INVESTIGATION INTO THE EFFECTS OF FK506 (A NOVEL ADENOSINE-1 RECEPTOR ANTAGONIST) IN CIRRHOTIC PATIENTS WITH ASCITES: A PILOT STUDY. 

A22 Gut 1996; T86

Paediatric nutrition T86–T87

Efficacy and Safety of Supplementary Nasogastric Feeding in Children Undergoing Bone Marrow Transplantation A Papadopoulou, A McDonald*, MD Williams**, PJ Darbyshire*, JW Booth Institute of Child Health, University of Birmingham and *The Children’s Hospital, Birmingham

Nutritional insult following bone marrow transplantation is complex and its nutritional management challenging. Enteral nutrition (EN) is cheaper and easier to provide than parenteral nutrition, but its tolerance and effectiveness in reversing nutritional depletion following BMT is poorly defined. We therefore prospectively assessed nutritional status, well being and nutritional biochemistry in 21 children (mean age 7.5 years; 14 males) who received nasogastric feeding following BMT (mean duration of 17 days) and in 8 children (mean age 8 years, 4 males) who refused enteral nutrition and who received dietetic advice only. Results: Enteral nutrition was stopped prematurely in 6 patients. Greater increases in weight (p=0.005), and mid arm circumference (p=0.01), were observed in the EN group, while positive correlations were found between the duration of feeds and improvement in weight (r=0.75; p<0.0001), and in mid arm circumference (r=0.74; p<0.0004). Vomiting, diarrhoea and fever were not more frequent in the EN group, while febrile episodes and diarrhoea tended to have a quicker duration than in the dietetic counselling group (p=0.05 and p=0.06, respectively). Diarrhoea occurring during EN was not associated with fat malabsorption, but carbohydrate malabsorption was associated only with rotavirus infection. However, enteral feeding did not affect bone marrow recovery, hospital stay, the general well-being of patients, or serum albumin concentration. Hypophosphataemia, zinc and selenium deficiency were common in both groups. In conclusion, enteral nutrition when tolerated is effective in reversing nutritional insult following BMT. With existing regimens nutritional biochemistry should be closely monitored in order to provide supplements when required.

Basic science T88–T101

Gastrin Precursors Are Present in Adenomas in the Mouse Polyposis Coli Model – APC17

A. Smith1, S. A. Wains2, J. Cook3, P. Clarke4, J. C. Mather5, R. Fodder4, A. Varo6, J. D. Hardcastle6

1 Dept. of Surgery, University of Nottingham, 2 Dept. of Biological and Nutritional Sciences, University of Newcastle, 3 University of Leiden, 4 Dept. of Physiology, University of Liverpool

The hormone gastrin is now a well recognised growth factor for colorectal adenocarcinomas. The gastrin gene has been shown to be activated in colorectal tumour cells, and precursor gastrin species have been identified. The precursor gastrin-17/acidic-17/gly-17/G17 has been shown to have a proliferative effect, and it has been postulated that it acts via an autocrine growth loop. The mouse model of polyposis coli, APC1638N, which contains a stable mutant APC gene, leads to the spontaneous production of intestinal and extraintestinal tumours, similar to the phenotype seen in human familial adenomatous polyposis. The aim of this study was to evaluate the role of gastrin precursors in the adenoma-carcinoma sequence.

METHODS. In this study mice were fed a high fat Western style diet. At termination the mice were examined and samples were taken from normal colonic mucosa and neoplasms which ranged from adenomas with moderate dysplasia to carcinoma-in-situ. Specimens were formalin-fixed, paraffin sections were made and stained with polyclonal antisera directed against progastrin, acid gastrin-17 and amidated G17. Binding of the primary was detected using the avidin-biotin complex technique. The samples were assessed for site and degree of staining.

