
Reply

Editor,—We thank Dr Arienti and colleagues for their attention to our work. It is correct that improved technology and operator experience alone do not explain our better results. Indeed, the higher accuracy of transabdominal sonography in our study is based principally on the use of revised definitions for the detection of intestinal complications. It is, therefore, a pleasure to have consensus on these definitions.

Despite some unresolved issues, many (mostly European) investigators have shown the value of bowel sonography in patients with Crohn’s disease. The time is ripe to offer the benefits of this imaging method to patients with Crohn’s disease worldwide.

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Association between colon cancer and adenocarcinoma of the oesophagus

Editor,—Recently, Lagergren and Nyren (Gut 1999;44:819–821) concluded that results from a population based cohort study in Sweden did not support a common link between colon cancer and oesophageal adenocarcinoma. However, there is consistent evidence that low intake of dietary fibre is associated with both diseases. In an analysis of 13 case control studies involving more than 5000 colorectal cancer cases, Howe and colleagues reported an inverse association between fibre intake and colorectal cancer in 12 of the 13 studies, and an odds ratio of 0.53 (95% confidence interval 0.47 to 0.61) for the highest quintile of fibre intake compared with the lowest, with trend analysis.1 Similarly, four case control studies have reported a significant inverse association between fibre intake and adenocarcinoma of the oesophagus and gastric cardia (table 1).2

In contrast, two studies which included cases of squamous cell carcinoma found no significant link between fibre intake and squamous cell carcinoma of the oesophagus.3 4 Clearly, the dramatic increase in the incidence of adenocarcinoma of the oesophagus in the USA and parts of Europe over past decades cannot be explained by secular trends in dietary fibre consumption. A more plausible explanation links increased rates of colorectal cancer in the USA to increases in the prevalence of obesity.5 6 This view is supported by evidence from observational studies that suggests that both overweight and symptomatic gastro-oesophageal reflux are linked to increased risk.7 8 Possible mechanisms for the observed protective effect of dietary fibre include the mechanical cleaning effect of the lower oesophageal mucosa, increased motility of potential carcinogens across the gastro-oesophageal juncture.

Table 1 Dietary fibre intake and adenocarcinoma of the oesophagus and gastric cardia

<table>
<thead>
<tr>
<th>Reference</th>
<th>Country</th>
<th>Sites</th>
<th>Comparison</th>
<th>Odds ratio</th>
<th>95% CI</th>
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<tbody>
<tr>
<td>8</td>
<td>Odds ratios adjusted for alcohol and tobacco use.</td>
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<tr>
<td>9</td>
<td>*95% confidence interval (CI) does not include 1.0. †Multiple logistic regression model.</td>
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OGC, oesophagi and gastric cardia; O, oesophagus.
tion, and the antioxidant effect of micronutri-
ten in fruits and vegetables. The lack of a significant link between colorectal cancer and oesophageal cancer in the Lagergren and Nyren study is not surprising, as the average year of entry to the cohort study was 1977 and median follow up was 2.1 years. Thus, a substantial proportion of the accumulated person years relates to a time period when the Swedish population was at a very low risk of developing oesophageal adenocarcinoma after a diagnosis of colon cancer remains small, because of the late onset of colon cancer. Furthermore, case control studies are likely to continue to be the most efficient type of observational study design for the investiga-
tion of possible common links between these two diseases.

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The primary hypothesis of our population based cohort study was not that colon cancer would subsequently develop into oesophageal cancer, but rather that there might be a common underlying link between the occurrence to these two tumours, independent of the time latency in their development. Hence, the individual follow up latency after colon cancer diagnosis was of minor importance. Therefore, it would seem reason-
able to assume a latency between exposure to the critical underlying factor, for example, insufficient dietary intake of fibre, and the development of oesophageal or colonic adenocarcinoma. Therefore, as long as selection or ascertainment biases are deemed to be small, the time period that fol-
lows immediately after diagnosis of colon cancer is the most informative. The total number of person years was more critical, and we were able to follow up more than 500 000 person years in our study. The rarity of oesophageal adenocarcinoma is a problem in any study of the aetiology of this tumour in any country, particularly if the studied expo-
sure is relatively rare. This problem explains our limited power to exclude a weak association. Nevertheless, we were able to identify more than 100 000 people with verified colon cancer and to follow them for sub-
sequent cancer development. This is a sub-
stantial number of exposed people. We agree that case control studies are generally more efficient than cohort studies when rare outcomes are to be investigated. However, in the case of our register based retrospective cohort study, a case control approach would not entail any advantage, as our cohort contained all individuals exposed to colon cancer in Sweden during the years 1985, 1988, and 1992, and all individuals among them who devel-
oped oesophageal adenocarcinoma during the same period. A case control study conducted in Sweden during this period would, at best, include the same number of exposed oesophageal adenocarcinoma cases as in our cohort study. Thus, the problem with low statistical power is not owing to study design, but that the study base (all resi-
dents of Sweden 1958–1992) was too small to generate a sufficient number of individuals with the combination of colon cancer and oesophageal adenocarcinoma.

