Should patients with inflammatory bowel disease smoke?

SIR,—Dr Greg Holdstock and others (4 February, p 362) say that the aim of their study was to determine whether smoking is associated with less severe forms of ulcerative colitis or Crohn's disease. After analysing 172 variables in three groups of patients they present seven significant differences. For three of the differences the significance levels are modest ($p < 0.05$, 0.05 , and 0.04), and in assessing the importance of their results it would be helpful to know just how many comparisons were attempted. Restricting oneself to the eight variables in the published table a minimum of 24 comparisons are possible and therefore at least one "significant" difference might be expected to occur through chance alone.

It would appear also that the Student's t test has been applied to such variables as number of relapses per patient, number of hospital admissions per patient, bowel frequency, and severity of pain. For all these variables the standard deviations quoted indicate that the data are far from normally distributed. We suspect that any differences result from the behaviour of a small minority of patients, who may not be representative of patients with Crohn's disease in general. Our reason for saying this is that the prevalence of the two types of inflammatory bowel disease in their study is quite different from the two to one ratio of ulcerative colitis to Crohn's disease usually found in the United Kingdom.

Dr Holdstock and his colleagues then suggest that because they found a low prevalence of smoking among patients with ulcerative colitis, "Smokers therefore appear to have a reduced risk of developing the disease." This suggestion is plainly unwarranted because they present no evidence about the smoking habits of patients before onset of disease (or indeed any control data). They will find such evidence if they consult their references, in which we show (using control data) that the association does antedate onset of disease.¹

They proceed to suggest that smoking leads to Crohn's disease rather than ulcerative colitis. As they produce no evidence comparing the smoking habits of patients with Crohn's disease with those of controls their speculations are ill founded. Nevertheless, they may be right, for we have found that patients with Crohn's disease are more likely to be smokers than matched controls and the association antedates onset of disease.²

M J S LANGMAN
R F A LOGAN

Department of Therapeutics,
University Hospital,
Nottingham NG7 2UH

¹ Logan R, Edmond M, Langman MJS. Is non-smoking associated with ulcerative colitis? *Gut* 1983;24: A499.

² Logan RFA, Somerville KW, Edmond M, Langman MJS. Is cigarette smoking associated with Crohn's disease? *Gut* 1983;24:A980-1.

*.*The authors reply below.—ED, *BMJ*.

SIR,—Unfortunately, some methodological and other detail was omitted when the paper was changed from a full paper to a short report.

We appreciate the concern of Professor Langman and Dr Logan regarding the

statistical analysis and are aware of the possibility of a type I statistical error. We did attempt to get round this to some degree, however, by using logarithms of true variables with large standard deviations, and we checked the results using the Mann-Whitney test. As stated in the text, 172 variables were analysed. The purpose of our paper was to "ascertain whether smoking is associated with less severe disease." We provided some evidence to suggest that within groups those patients who smoked had more frequent and more severe relapses. Some 69% of those with continuous symptoms were current smokers compared with 20% of those with intermittent symptoms. We therefore cannot agree that our conclusions are not justified. We state, "Our findings, together with the known health hazard of smoking, lead us to conclude that, for the present, patients with inflammatory bowel disease should be advised to stop smoking."

We agree that the ratio of Crohn's disease to ulcerative colitis in our study population is somewhat higher than in most other reports. Nevertheless, the diagnosis was made without knowledge of smoking habits and we do not accept that this would influence our results. This ratio reflects our experience and belief that the incidence of Crohn's colitis is increasing, but we accept that it may indicate differences in diagnostic criteria.

Our study was not designed to examine the possibility that smoking is a factor either protecting against ulcerative colitis or causing Crohn's disease, and this was mentioned only as a possible explanation for our finding in the light of previous reports. We are pleased that the results of Professor Langman's studies support the hypothesis. We believe that studies designed to show the effect of smoking on inflammatory bowel disease should look at the disease as a whole rather than at the individual subgroups.