RESULTS. Progastrin was present in all the neoplastic samples and 7/8 normal mucosa. The intensity of staining of progastrin was greater in the neoplastic samples than in normal samples. In addition, in normal mucosa progastrin was confined to the cytoplasm of epithelial cells compared to widespread cytoplasmic staining and, in other cases, a granular and luminal edge staining, indicative of secretion. Gly-G17 was present in 9/10 neoplastic samples in the cytoplasm and stroma but was absent in normal mucosa. No normal amidated gastrin was found in any sample.

CONCLUSIONS. There was evidence of over expression and secretion of progastrin and de novo expression and secretion of gly-G17 in neoplastic tissue from adenoma to carcinoma-in-situ. This indicates that progastrin/gly-G17 mediated autocrine pathways may be early events in the adenoma-carcinoma sequence. Furthermore this model may be useful in assessing the efficacy of novel anti-gastrin therapies such as the immunoconjugate, Gastrimmune in pre-malignant conditions.

This study was funded by Aplton Corporation, California, USA.
INTERACTIONS BETWEEN GASTRIN AND TGFa-MEDIATED AUTOCORBINE PATHWAYS IN COLORECTAL CANCER

It is postulated that an autocrine network exists in colorectal cancer mediated by gastrin (precursor forms) and TGFalpha. The aim of this study was to correlate precursor gastrin and gastrin/CCKB receptor expression with EGF/TGFalpha and EGF receptor expression in a series of colorectal tumours.

Tumour samples (n=51), normal adjacent to tumour (n=26) and normal from non-malignant sources (n=5) were examined by immunohistochemistry using antibodies directed against progastrin, glycine-extended gastrin-17 (GlyG17) amidated G17, the gastrin/CCKB receptor (GRP), EGF, TGFalpha and the EGF receptor (EGFR). Primary antibody binding was detected with the avidin-biotin complex method. Intensity of staining was scored on a scale from 0 to 6 in a blinded assessment.

Results: Tumour samples with high levels of normal mucosa adjacent to the tumour. Normal mucosa from a non-malignant source stained weakly for progastrin and EGFR but no other parameter. Correlations were made between individual parameters. Expression of progastrin and GlyG17 correlated with GRP (p<0.01) for tumours, p<0.05 for adjacent normal mucosa. TGFalpha expression correlated with progastrin, GlyG17 and GRP for tumours but not adjacent normal mucosa (p<0.01, 0.05, 0.05, respectively).

In conclusion TGFalpha immunoreactivity correlates with parameters associated with the gastrin- mediated-autocrine pathway in tumours but not adjacent normal mucosa. Colorectal mucosa adjacent to the tumour expressed GlyG17, GRP and TGFalpha which were not expressed in normal colorectal mucosa from non-malignant sources. Thus gastrin and TGFalpha autocrine pathways may act cooperatively in colorectal tumour growth and may be early events in the adenoma-carcinoma sequence.

FREQUENT ABNORMAL EXPRESSION OF E-CADHERIN AND CATENINS, BUT INFREQUENT TRUNCATION OF THE APC PROTEIN IN GASTRIC CARCINOMA CELL LINES

Results: To examine the expression of E-cadherin, a, b and cy-catenin, p120 and APC in gastric carcinoma cell lines, and to look for evidence of mutations and disruption of the cadherin-catenin complex.

Methods: Expression of E-cadherin, a, b, and cy-catenin and p120 was examined by immunoprecipitation with antibodies to p-catenin.

Results: Bands of abnormal molecular weight suggesting mutations, were detected for E-cadherin (in Kato3 and MNK45), and b-catenin (in HSC38), while both E-cadherin and cy-catenin were absent in AGS. One gastric cell line (Kato3) had abnormal APC expression, and showed increased intensity of the b-catenin band. These mutations were associated with loss of localisation of complex components from the cell membrane, and loss of calcium dependent aggregation. Immunoprecipitation of b-catenin-associated proteins revealed loss of b-catenin binding to a-catenin in HSC39, and to p120 in MNK7, HSC39 and Kato3, suggesting functional disruption of the complex.

