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Systemic factors and luminal contents in gut adaptation

SIR,—I read with interest the elegant paper by Albert *et al* (*Gut* 1990; 31: 311–6). They state that the atrophic effect of diversion of luminal contents can be counteracted by systemic growth factors released as part of the adaptive response and, accordingly, systemic growth factors are not dependent on a permissive effect of luminal contents.

We used a different strategy to address a similar question.¹ The behaviour of jejunal to colonic mucosal autografts was studied in an experimental animal model of short bowel syndrome (SBS). Histological appearances, enterocyte enzyme activities, and *in vitro* glucose transport were studied at the donor and recipient graft sites in control, SBS, and gastrocolic fistula 5 week old 150 g Sprague-Dawley rats. Small intestinal function was maintained in the jejunoileal graft after 80% small bowel resection; animals in which small bowel was not resected showed loss of graft function and enzyme activity. Total parenteral nutrition did not alter graft behaviour but improved the postoperative mortality of the procedures. In addition, after creation of a gastrocolic fistula, the jejunum to colon graft lost functional jejunal activities. This may be interpreted in one of two ways. Either jejunal chyme and pancreaticobiliary secretion is a prerequisite for adaptation, as has been widely proposed previously,^{2,3} or the same explanation may be given as proposed by Albert *et al* in their paper. This dual interpretation also applies to their study: it may, therefore, be premature to discount the importance of luminal nutrition.

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Reply

SIR,—The results of our study indicated, as stated by the correspondents, that the action of systemic growth factors released in response to the short bowel syndrome, are not dependent on the presence of luminal contents: they induce proliferation in bypassed loops where neither nutrients nor pancreaticobiliary secretions are present. This conclusion should not be misinterpreted to mean that we consider that luminal nutrients or pancreaticobiliary secretions, or both, are unimportant in other situations. We agree that the importance of luminal contents in the adaptive process should not be discounted. However, such factors could not have been responsible for the proliferative response seen in the bypassed loop in the rats subjected to 85% jejunoileal bypass. Previous studies have indicated the presence of circulating growth factors in the short bowel syndrome, but it has remained possible that these are simply due to an epiphenomenon related to leakage of locally acting growth factors into the circulation. Our findings suggest that they play an important, and not a minor secondary, part.

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Why do patients with ulcerative colitis relapse?

SIR,—When dedicated research workers spend much effort employing methodology bound to fail in its objective it is always regrettable. In this instance I refer to reliance on self reported answers by ulcerative colitis sufferers to questionnaires about their emotional feelings and allegedly stressful life events reported in the article by Riley *et al* (*Gut* 1990; 31: 179–83).

Given the degree of unanimity of view expressed by critics of the above methods,^{1–5} and of the comparable futility of dependence on only one or two interviews to uncover emotional factors^{6–12} in inflammatory bowel disease, it is surprising that the authors of the article did not discuss these criticisms, or even refer to them.

It is now 40 years since I commented on these emotional aspects in a large series¹³: 'Colitis patients may be ready talkers about their symptoms but they are "dumb" about their emotional feelings.' Suggestions were then offered on how they could be helped to do so. McMahon *et al.*,¹ in criticising the insensitivity of studies by Feldman *et al.*¹⁴ and Mendeloff *et al.*,¹⁵ wrote that 'they view psychopathology largely in symptomatic terms, and rely heavily on patients' self-reports,' and added that 'patients with inflammatory bowel disease are notoriously poor reporters of their inner psychic life.' They also cited Schmitt,² who wrote that 'it is not surprising that ulcerative colitis patients [referring to Mendeloff's study] report fewer stressful life events in their lives' and 'the patients' extensive use of denial suggests that they are often unaware of the stress at a conscious level.' Ford and coworkers,⁷ writing of comparable experience of regional enteritis, said that 'it should not be surprising if patients with a degree of rigidity, repression and guardedness noted in our study were to answer negatively to direct questions about emotional stress' and 'before these very guarded patients can allow themselves to talk freely a relationship must be established.' Summarising the serious limitations of inventories and questionnaires in the investigation of all psychosomatic disorders, Pelsner and I¹ have said: 'It is inevitable that questionnaires/inventory techniques will always fail to uncover deeply repressed and unconscious feelings in patients whose very disability centres on their poverty of emotional expression.'

In an attempt to be constructive I would respectfully suggest that the answer to Riley *et al.*'s question is open to any clinician who is prepared to acquire the sensitive interviewing techniques needed to uncover the typically provocative emotional stresses in these guarded patients, and to recognise that the process is likely to entail interviews over many weeks.^{1,3,6,7}

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Reply

SIR,—Dr Paulley criticises our use of self rated questionnaires in patients with ulcerative colitis as he believes that colitis 'centres on a poverty of emotional expression.' Dr Paulley and others^{1–4} have for many years championed the psychosomatic model of ulcerative colitis. Their observations are extensive but, as all are uncontrolled, we would respectfully suggest that they are of limited value. Controlled studies have consistently failed to show an excess of psychiatric illness or stressful life events in patients with colitis.^{5–8}

The rating scales we used have been well validated in both healthy subjects and patients with psychiatric illness.^{9–11} There is no objective evidence to suggest that patients with colitis have difficulty expressing their feelings through such scales.

In our experience most patients with ulcerative colitis will openly discuss psychological conflicts, and, despite our negative findings, many believe that stressful life events precipitate relapse. However, rumination, by both patient and doctor, is particularly common at times of disease relapse. Appropriately controlled studies are, therefore, essential if such biases are to be overcome.

In order to investigate events that may precipitate colitis relapse we studied a cohort of patients in established disease remission. Patients were assessed at 12 weekly intervals and were followed up to either relapse or completion at 48 weeks. The main advantage of such a study is that the patient serves as his or her own control. Using such a design we found that the frequency and severity of life events were equally matched in patients in relapse and remission and that those who relapsed graded life events no more stressful than those who stayed in remission. Anxiety and depression ratings were also similar in the two groups and did not correlate with disease activity.

We did not assess personality characteristics in our patients, although personality differences from controls are often cited to support the role of psychological factors in the patho-