participants were approved by the ethical committee of each participating centre. The patients with treatment failure received triple therapy at any time during the study if they asked for it, or at the end of the study if they did not wish to be enrolled in the follow-up study. Those who were enrolled in the second study received triple therapy at the end of follow-up or if the ulcer recurred. Furthermore, during the trial, we used a dual therapy with amoxicillin and not with clarithromycin so that in the case of treatment failure, clarithromycin-based triple therapy could be subsequently used without the risk of acquired resistance to clarithromycin and consequent reduced efficacy.¹

Dr Graham and Dr Yamaoka also suggest that rather than focusing on the similar efficacy of 1 and 2 weeks of triple therapy, we should have highlighted that the efficacy of this therapy is "unacceptably low", regardless of its duration. As we discuss in our paper, the eradication rates obtained in our study (1 week of triple therapy intent to treat 79.7%, per protocol 83.6%; 2 weeks of triple therapy intent to treat 81.7%, per protocol 84.9%) add to the body of evidence showing that the efficacy of triple therapy has undoubtedly decreased in recent years. However, these are not substantially below the threshold of 80% defined by the Maastricht III Consensus Report as clinically unacceptable for the treatment of *H. pylori* infection.² Moreover, triple therapy, referred to by Dr Graham and Dr Yamaoka as a "legacy" treatment, is the treatment recommended by the Maastricht III Consensus Report. Dr Graham is himself one of the authors of this report. We must agree with this recent recommendation, at least until the higher efficacy demonstrated for new treatments³ has been confirmed by other investigators and in large studies in different regions worldwide.

We argue that the importance of our study lies rather in its large sample size and demonstration that extending the duration of triple therapy from 1 to 2 weeks does not significantly increase the rate of *H. pylori* eradication (the difference in eradication rate between 1 and 2 weeks of triple therapy was 2%; 95% CI -4% to 8%). Our trial involved a large number of centres (n = 81) in the south, centre and north of Italy, and so patients were recruited from a broad population, providing a good basis for the generalisation of results. Before our study, two meta-analyses reported that compared with 1 week of treatment, 2 weeks of triple therapy increases the eradication rate by 9–12%.^{4,5} However, although the results of meta-analyses are relevant, they cannot substitute for large clinical trials. Indeed, in their meta-analysis, Calvet *et al.*⁴ specifically mentioned that their results suggest that larger studies comparing 1 and 2 weeks of therapy are needed. We think that our study has addressed this need.

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Competing interests: RMZ has received reimbursement from Janssen-Cilag and Abbot for attending symposia, and fees for speaking from AstraZeneca, Takeda and Abbott. FB has received fees for speaking from AstraZeneca and Takeda, reimbursement for organising education from Altana and has carried out consultancy work for AstraZeneca.

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Risk of cancer and acid suppressant treatment

Garcia Rodriguez *et al.* (*Gut* 2006;55:1538–44) use data from the General Practitioner Research Database (GPRD) to report that the odds ratios (ORs) for incidences of non-cardia gastric cancer were increased fourfold in those who received acid suppressant treatment for ulcer indications, compared with those who did not, and fivefold for oesophageal adenocarcinoma in those with oesophageal indications. They consider that the increased risks reflect the indications for treatment and not drug toxicity. Although their conclusions may be right, their data raise some important concerns.

The GPRD has particular strengths in recording prescriptions, but case details can be less complete, which may explain why 482 of the oesophageal cancer cases were excluded because of unspecified histology, leaving 287 with adenocarcinoma for comparison with their controls without cancer. The assumption that these 287 were representative of other unidentified adenocarcinoma cases may or may not be sound.

Secondly, their paper suggests that gastro-oesophageal reflux or hiatal hernia is associated with this increased risk of oesophageal cancer, a conclusion that, if accepted, would result in radical reappraisal of current attitudes limiting cancer surveillance to patients with Barrett's oesophagus. We previously published data, 4 years ago in *Gut*¹ based on a surveillance of 18 000 patients who were prescribed omeprazole, and compared death rates from the main individual causes with those found nationally. Their dataset and ours were obtained by different methods, but they are, or should be, complementary, giving information on the risk of developing cancer and mortality ascribed to cancer (ours). We found 27 deaths from oesophageal cancer in 4 years of follow-up in 5517 patients with Barrett's disease, oesophageal stricture, ulcer or inflammation (OR 3.30 (95% CI 2.17 to 4.80)). By contrast, they found 16 cases among long-term users of acid suppressants, with all oesophageal indications (OR 5.42 (95% CI 3.13 to 9.29)). This OR is just compatible with ours of 3.30 in a restricted subset with severe oesophageal disease, but must be markedly higher if risk is, in fact, concentrated in a particular

subset with severe disease, such as that chosen by us. Table 5 in the paper indicates that only one of their oesophageal adenocarcinoma cases received long-term acid suppression for reflux as the sole indication, making it difficult to conclude that reflux alone is a risk factor, a finding consonant with ours, where 6 oesophageal cancer deaths were found in 6984 patients with hiatal hernia or reflux only OR 1.02 (95% CI 0.37 to 2.22).

Finally, they conclude that patients with ulcer indications treated by acid suppression have raised risks of gastric cardia and, particularly, non-cardia cancers. These findings are based on data from 522 gastric cancer cases, with 501 others excluded from consideration because gastric subsites were unclear. Table 7 shows that the raised ORs are derived from 3 cardia cases with ulcer indications, and 11 with non-cardia cancer, the OR for the cardia cancer cases being compatible with unity (OR 2.18, 95% CI 0.67 to 7.03). Retrospective consideration is always beguiling, particularly when derived from subset studies, and before accepting these, would it not have been sensible to compute risks for all gastric cancers, irrespective of subsite? We found that the chances of death due to gastric cancer fell to the population expectation after 2 years of follow-up in our total series of 51 cases, suggesting that any excess of earlier deaths in those who take drugs was likely to reflect acid suppressants prescribed for patients who already had gastric cancer, a conclusion consonant with results obtained in similar studies of cimetidine and published over 20 years ago.²

Garcia Rodriguez *et al.* agree with our earlier conclusion that confounding by treatment indication explains apparent increased risks of upper digestive cancer in those who receive acid suppressant treatment. However, their conclusions from data exploring oesophageal cancer risks associated with reflux and hiatal hernia, and gastric subsite risks with ulcer indications, seem insecure.

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Competing interests: None.

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Non-invasive measurement of small-bowel motility by MRI after abdominal surgery

Abdominal surgery under general anaesthesia causes postoperative ileus¹ with an extended recovery time.² Prolonged ileus creates major healthcare problems costing an estimated US\$