Conclusions: Mutations of E-cadherin, a- and b-catenin, were associated with abnormalities of E-cadherin-catenin complex composition, loss of membranous localisation to the adherens junction and loss of calcium-dependent aggregation in 4 gastric carcinoma cell lines. Truncation of APC protein was detected in one cell line with apparent increased beta-catenin expression.

EDIBLE MUSHROOM (Agaricus bisporus) LECTIN, A CELL GROWTH INHIBITOR, STOPS GROWTH OF HT29 COLON CANCER CELLS IN G, AND DECREASES THE EXPRESSION OF c-myc

Purpose: The Thomsen-Friedenreich (TF) antigen (GaBI-3GalNAc) is a common oesophageal carbohydrate antigen in intestinal epithelium. Our previous work has shown that the non-cytotoxic TF-binding lectin from the edible mushroom Agaricus bisporus (ABL) inhibits proliferation in a range of normal and malignant epithelial cells (Cancer Res. 1993;53: 4627) and has to be internalized to produce its inhibitory effect (Gastro.1995;108(4):A558). The present study was designed to assess the relationship of this inhibitory effect by ABL to cell cycle and the expression of protooncogenes c-myc and p53.

Methods: 1) HT29 colon cancer cells were cultured in Dulbecco’s Modified Eagle’s Medium (DMEM) with 5% fetal calf serum (FCS). The cells were partially synchronized by culturing in 0.5% FCS for 2 days. ABL (80/20mg/ml) or PBS (control) was added for 8 hours at 37°C. 4% FCS was added to stimulate cell growth. Cells were fixed at various times in ethanol and stained with propidium iodide. Total DNA content per cell was assessed using a FACscan. 2) Subconfluent HT29 cells were preincubated in serum-free DMEM for 1 day before addition of ABL (30mg/ml). RNA was extracted at different times by the guanidinium thiocyanate-phenol-chloroform method. Northern blots were probed with cDNA for c-myc or p53.

Results: After 21 hours addition of FCS, the proportion of ABL treated cells in G, had increased from 81.6% to 90.6% compared with a decrease of 35.0% (n=3) in control. The expression of c-myc MRNA was decreased by 48% (n=3) in the presence of ABL and this effect on c-myc of ABL was abolished by co-incubation with asialo fetuin which expresses GaBl3GalNac. No significant effect on p53 mRNA expression was found.

Conclusion: Mushroom lectin inhibits proliferation by holding cells in G,. The decrease of c-myc mRNA expression may provide a partial explanation for the anti-inhibitory effect.

BIOCHEMICAL AND IMMUNOLOGICAL ANALYSIS OF THE HEAT SHOCK PROTEIN RESPONSE IN HUMAN GASTROINTESTINAL EPITHELIAL, David Boroom, Wendy Hall, Peter E. Ross, John Dillon, and Ted R. Hupp: Department of Biomedical Sciences, University of Dundee, DUNDEE DD1 5SS

INTRODUCTION: Prolonged exposure of oesophageal squamous epithelium to acid, carcinogens, and heat is thought to contribute to reflux oesophagitis and Barrett’s metaplasia, and adenocarcinoma, providing a model for studying the evolution of stress-response and carcinogenesis in this tissue. Reported here is a biochemical and immunological analysis of the heat shock-protein response in human oesophageal biopsies in response to thermal shock.

METHODS: Oesophageal biopsies were subjected to heat shock at 45°C or 55°C for 20 minutes followed by recovery at 37°C for 4 hours. Samples were then lysed for analysis which included: (i) steady state protein synthesis using denaturing SDS-PAGE, (ii) immunohistochemical detection of HSP70 family members, and (iii) rates of protein synthesis by pulse-labeling for four hours post-heat shock with 3H-methionine.

RESULTS: Two types of stress-responses were found in the oesophageal biopsies; one stress occurs immediately following sample collection as defined by a perturbation of the prominent polypeptide associated with normal epithelium and absent in inflamed tissue. The second stress-response occurs after in vitro thermal shock where the state protein synthesis rates and ratios of protein synthesis can be detected for a limited number of polypeptides. In addition, endocytosis of fluorescent microspheres is not perturbed in vitro following severe heat shock (see Hall et al), suggesting that this tissue may have evolved a specific mechanism to maintain cellular function in response to heat fluctuations.

