journey into the exciting life of gut microflora and their impact on colon function in health and disease, and the intimate critical relationship between diet, bacteria, and quality of life. Gastroenterologists are still recovering from the impact that a bacterium, Helicobacter pylori, has had on upper gastrointestinal tract pathology. In this context it is interesting to note that the large bowel is the most heavily colonised part of the gastrointestinal tract yielding up to 10^{13} bacteria per gram of intestinal contents in healthy human subjects. It is a complex ecosystem in which the numerous and different species of bacteria degrade and ferment substrates that have escaped digestion in the small bowel. Major genera include bacteroides, bifidobacteria, lactobacilli, clostridia, and enterobacteria, and the many products of bacterial fermentation of the substrate that reaches the colon are short chain fatty acids (SCFA) and gases, including hydrogen, carbon dioxide, and in some individuals, methane. The relevance of SCFA is that they act as a source of energy for intestinal mucosal cells and reduce the pH of colonic contents. One particular SCFA, butyrate, may be important in protecting against colon cancer. The relevance of fermentation to human metabolism can be gauged from the fact that the energy equivalent of 15–40 g of carbohydrate is metabolised by the human body. The book outlines the technological revolution that has occurred in understanding the natural microbial world. Molecular biology has invaded gut microbiology with the limitations of enrichment cultural techniques being surpassed by the detection of genomic DNA or analysis of rRNA.

A fascinating aspect of the book that concerns and affects all of us is the chapters on food. With the craze of low carbohydrate diets to counter obesity sweeping the USA, it is refreshing to realise the importance of carbohydrates and the concept of functional food. These new approaches are concerned with enzymes, bacteria, and foods that target the large intestine. These foods contain an ingredient that does not undergo significant modification during transit through the small intestine but reach the colon where they are utilised by the resident bacteria producing metabolites that influence the physiological and biochemical processes in a beneficial manner. Dietary fibre is the best known of the “colonic foods” and is divided into soluble and insoluble fibre. Soluble fibres include pectin, guar gum, B-glucan, and psyllium, and result in modest reductions in blood lipids affecting total and LDL cholesterol fractions. Insoluble fibres (cellulose and lignin) are mainly responsible for faecal bulking. Dietary fibre may play a protective role in diverticular disease and colorectal cancer. Other functional foods are the fructans and resistant starch, which, in animal models, affect triglyceride rich fractions. A novel and potentially important approach to prevention and therapy of colonic diseases is the concept of prebiotics and probiotics. The probiotic approach involves adding live microorganisms to the gastrointestinal tract while prebiotics enhance certain components of the existing flora. Probiotics have potential in the prevention and treatment of rotavirus diarrhoea, lactose malabsorption, and food allergy. Tentative claims for benefits of prebiotics include reduction in obesity, improved control of non-insulin dependent diabetes, reduction in the risk of atherosclerotic cardiovascular disease, and prophylaxis of acute gastroenteritis.

How does the above affect individuals? It seems that we should include the following foods in our diet: garlic, onions, asparagus, chicory, dandelion, artichokes, soy beans, leeks, Jerusalem artichokes, wheat, bananas, and rye. Quite a tall order!

The authors have made an important contribution to the concept of local and systemic effects of prebiotics for the patient’s health and improvement of a healthy colon and the evolution of the idea that functional foods have prophylactic and therapeutic properties. A minor criticism is that there is repetition of ideas in certain chapters. I would highly recommend this excellent work for gastroenterologists as a seminal study. This book should be used as a guide not only for gastroenterologists, primary care physicians, and nutritionists but also for all health workers.
CORRECTION


**P98**

**AN INVESTIGATION INTO THE IMPACT OF ALGINATES AND EPIDERMAL GROWTH FACTOR ON ENDOCYTOSIS-A STUDY IN FOUR OESOPHAGEAL CELL LINES**

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**Introduction:** Endocytosis is a process whereby eukaryotic cells take up extracellular material by a variety of different mechanisms. These endocytic functions are of great importance and are involved in the regulation of cell surface receptor expression, maintenance of cell polarity, cholesterol homeostasis and a host of other physiological processes. In this investigation we looked specifically at fluid phase endocytosis and the impact alginates and epidermal growth factor (EGF) have on this activity.

**Background:** Alginates are extracted from seaweed with their structure and properties related to the type of seaweeds. They are carbohydrate polymers made up of D-mannuronic (M block) and L-guluronic (G block) acid residues, and may also be made up of sequences of mixed residues (MG blocks). These carbohydrate polymers appear to promote migration and restitution in gastrointestinal epithelial cells in *vivo* and in *vitro* by modulating the expression and functional activity of cell junctional proteins such as the E-cadherin-catenin complex. EGF is a 6kd polypeptide that has a role in tissue repair, cell proliferation, ulcer healing and cell migration. EGF also inhibits acid production and imparts a cytoprotective mechanism protecting the oesophageal mucosa from gastric refluxate.

