Effect of Helicobacter pylori on the gastric mucous gel

EDITOR,—In their paper on the basis of viscometric studies performed on human gastric mucus Markesich et al challenge the concept that Helicobacter pylori has any unfavourable effects on the mucus layer coating the gastric mucosa (Gut 1995; 36: 327–9).

Their results are clearly at variance with the bulk of the experiments carried out in this area1 and Markesich and colleagues try to show the reasons for this discrepancy by pointing out the potential drawbacks of the previous studies.

Even if we accept Markesich’s concept, however, the results of some experiments on H pylori have to be discarded because they are not performed on human mucus, other data are consistent with a mucus damaging effect of the microorganism in bacteria. Assessing the quality of gastric soluble mucus in humans we found that, although on the whole mucus is normal both in chronic gastritis and in duodenal ulcer, the lowest values are detectable in subjects with H pylori infection.2

Sidebotham et al also observed a breakdown of gastric mucus in patients infected with H pylori, although they ascribed this phenomenon to a carbonate-bicarbonate buffer at the mucosal surface due to the urease activity of the germ, rather than to a direct mucolytic activity.3 Measurements of the gastric mucous gel thickness in vivo showed that the mucous coating is significantly thinner in H pylori infected patients,4 possibly resulting from increased erosion.5

In keeping with the above results H pylori infection is also related to reduced mucus hydropobicity of human gastric mucus, which returns to normal after successful eradication.6

The reason for the discrepancy of Markesich’s results is probably the method used. Reference should be made to the study7 to justify the use of viscometry analysis for assessing the characteristics of mucus, but it is well recognised that viscometry is an unreliable technique for evaluating mucus properties.8 The finding that gastric mucus viscosity is increased in duodenal ulcer patients9 is only a further demonstration of how misleading the method can be.

As only luminal mucus was examined in Markesich’s study, it must be also considered that proteolytic enzymes produced by H pylori could promote a greater peptic erosion of the adherent mucus gel with consequent increase in the mucoprotein content of gastric juice influencing viscosity measurements. Eradication of H pylori would clearly reduce mucus shedding and thus apparently decrease the viscosity of intraluminal mucus.1

M GUSLANDI
Gastroenterology Unit,
San Raffaele Hospital,
University of Milan,
Italy


Reply

EDITOR,—Guslandi takes issue with our finding that gastric mucus gel in H pylori patients has a higher viscosity than in the unaffected. We simply gathered and tested the clear mucous gel that is normally present in the stomach. We used standard methodology for evaluating the viscosity of a gel. Our data were both consistent and reproducible.

There are many possible reasons why the gastric mucous gel in H pylori infected patients may be more viscous than those without. As the mucus cells seem to be under pressure to actively secrete, the gel may be younger that in the unaffected. It may also contain many more cells and cellular debris including DNA, which will in itself enhance viscosity. We make no apologies for the fact that Dr Guslandi studied soluble mucus and obtained different results. This should not be a surprise as soluble mucus is ‘used’ mucus that has completed its function and is probably better considered a waste product. The fact that the mucous gel might be thinner in H pylori infected patients, tells us nothing of the rate of synthesis, erosion, effectiveness or viscosity. We have previously shown that changes in mucosal hydropobicity can be separated from the H pylori infection.2 This means that hydropobicity seems more likely to be due to the inflammatory infiltrate (as has been previously shown in the colon) than for any factor made by the bacteria.3 More than 40 years ago, Hollander described increased viscosity in response to damage and other workers in gastric physiology who previously published on breakdown of gastric mucus have had no difficulty incorporating our findings.4

The histology of peptic ulcer disease has been one of exploring what turned out to be blind alleys. Researchers have seen their cherished beliefs destroyed and discovered that their lifelong work has not been in the main stream. The only thing left is to investigate this blindly only as a minor tributary.5 The truth will be discovered over time and only by additional experimentation.

D Y GRAHAM
B S ANDAN
V A Medical Center (111D),
2002 Holcombe Blvd,
Houston, TX 77030, USA