CONCLUSION: These data establish a specific biochemical response of oesophageal epithelium to thermal stress and provides an important model to define the effects of other types of stress, including hypothermia, alcohol, and acid on metabolism. The system also provides a clear foundation for understanding mechanisms of the stress response in oesophageal tissue that are likely to have direct diagnostic and therapeutic implications in future.
ALDOSTERONE REGULATES pH-SENSITIVE APICAL K+ CHANNELS IN DISTAL COLONIC EPITHELIUM BY INFLUENCING Na+-H+ EXCHANGE.

INTRODUCTION: In renal tubular cells, aldosterone increases intracellular pH (pHi) by stimulating Na+-H+ exchange, and simultaneously enhances plasma membrane K+ conductance. Hypokalaemic secondary to distal K+ loading stimulates an active K+ secretory process in rat distal colon, which involves the increased activity of pH-sensitive large conductance (α1β2δG protein) K+ channels in the apical membrane of colonic surface cells (Butterfield & Sandle J Physiol 1995; 489(P, 121P).

Aim and Methods: To evaluate pHi, and its effect on the dynamics of apical K+ channel activity in distal colonic surface cells isolated from control and K+ loaded (8-fold dietary K+ enrichment for 10-14 days) rats, using fluorescence imaging (cells loaded with BCECF) and patch clamp techniques.

Results: Compared with cells from control animals, pHi was greater in cells from K+ loaded animals when bathed in either NaCl solution, pH 7.4 (7.7 (0.05) vs 6.9 (0.03), P < 0.001, n = 3) or Na2SO4 solution, pH 7.4 (7.6 (0.15) vs 7.1 (0.08), P < 0.02, n = 3). The addition of 1 μM ethyl-isopropylamide (EIPA, a specific inhibitor of Na+-H+ exchange), decreased pHi in K+ loaded animals (from 7.7 (0.05) to 7.5 (0.02), P < 0.001). However, in the presence of NaCl solution, and from 7.6 (0.15) to 7.0 (0.06), P < 0.02 (the presence of Na2SO4 solution), but had no effect in cells from control animals. Using cell-attached patches on surface cells isolated from distal colon of K+ loaded animals (Na2SO4 solution bath, K2SO4 solution pipette, zero command voltage), the addition of 1 μM EIPA decreased single channel open probability from 0.92 (0.02) to 0.45 (0.05), P < 0.0001, n = 5), and the effect was fully reversible on washing out EIPA.

Conclusion: Intracellular alkalisation is associated with the increase in apical K+ channel activity seen in surface cells of rat distal colon during hyperaldosteronism secondary to dietary K+ loading, and the rise in pHi is mediated by Na+-H+ exchange.

NITRIC OXIDE REGULATES THE PRODUCTION OF INTERLEUKIN-1 α AND β IN CD BIOPSIES DEMONSTRATED BY SMALL INTESTINAL ORGAN CULTURE.

Beckett C.G., Dell’Olio D. and Ciclitira P.J., Gastroenterology Unit, UMD, St Thomas’ Hospital, London. SE1 7EH.

Aims: In certain immunologically mediated inflammatory conditions IL-1 beta has been shown to play an important role in the regulation of nitric oxide (NO) production. We wished to determine the relationship between NO and IL-1 beta in coeliac disease (CD) by studying jejunal biopsies from patients with this condition using a small intestinal organ culture system.

Methods: Small intestinal biopsies from patients with treated CD and 6 disease controls (DC) were cultured for 18 hours with medium alone (OCM), ovalbumin (OVALB) (1mg/ml), FFIII (1mg/ml). FFIII+LNMMA (1mg/ml) or LNMMA alone. NO production was determined by measuring nitrite (NO2) levels using a colorimetric (Griess) reaction. ELISA kits (Biotek) were used to determine concentrations of IL-1 beta in the culture supernatant.