**Methods:** In this study we have used four oesophageal carcinoma cell lines, 2 squamous cell carcinomas and two adenocarcinomas. Cells were incubated with combinations of fluorescent microspheres (0.02μm), alginate and EGF for 1 hour, and then analysed by FACScan®. Alginates were used at a concentration of 2mg/ml and EGF at 10ng/ml.

**Results:**

- All alginates used in this study up-regulate fluid phase endocytosis.
- EGF up-regulates endocytosis.
- Incubation with EGF and alginate up regulates fluid phase endocytosis.
- Levels of up-regulation varied depending on alginate used.
- Alginates up-regulate fluid phase endocytosis more than physiological levels of EGF.

**Conclusions:** We have shown that both alginates and EGF up-regulate fluid phase endocytosis in all cell lines used in this study. However alginates up-regulate this process significantly whereas EGF does not. The mechanism for this alginate action is not yet identified, but it is possible that alginates interact with the receptor for EGF.

9th Asian Conference on Diarrheal Diseases and Nutrition

This meeting will be held on 28–30 September 2001 in New Delhi, India. The organisers hope the meeting will promote meaningful and effective collaboration among individuals/institutions towards control of the major health problems in Asia, particularly those affecting women and children. Further information: Professor M K Bhan, Coordinator, Centre for Diarrheal Disease and Nutrition Research, All India Institute of Medical Sciences, New Delhi. Tel: +91 11 6963822; fax: +91 11 6862662; email: ascodd2001@rediffmail.com

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Notes

Summer Abdominal Imaging Conference

A five day course designed for the practising radiologist with a primary interest in abdominal imaging, emphasising the most recent advances in helical CT, MRI, US, and gastrointestinal imaging. It will be held on 23–27 July 2001 in Banff Springs, Canadian Rockies. Twenty-five category 1 credit hours. Further information: Janice Ford Benner, University of Pennsylvania Medical Center (Radiology), 3400 Spruce Street, 1 Silverstein Building, Philadelphia, PA 19104, USA. Tel: +1 215 662 6904; fax: +1 215 349 5925.

Postgraduate Gastroenterology

A course designed for consultants and registrars, including those who do not specialise in gastroenterology, will be held on 9–12 September 2001 in Oxford, UK. Further information: Professor DP Jewell, Gastroenterology Unit, Radcliffe Infirmary, Woodstock Road, Oxford OX2 6HE. Tel: +44 (0)1865 224829; fax: +44 (0)1865 790792; email: derek.jewell@ndm.ox.ac.uk; website: www.medicine.ox.ac.uk/gastro/gastrocourse.htm

British Association for the Study of the Liver

The 2001 BASL meeting will be held on 13–14 September in London, UK. Further information: Jackie Carter, Centre for Liver Research, University of Newcastle, Floor 4, William Leech Building, Medical School, Framlington Place, Newcastle upon Tyne NE2 4HH, UK. Tel: +44 (0)191 222 5640; fax: +44 (0)191 222 0723; email: j.a.carter@ncl.ac.uk

Torino-Toronto First Joined Workshop on Therapeutic Endoscopy

This workshop will be held on 13–15 September 2001 in Turin, Italy. Further information: Anna Botto, MAF Servizi, Congress Division, Via GB Vico, 7, 10128 Turin, Italy. Tel: +39 011 505 900; fax: +39 011 505 976; email: abotto@mafservizi.it

EACP Scientific and Second Annual General Meeting

The European Association of Coloproctology will hold this meeting on 14–15 September 2001. Further information: Lindsey Whitehouse, Integrity International Event Services, Conference House, 152 Morrison Street, Edinburgh EH3 8EB, UK. Tel: +44 (0)131 200 6055; fax: +44 (0)131 476 4646; email: enquiries@integrity-events.com; website: www.eacp.org

Asia Pacific Digestive Disease Week

The inaugural APDW will be held on 23–27 September 2001 in Sydney, Australia. This meeting will include a live endoscopy workshop, a wide range of other workshops, and a comprehensive scientific programme including original research and clinical symposia. Further information: Conference Secretariat, Gastroenterological Society of Australia, 145 Macquarie Street, NSW 2000, Australia. Tel: +61 (0)2 9256 5454; fax: +61 (0)2 9241 4586; website: www.gesa.org.au

RETRACITION

The authors of abstract number 071 of the BSG Annual Meeting abstract book (Gut 2000;48(suppl I):A20) would like to publish a retraction. This is due to the discovery of an error in the data presented which changes the conclusions of the abstract. The potential error was of a technical nature which the authors were unable to resolve until the return of specific technical support to the laboratory.

The authors have found that their genotyping of the IL-10 polymorphism is inverted on what was presented in the abstract, meaning that the association of ulcerative colitis is with the high producing IL-10 allele. They were only recently able to confirm this by direct DNA sequencing.

The authors would like to apologise for the error.