GREG HOLDSTOCK
D A SAVAGE
RALPH WRIGHT

Southampton General Hospital,
Southampton SO9 4XY

Hypercholesterolaemia and coronary heart disease: an answer

SIR,—The report of the Lipid Research Clinic's coronary primary prevention trial^{1,2} has prompted considerable press comment and was the subject of a recent leading article in the *BMJ* (11 February, p 423) as well as in the *Lancet*.³ The leading articles drew similar conclusions—that "screening" for high cholesterol concentrations was of little value in the general population, as the subjects in the study were drawn from the upper 5% of the cholesterol distribution.

It is obvious that the actual cholesterol value represented by the 95th percentile will vary with the population studied. In the Lipid Research Clinic's trial it was 6.9 mmol/l (265 mg/100 ml), derived from a pilot study of 1364 men aged from 40 to 49 years. Patients attending the BUPA medical centre in London do not fare so well: 6.9 mmol/l represents the 80th percentile of men aged from 35 to 44 years and the 74th percentile in men from 45 to 54 years of age. Our 95th percentiles for the two age groups are 8.0 and 8.2 mmol/l (307 and 315 mg/100 ml) respectively.

As more than one fifth of our patients are

in the high risk group according to the American criterion we would suggest that hypercholesterolaemia, far from being a rare condition, is extremely common among British men. We believe that the possibility of substantially reducing coronary morbidity and mortality in a group of this size justifies efforts to extend screening of cholesterol concentrations to the whole population, rather than limiting it to patients with premature coronary heart disease or the relatives of those known to have hypercholesterolaemia.

CAROLYN D RITCHIE
ALAN BAILEY

BUPA Medical Centre,
London WC1X 8DU

¹ Anonymous. The Lipid Research Clinics coronary primary prevention trial results. I. Reduction in incidence of coronary heart disease. *JAMA* 1984; 251:351-64.

² Anonymous. The Lipid Research Clinics coronary primary prevention trial results. II. The relationship of reduction in incidence of coronary heart disease to cholesterol lowering. *JAMA* 1984;251: 365-74.

³ Anonymous. Is reduction of blood cholesterol effective? *Lancet* 1984;i:317-8.

Guide wire manipulation of Crosby jejunal biopsy capsule

SIR,—I have used a rapid technique for obtaining biopsy specimens from the small bowel that is similar to that described by Dr Robert L Law (28 January, p 286). I use the paediatric Watson capsule with an x ray opaque catheter (No 2513, Surgimed A/S Tubing, Denmark; diameter 1.14-1.58 mm). I find the Teflon coated guide wire too stiff to use in children as I want the wire also to pass into the duodenum, so I use the safety guide wire from William Cook Europe A/S, Denmark (type SF). The wire is positioned in the tube from the beginning, and if the patient is in an upright position when swallowing the tube the capsule usually goes straight towards the pylorus with no coiling of the tube. When infants and young children are in a lying position the capsule often turns towards the fundus of the stomach. In these cases I take it back to the cardia, put the child on his or her right side and twist the tube at the same time as I push it; sometimes it helps to withdraw the guide wire a few centimetres.

During the procedure I screen rapidly every now and then to control the position of the tube and capsule; the x ray field size is kept to a minimum. A firm push takes the capsule through the pylorus. I withdraw the guide wires in the descending duodenum, but sometimes the capsule is already near the duodenojejunal flexure when I screen again. This procedure can be seen on a teaching videotape about small bowel biopsy in children (available from this hospital).

For this procedure I use diazepam 0.4 mg/kg per rectum and pethidine 1 mg/kg intra-

Comparison of time taken to retrieve a biopsy specimen from the small bowel using paediatric capsule and adult capsule

	Time between insertion of tube and retrieval of biopsy specimen (minutes)		Duration of fluoroscopy (minutes)	
	Mean	SD	Mean	SD
Watson paediatric capsule (n = 178)	8.6	3.5	1.0	0.7
Watson adult capsule (n = 21)	12.2	7.1	1.6	1.3