Results: Medium (range) NO2(μg/ml) and IL-1 beta(ng/ml) in tissue:

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<th>OCM</th>
<th>FFIII</th>
<th>FFIII+LNMMA</th>
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<tr>
<td>CD</td>
<td>170(34)</td>
<td>48(16-148)</td>
<td>0(0)</td>
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<tr>
<td>DC</td>
<td>100(36)</td>
<td>80(0)</td>
<td>0(0)</td>
</tr>
<tr>
<td>IL-1 beta</td>
<td>199(180-360)</td>
<td>3(0-8)</td>
<td>515(9-900)</td>
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<tr>
<td>DC</td>
<td>479(166-636)</td>
<td>265(164-441)</td>
<td>47(58-1270)</td>
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</table>

There was a significant elevation in IL-1 beta in CD biopsies cultured with FFIII compared to OCM(p<0.05) but when cultured with FFIII+LNMMA there was a significant increase in IL-1 beta (p<0.05) compared to OCM or FFIII alone. The CD biopsies cultured with FFIII produced significantly greater NO compared to OCM(p<0.05) and this could be blocked with LNMMA(p<0.01). These changes were not seen in disease controls or with the internal controls of OVALB and LNMMA alone.

Conclusion: Reduced levels of IL-1 beta were found in CD biopsies cultured with FFIII alone. Elevated levels of IL-1 beta were found when CD biopsies were cultured with FFIII+LNMMA, an inhibitor of NO. The findings suggest that NO is involved in the regulation of IL-1 beta production in the small intestine of patients with CD.
T98

**QUANTITATIVE CULTURE OF HELICOBACTER PYLORI FROM GASTRIC JUICE - TRANSMISSION POTENTIAL**

St Bartholomew's and the London Hospital School of Medicine and Dentistry, London, UK.

Introduction. Oral-oral spread of Helicobacter pylori is difficult to study, but it has been suggested that gastric juice, reaching the mouth, may be an important element in spread of the infection. To evaluate the potential of gastric juice in the spread of H. pylori, the presence of viable organisms in this fluid was examined.

Method. Gastric biopsy and gastric juice samples were collected from 64 patients attending for routine gastroscopy. The pH of gastric juice was measured and samples were neutralised with 0.67M Tris (pH 7.4) immediately after collection. Samples were spun at 13000 rpm and the pellet cultured onto Brain Heart Infusion agar containing H. pylori selective supplements. Plates were incubated microaerophilically at 37°C for 7 days. 500μl of pellet was kept for DNA extraction and subsequent polymerase chain reaction (PCR) detection of the ureA gene of H. pylori. The CLO test was performed on gastric biopsy samples.

Results. H. pylori was detected in 50% and 42% of 64 patients by CLO of biopsy and ureA PCR of gastric juice respectively. Culture of H. pylori was achieved from the gastric juice of 6 (9%) patients. Colonies varied in size to biopsy and survived related subculture. Cultures ranged from 1 colony in 15 ml to 2 x 10^5/ml of gastric juice. H. pylori was recovered from gastric juice only when the sample volume was >10ml. 6/8 patients who were positive in both CLO and PCR tests, and from whom >10ml of gastric juice was collected, yielded positive H. pylori cultures.

Conclusion. The demonstration of culturable forms of H. pylori from gastric juice indicates that this fluid could be a factor in transmission. The frequency with which H. pylori positive gastric juice reaches the mouth is unknown.

