surface membrane as FMLP interacts with a surface receptor but PMA interacts with an intracellular receptor to activate protein kinase c which stimulates the oxidase enzyme.

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References

- 1 Miyachi Y, Yoshioka A, Imamura S, Niwa Y. Effect of sulphasalazine and its metabolites on the generation of reactive oxygen species. *Gut* 1987; **28**: 190–5.
- 2 Goldstein IM, Cerquerira M, Lind S, Kaplan HB. Evidence that the superoxide generating system of human leukocytes is associated with the cell surface. *J Clin Invest* 1977; **59**: 249–54.
- 3 Halliwell B, Gutteridge JMC. The importance of free radicals and catalytic metal ions in human disease. *Mol Aspects Med* 1985; **8**: 89–193.
- 4 Carlin G, Smedegard G, Ahman H. Inhibition of neutrophil superoxide production by sulphasalazine and indomethacin [Abstract 39]. Society for Free Radical Research (Winter Meeting) 1986.

Reply

SIR,—I wish to thank Dr Andrew Williams for his interesting and constructive comments on my recent paper.¹ I agree with his opinion that the agent that inhibits neutrophil derived superoxide production also logically inhibits hydroxyl radical and hydrogen peroxide generation. In our experiments, SP mildly inhibited superoxide generation in the neutrophil system and also showed inhibitory trends in the xanthine-xanthine oxidase system, although the latter was insignificant. If SP had an SOD like activity, it will raise the level of hydrogen peroxide as we have previously reported.² In this case, it is not unlikely that SP inhibits superoxide production slightly without apparently affecting hydrogen peroxide or hydroxyl radical levels, because the expected slight reduction in these levels induced by the mild suppression of superoxide production may be masked. Another possible explanation for this dissociation is a time gap between each assay being done, although this is less likely.

As mentioned in the paper, these agents seem to affect the oxygen metabolism of neutrophils with somewhat different and probably multiple modes of action, which makes the issue complicated.

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References

- 1 Miyachi Y, Yoshioka A, Imamura S, Niwa Y. Effect of sulphasalazine and its metabolites on the generation of reactive oxygen species. *Gut* 1987; **28**: 190–5.
- 2 Niwa Y, Sakane T, Miyachi Y. Dissociation of the inhibitory effect of dapsone on the generation of oxygen intermediates – in comparison with that of colchicine and various scavengers. *Biochem Pharmacol* 1984; **33**: 2355–60.

Teeth and benign oesophageal stricture

SIR,—Maxton *et al* (*Gut* 1987; **28**: 61–3) have found that benign oesophageal strictures occur more frequently in a group of edentulous patients attending for endoscopy than in a control group with normal dentition. They suggest that edentulous patients eat less solid food than controls and the lower oesophagus is therefore subject to less dilatation with a greater tendency to stricture.

An alternative explanation, however, is that the edentulous patients because of poor masticatory function, chew solid food less efficiently and therefore swallow larger boluses which are more likely to obstruct their strictures. In this way they have more symptoms of dysphagia and thus present earlier for dilatation. The authors have assessed neither the dietary intake or masticatory abilities of their patient groups, and whilst other papers have suggested that edentulous patients eat less solid food than 'normal', these factors have not been established in this study.

We would agree that patients with strictures should be urged to use dentures, not primarily to reduce stricture formation but to encourage better chewing and therefore less bolus impaction and dysphagia.

The discrepancy between severe symptomatic oesophagitis and the relatively symptom free patients who appear to develop stricture does not invalidate oesophagitis as a cause of stricture formation. It is possible that those patients who are symptom free have the most severe reflux oesophagitis precisely because they are unaware of their oesophageal damage and do not present until a stricture has formed.

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Reply

SIR,—Drs Tait and McKinlay add another interesting suggestion for the cause of the increased prevalence of benign oesophageal strictures in the elderly with few or no natural teeth.

Because of their less efficient chewing, however, edentulous patients tend to choose softer, less solid food rather than eating larger poorly masticated boluses of more solid food. Thus, nuts and tough meat are rarely eaten by edentulous patients. We are