The symposium on which this supplement is based proved to be both interesting and intriguing. Blum (p. 3) opened the presentation with a discussion of his ‘state of the art’ approach to the control of acid secretion for the treatment of peptic ulcer disease. He demonstrated differences between omeprazole and the H₂-receptor antagonists and while ranitidine may appear to have been singled out, this is because in most comparative trials omeprazole has been compared with the pre-existing gold standard, ranitidine. Blum’s meta-analysis and studies by others support his conclusions of a significant advantage for omeprazole over ranitidine in a large number of trials in duodenal ulcer, gastric ulcer and gastro-oesophageal reflux disease. Walsh (p. 11) and Carlsson (p. 17) reviewed the trophic effects of gastrin, and presented confirmation that the development of gastric ECL cell hyperplasia and gastric ECL cell carcinoids in rats can result from profound hypergastrinaemia achieved by a variety of drugs or surgical techniques.

The proposition by Burinson et al. [1] and Burinson [2] that omeprazole might cause gastric genotoxicity cannot be supported by their data, which were seen in full during the symposium. Furthermore, the independent work of Wright (p. 24), Sachs (p. 31, 35) and from the Astra laboratories by Wallmark et al. (p. 39) has failed to reproduce the results of Burinson et al. [1]. The theoretical arguments refuting the genotoxic potential of the omeprazole sulphenamide also discount this possibility. Indeed, the suggestions by Burinson et al. run contrary to all the evidence obtained from the extensive approved genotoxicity, mutagenicity, and carcinogenicity studies which have been undertaken (p. 45).

The clinical data shown during the symposium confirmed our existing knowledge of the efficacy and safety of omeprazole in the treatment of reflux oesophagitis (p. 59, 64, 69, 72). New long-term data on treatment of this troublesome condition were also presented which suggest an important role for omeprazole in the prevention of recurrence (p. 69). The database for omeprazole has been expanded without evidence of significant adverse drug events.

So where does this leave the practising clinician? As I intimated in my introduction, I believe that everyone must evaluate this information for himself in a careful and objective way. For myself, I shall continue to prescribe omeprazole for those patients in whom I believe it is indicated. I shall also continue to prescribe omeprazole for patients long-term under the sort of circumstances described by Brunner (p. 64) and within the compassionate use programme which follows patients under protocol.

References