T99

**TNFα AND LPS INCREASES HUMAN PERIPHERAL BLOOD LYMPHOCYTE ADHESION TO HIMECs WHICH IS PARTIALLY BLOCKED BY MONOCLONAL ANTIBODY TO α5D. THOMPSON, J. WILLIAMS; J. RHODES AND S. L. BLOOM. DEPT. OF MEDICINE, UNIVERSITY OF LIVERPOOL, LIVERPOOL, UK**

**Background** Bacterial products may play a fundamental role in the pathogenesis of inflammatory bowel disease (IBD). A weakened mucoid barrier may allow the influx of bacterial products into the lamina propria which may have a direct effect on endothelium and result in increased lymphocyte recruitment. Using Human Intestinal Microvascular Endothelial Cells (HIMECs), we measured the effect of TNFα, butyrate, lipopolysaccharide (LPS) and F-Met-Leu-Phe (FMLP) on the adhesion of 3H-Chromium labelled human lymphocytes.

**Methods** Mononuclear cells from the peripheral blood of healthy volunteers (n=3) obtained from patients undergoing resection for colonic cancer or IBD were used to isolate HIMECs, as described (Gut 38/4: A635 1996). HIMECs were plated onto 24 well plates, grown to confluency and incubated with butyrate, LPS, FMLP or TNF α. Peripheral blood lymphocytes (PBLs), from healthy volunteers, were isolated using 'Lymphoprep' and labelled with 111Cr/. The PBLs were incubated with anti-α5 (Serotec), a lymphocyte integrin, and incubated with the HIMECs for one hour. Gamma counts in the supernatant, the standardised washes and the mononuclears, removed by detergent, were used to calculate percentage lymphocyte adhesion. 

**Results** The adhesion of PBLs to nonstimulated HIMECs monolayers was 27% (SD=3) (x3 wells). Preincubation with TNFα (10ng/ml) and LPS (100ng/ml) increased this to 74% (SD= 4 p<0.01 Mann Whitney) and 52% (SD =9 p<0.05) though various concentrations of butyrate and FMLP had no effect. Preincubation with Anti α5 reduced adhesion by 25% (p<0.05) on TNFα and 15% (p<0.05) on LPS stimulated cells.

**Conclusions** TNFα and LPS have a direct effect on HIMECs which results in increased PBL adhesion. Preincubation with antibody to α5 reduced adhesion significantly but not substantially suggesting that other molecules or mechanisms may facilitate adhesion of PBLs to TNFα and LPS stimulated endothelium.

T100

**CLOSTRIDIUM DIFFICILE TOXIN A INDUCES PRODUCTION OF TRANSFORMING GROWTH FACTOR ß (TGFß) BY INTESTINAL EPITHELIAL CELLS. S. MARK*, S. HYDE, S.P. BORRITTELL*, Y.R. MAHIDA. DEPARTMENT OF GASTROENTEROLOGY & INSTITUTE OF INFECTION & IMMUNITY, UNIVERSITY HOSPITAL NORTHERN, LIVERPOOL, UK**

TGFß accelerates wound repair and enhances epithelial cell "restoration". C. difficile toxin A induces severe intestinal inflammation. In vitro, high concentrations of toxin A induce production of interleukin-8 by human intestinal epithelial cells. We therefore measured the effect of toxin A production by a human intestinal epithelial cell line exposed to toxin A.

**Methods.** Confluent monolayers of T84 cells were exposed to 10,1000 and 10000ng/ml of purified toxin A for 3h. After washing off the toxin, monolayers were cultured in medium for 24h. Total TGFß present in the supernatants was assayed after acidification (and subsequent neutralisation) of the supernatants. Acid-unreacted activators were also assayed to determine the amount of biologically active TGFß present. TGFß bioassays were performed in triplicate using Mvl1u cells.

**Results.** Mean±SDng/ml total TGFß produced in 3 experiments:

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<th>II</th>
<th>III</th>
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<tr>
<td>10ng/ml toxin A</td>
<td>177(±47)</td>
<td>324(±47)</td>
<td>90(±55)</td>
</tr>
<tr>
<td>100ng/ml toxin A</td>
<td>87(±93)</td>
<td>112(±64)</td>
<td>78(±288)</td>
</tr>
<tr>
<td>1000ng/ml toxin A</td>
<td>256(±122)</td>
<td>927(±470)</td>
<td>712(±259)</td>
</tr>
<tr>
<td>10000ng/ml toxin A</td>
<td>198(±45)</td>
<td>508(±64)</td>
<td>390(±51)</td>
</tr>
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</table>

Control vs 10ng/ml toxin A: p<0.001.

