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Histochemical demonstration of desialation and desulphation by faecal extracts

SIR,—We were interested in the paper by Rhodes *et al* (*Gut* 1985; **26**: 1312-8) in which they suggest that ulcerative colitis could be caused by an inherited defect in colonic mucus rendering it susceptible to enzymatic degradation by bacterial enzymes. We have shown that there does indeed exist within the general population heterogeneity in colorectal mucus.¹ We found that 8% of subjects (in a survey of 110 patients) secreted sialic acid lacking O-acetyl substituents when the remaining patients secreted O-acetyl sialic acid. Recently we have noted that the sialic acid lacking O-acetyl substituents shows increased susceptibility to sialidase digestion (unpublished observations). Sialic acid lacking O-acetyl substituents is found more frequently in patients with ulcerative colitis² and we are trying to determine if this is acquired or represents a genetic condition that predisposed to the disease. We wonder if the failure by Rhodes *et al* to differentiate between colitics and controls could be due to an over representation of subjects with the abnormal mucus within their control group.

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Reply

SIR,—The finding by Drs Jass and Sugihara that 8% of normal subjects secrete colonic sialomucins that are less O-acetylated and less sialidase resistant than normal is very interesting and it is certainly plausible that this may be a risk factor for the development of

colitis. It seems unlikely, however, that the 17 control subjects in our study, all of whom had the irritable bowel syndrome and normal rectal histology, should be particularly likely to come from this 8% of the population. Further work is clearly needed to determine the structural and functional abnormalities of colonic mucus in ulcerative colitis.

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Twenty four hour intragastric acidity analysis for the future

SIR,—We read with interest the article by R P Walt (*Gut* 1986; **27**: 1-9) which considers the consequences of expressing an ensemble of acidity data by an index such as the mean or median value of a group of pH or hydrogen ion activity values. While in agreement with much of the article, we believe it may itself propagate further misunderstanding, if we have correctly interpreted the text.

Hourly hydrogen ion activity in a duodenal ulcer patient is presented (Fig. 1) with hourly pH. Mean pH is 1.67, the antilog of which is 21.3 mmoles/litre but the mean hydrogen ion activity is 34.7 mmoles/litre. This discrepancy is considered to be 'an unresolved problem of analysis' because these values 'should be equivalent, as they are derived from the same data'. By equivalent, we assume that the author means numerically identical. But it should be recalled that to average the logarithms of n numbers is to calculate the nth root of the product of those n numbers: this is equivalent to calculating the geometric mean. Averaging logarithms of numbers is not the same process as averaging the numbers themselves. It is well known¹⁻³ that the geometric and arithmetical means will *not* be equivalent. The numerical difference between mean pH and antilog mean hydrogen ion activity (Fig. 5) is therefore to be expected as two quite distinct processes have been carried out to calculate these figures. The numerical difference is only an unresolved analytical problem if it is assumed that none should exist. As a difference does exist, the use of median values to minimise this computational difference then seems to be of doubtful value.

The author seems to have misunderstood a fairly basic point and in doing so has equated the problem of analysis with that of representation. What concerns us is not that the author has apparently erred; everyone makes mistakes. What is puzzling is that the referees, who often exercise unchallengeable rights of imprimatur, have also failed to grasp a