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Diverion colitis as a trigger for ulcerative colitis

EDITOR,—Lim and colleagues recently pre-

presented three cases of diversion colitis which seemed to act as a trigger for instream ulcerative colitis (Gut 1999;44:279–282). We would like to present a fourth case which has recently come to our attention and which adds to the literature.

In 1994, a 75 year old woman presented with a five year history of faecal soiling and urinary incontinence. A former smoker of 50 years, the patient had had a traumatic forceps delivery and episiotomy when she was 36 years old. Her mother died of unspecified colitis. Rigid sigmoidoscopy and barium enema were normal, but anorectal ultrasound showed defects of the internal and external sphincters. Anterolateral repair of the anal sphincter did not control the symptoms and in September 1997 an end colostomy was performed for her disabling faecal incontinence.

In July 1998, the patient presented with blood and mucus per rectum, and an anterior mucosal prolapse was diagnosed and re-
paired. However, her symptoms persisted and in October 1998 flexible sigmoidoscopy showed a granular, congested, and oedema-
tous mucosa with contact bleeding through-
out the rectosigmoid. A histological study showed a mixed inflammatory cell infiltrate with distortion of the crypt architecture and cryptitis, and a diagnosis of diversion colitis was made; the rectal symptoms responded quickly to topical steroid enemas.

Four months later, the patient developed increased stomal frequency and bleeding into the stoma bag. Colonoscopic examination of the in-stream colon, via the colostomy, revealed an active distal colitis with granular, oedematous, congested, and friable mucosa. Histology showed a mixed inflam-

matory cell infiltrate in the lamina propria with cryptitis, crypt abscess formation, and a reduction in the number of goblet cell. Ulcerative colitis was diagnosed and treated with oral mesalazine and topical steroid enema per stoma. Symptoms quickly im-

proved and there has been no further bleeding from the rectum or stoma.

Although the histological features of ulcer-
cative colitis and diversion colitis are indis-
tinguishable, the clinical history in this case suggests that ulcerative colitis developed after true diversion colitis. It is possible that this patient’s colitic process represented an idio-

pathic ulcerative colitis, but it seems much more likely that the colostomy with faecal diversion was the initiating factor and that, as speculated in the cases studied by Lim and colleagues, diversion colitis is a risk factor for ulcerative colitis. Hypotheses on the patho-
genesis of both diversion colitis and ulcerative colitis should take into account cases such as these.
We showed recently that having a child on HPN has a major impact on the quality of life of the parents. We studied 11 parents of children with chronic intestinal failure requiring HPN. Following an initial focus group meeting to identify important issues, semi-structured interviews were held with the parents. The General Health Questionnaire (GHQ-28) and a questionnaire developed for the British Artificial Nutrition Survey (BANS) were also administered. A control group of 11 parents with age matched healthy children also answered the BANS questionnaire.

The GHQ-28 showed that seven of the 11 parents with children on HPN exceeded the threshold for psychiatric morbidity. The BANS described a significant deterioration before and after the child’s illness for social life (p<0.007), family life (p<0.007), sex life (p<0.003), and work (p<0.004) in these parents compared with controls. Parents caring for children on HPN were also more likely to be physically tired and to have difficulties in taking holidays, going shopping and spending life (p<0.007), family life (p<0.007), sex life (p<0.003), and work (p<0.004) in these parents compared with controls. Parents caring for children on HPN were also more likely to be physically tired and to have difficulties in taking holidays, going shopping and spending life (p<0.007), family life (p<0.007), sex life (p<0.003), and work (p<0.004) in these parents compared with controls. Parents caring for children on HPN were also more likely to be physically tired and to have difficulties in taking holidays, going shopping and spending life (p<0.007), family life (p<0.007), sex life (p<0.003), and work (p<0.004) in these parents compared with controls. Parents caring for children on HPN were also more likely to be physically tired and to have difficulties in taking holidays, going shopping and spending life (p<0.007), family life (p<0.007), sex life (p<0.003), and work (p<0.004) in these parents compared with controls. Parents caring for children on HPN were also more likely to be physically tired and to have difficulties in taking holidays, going shopping and spending.
Quality of life of parents of children on home parenteral nutrition

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