Studies with acid-unreacted supernatants showed that majority of the TGFß released in response to toxin A is in the biologically active form. Culture (in medium only) of toxin A pre-exposed cells (at 10 & 100ng/ml) for a further 24h period also induced production of TGFß.

**Conclusions.** Low concentrations of C. difficile toxin A induced production of TGFß. Such a response was not prominent at the highest concentrations of toxin A, which induces production of interleukin-8. Thus, epithelial cell exposure to large amounts of toxin A may lead to an inflammatory response whereas small amounts of the toxin may allow the colonic mucosa in vivo to heal rapidly via the action of TGFß.
SMALL INTESTINAL T CELL ACTIVATION IN TROPICAL ENTEROPATHY. AM Vaitich, P Kelly, JOM Poboe, S Segal, SK Spies, MG Farthing. Digestive Diseases Research Centre, St Bartholomew's & the Royal London School of Medicine & Dentistry, London. 2University Teaching Hospital, Lusaka, Zambia, and Gastroenterology Divisions of the Universities of Witwatersrand and Pretoria, South Africa.

Introduction and aims Tropical enteropathy has been described in the healthy indigenous populations of several African countries. The mechanisms underlying the small intestinal mucosal changes in this syndrome remain unclear. T cell activation has been demonstrated to result in villous atrophy and crypt hyperplasia, and we therefore investigated whether this process has a role in the pathogenesis of tropical enteropathy.

Patients and methods 10 black Zambian subjects, 20 black South African subjects, and 22 white South African subjects undergoing routine upper GI endoscopy for dyspepsia (GU, DU, diarrhoea and AIDS excluded) were studied. Distal duodenal villous height (VH) and crypt depth (CD) was measured by computerised image analysis of formalin-fixed paraffin-processed biopsies. Snap-frozen duodenal biopsies were subjected to dual colour immunofluorescence staining for CD69/CD3, allowing quantitative assessment of T cell expression of the activation marker CD69.

Results Median VH was lower (308 (185-402) mm) vs 412 (235-450) mm p < 0.05, and CD greater (190 (141-206) vs 154 (119-180) mm p < 0.05), in Zambians compared to black South Africans. There was no significant difference in VH or CD between black and white South Africans. Median percentage CD69/CD3 was greater in Zambians compared to black South Africans (80 (63-100) vs 60.3 (33-86) p < 0.001) but while South African CD69 expression (66.5 (35-95.5)) was not significantly different from that of black South Africans.

Conclusions Tropical enteropathy is present in the black Zambian population but not in the relatively less deprived urban black South African population. Tropical enteropathy is associated with small intestinal mucosal T cell activation.

Endoscopy and oesophagus T102–T116

WIRELESS TRANSMISSION OF A COLOUR TELEVISION MOVING IMAGE FROM THE STOMACH USING A MINIATURE CCD CAMEL-LIGHT SOURCE AND MICROWAVE TRANSMITTER.

CP Swain, F Gong, TN Mills. GI Science Research Unit, Royal London Hospital, London and Department of Medical Physics, University College London.

An experimental endoscope was constructed using a miniature CCD camera, video processor, light source, microwave transmitter and battery. A microwave receiver was positioned approximately 50 cm from the endoscope and connected to a colour video monitor. Using this arrangement, high quality colour moving television images were transmitted from a model stomach to the external receiver without wires, fibre optic bundles or cables. The ability of this system to transmit moving images through the abdomen was tested by placing the device in a box behind a volunteer’s back and the receiver in front of his abdomen. In other experiments the “endoscope” was placed inside the mouth and again good quality images were received. In separate experiments the endoscope was placed inside post-mortem stomachs and transgastric moving television pictures were transmitted without wires, cables or fibre optic bundles.

