LETTERS TO THE EDITOR

Colonic mucus and colitis

Sir,—I found the review on mucus and colitis by J M Rhodes (Gut 1989; 30: 1660–6) most interesting, but I was surprised by the absence of several pertinent references. It must surely be relevant that about 8% of the general population show a constitutional lack of the O acetylated form of mucin. Such mucosal alterations are likely to be secondary to inflammatory injury. Thus mucin heterogeneity, whether genetically determined or acquired, appears to be unimportant in the aetiology of ulcerative colitis. Perhaps the observations should be added to the other negative findings catalogued by J M Rhodes. I suggest that they reduce the likelihood of a 'mucus/bacteria' hypothesis.

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Reply

Sir,—I am sorry that Professor Jass’s excellent papers were not referred to in my leading article. Thus, however, intended as a presentation of hypotheses to act as stimuli for further studies rather than as a comprehensive review of published work. As pointed out in the article a change in the O acetylation of mucin sialic acids is just as resistant form of sialic acid. These people do not appear to show an increased premonence to ulcerative colitis.1 Patchy alteration in sialic acid structure (loss of O acetylated groups) is seen in hyperplastic epithelium in colitic biopsy material. Such mucosal alterations are likely to be secondary to inflammatory injury. Thus mucin heterogeneity, whether genetically determined or acquired, appears to be unimportant in the aetiology of ulcerative colitis. Perhaps the observations should be added to the other negative findings catalogued by J M Rhodes. I suggest that they reduce the likelihood of a ‘mucus/bacteria’ hypothesis.

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Scavenger effect of sulphasalazine (SASP), 5-aminosalicylic acid (5-ASA), and olsalazine (OAZ)

Sir,—We read with interest the paper by Williams and Hallett1 on the action of SASP and 5-ASA on toxic oxygen metabolite production by neutrophils. The authors conclude by suggesting a scavenger effect is induced by both drugs, as previously reported.2

We have recently concluded a similar experiment, evaluating the influence of SASP, its metabolites (sulphasalazine and 5-ASA) and OAZ, on the generation of oxygen anion (O2−) by activated neutrophils and by a cell free xanthine-xanthine oxidase system. Human neutrophils were prepared from heparinised peripheral blood of healthy volunteers by using a combined dextran/Ficoll-Hypaque separa-
tion procedure and hypotonic lysis to remove contaminating erythrocytes. The resulting cells (>95% neutrophils) were washed twice in phosphate buffer (pH 7.4) and then activated using 0.1 μg/ml of polymyxin B-streptate acetate.

The production of O2− by RBC, activated by the catalysed reaction xanthine oxidase upon xanthine, was induced by incubating 0.05 IU/ml of dialysed xanthine oxidase in 100 mM k-phosphate buffer (pH 7.8) containing 0.1 mM EDTA and 5 mM xanthine. O2− generation was measured either after spectrophotometrically reducing cytochrome c (c5t c) at 550 nm in a cuvette maintained at 37°C, or monitoring the spectrophotometric light emission at 37°C on a Perkin-Elmer luminescence spectrophotometer.

For testing the scavenger effect of SASP and OAZ, we could not use the chemiluminescence method, because of its intense yellow colour in solution, which would interfere with the light emission. We therefore used the reduc-
tion of cyt c assay for evaluating the effect of SASP, OAZ and also for sulphapyridine, but we could not use the assay for 5-ASA. In fact 5-ASA caused a direct chemical reduction of cyt c effect, already reported by Neal et al.3 Therefore, investigating the action of 5-ASA, we adopted the chemiluminescence method. In our study, 5-ASA and SASP both produced a dose dependent inhibition of superoxide anions in both the neutrophils and cell free xanthine-xanthine oxidase system, 5-AWA being the most powerful (>50% of inhibition at 10 μM, the lowest concentration used). In contrast, sulphapyridine showed a dose dependent inhibitory effect on the cellular system, not modifying the activity of xanthine oxidase.

As can be seen, our data are only partly in agreement with the findings of Williams and Hallett. In our opinion, this study is not without certain methodological limitations. The authors, in their experiment, did not report the interference of SASP on light emission, however, using a low colour density solution, therefore the inhibition on chemi-
luminescent response determined by SASP in their experiment could be partly attributed to the quenching effect on light emission by this drug. Finally, absorbance would highly increase by a direct chemical reduction provoked by 5-ASA on cyt c, as already mentioned. This, again, might determine a limitation of their results.

4 Craven PA, Plantisell J, Saito A, De Rubertis FR. Actions of sulphasalazine and 5-aminosalicylic acid as reactive oxygen scavengers in the suppres-

5 Neal TM, Winterbourn CC, Vissers MC. Inhibi-

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