Video signal transmission was accomplished using a miniature 10 mW microwave transmitter operating at 10.3 GHz. The light source was a miniature halogen lamp. The miniature video camera, integral controller, transmitter and lamp were powered by small low voltage batteries.

Conclusion: these experiments demonstrate the feasibility of constructing a new type of endoscope which can transmit high quality moving colour television images from the gastrointestinal tract without requiring fibre optic or electrical cables to be passed through the mouth or anus.

COMPARISON OF SMALL BOWEL RADIOLOGY AND PUSH ENTEROSCOPY IN THE INVESTIGATION OF SMALL INTESTINAL PATHOLOGY.

LY Yiannakou, Colette Hawkins, C Garvey, A.J.Morris. Gastroenterology Units, St. Thomas' Hospital, London & *The Royal London University Hospital, Liverpool.

Introduction & Methods: Push enteroscopy (PE) is a promising technique for visualisation, biopsy, and therapy of the proximal small intestine and early results of its efficacy are encouraging. We have compared this technique with small bowel radiology (SBR) in 41 patients where there was a strong suspicion of small intestinal disease. Indications for investigation were: anaemia (11), overt bleeding (8), diarrhoea (7), abdominal pain (3), diarrhoea with abdominal pain (5), and polyposis syndromes (3). Twenty patients had small bowel meals and 21 small bowel enemas (enteroclysis). PE was performed under sedation using the Olympus SIF10 endoscope. All procedures were well tolerated.

Results: Seventeen patients (42%) were found to have small intestinal abnormalities. These were detected by both modalities in 4 patients (jejunal tumour, lymphangiectasia, and 2 cases of Crohn's disease), by PE alone in 7 cases (6 cases of angiodysplasia and one case of coeliac disease), and by SBR alone in 6 cases (jejunal tumour, jejunal ulcer, jejunal stricture, and 3 cases of ileal Crohn's disease). The 3 jejunal lesions missed by PE were all in the distal jejunum. In patients with Crohn's disease SBR was superior at showing extent of disease, but PE allowed biopsy to assess disease activity. PE was used to provide laser photocoagulation to 4 lesions that were actively bleeding and this was effective in all cases.

Conclusions: PE will often identify lesions missed by SBR, and vice versa. When lesions are identified by both techniques the information obtained is additive. SBR is more effective for distal disease and assessing extent of disease. PE allows mucosal visualisation and is particularly useful for identifying angiodysplasia. It also allows biopsy and treatment of any lesions detected. PE and SBR are therefore complimentary and should be used together in the investigation of small intestinal disease.

MEASUREMENT OF INSERTION DEPTH DURING FLEXIBLE SIGMOIDOSCOPY(PS) OR COLONOSCOPY BY ELECTROMAGNETIC IMAGING(EMI) : IMPLICATIONS FOR COLORECTAL CANCER SCREENING.

Bell GB, Saunders B, Williams CB, Pitt R, and Bladen J. Endoscopy Units of the Ipcwh and St Mark’s Hospitals.

Introduction

Approximately 70% of all colorectal cancers are situated distal to the splenic flexure(SF). Previous attempts have been made to assess depth of insertion of a 60cm FS using either anatomical landmarks or a single plain abdominal X-ray both of which can be unreliable.

Methods and Results

We have used EMI which in real time gives information regarding scope position, configuration and depth of insertion (Lancet 1993;341:719-722. Gut 1995;36:913-917). Retrospectively using stored images we have examined 20 patients who had a FS and 84 who underwent colonoscopy. When the endoscope had been inserted to 60cm we checked the tip position. In 41.4% cases the scope was still in the proximal sigmoid, in 20.2% the tip had only just passed or just beyond the sigmoiddescending colon junction, in 5.6% it was in mid descending colon, in 11.5% it had reached the splenic flexure while in 17.3% it was in the distal or mid transverse colon.

Conclusion

Using a 60cm FS the splenic flexure is reached (or passed) in less than 30% of cases. Longer instruments or better ways of preventing loop formation of existing length scopes are required to optimise this screening method.