with *H pylori* related diseases differed from the *H pylori* negative subgroup they have studied.

J ANDREWS
Gastroenterology Department, Radcliffe Infirmary, Oxford OX2 6HE

4 Hills BA. Gastric mucosal barrier: evidence for Helicobacter pylori ingesting gastric surfactant and deriving protection from it. Gut 1993; 34: 588-93.

Reply

EDITOR,—We appreciate the interesting comments of Dr Jane Andrews on our article. Her letter touches an important problem: the presence of *H pylori* in the samples analysed and its relation with surface hydrophobicity.¹

In fact, *H pylori* infection, the main cause of chronic gastritis and ulcer disease, can modify the phospholipid composition² and the gastric surfactant hydrophobicity³ because of the presence of the bacterial phospholipase A.⁴

Our study was, therefore, deliberately restricted to a selected subgroup of patients without histological evidence of gastric *H pylori* infection. We agree that an additional study needs to be done in a population that includes subjects with *H pylori* infection. We are in the process of performing such a study. The evaluation of the available data shows that *H pylori* infection induces significant variations of gastric phospholipid in the *H pylori* negative subgroup.⁵ We believe this confirms that our methodology is valid for the biochemical analysis of gastrointestinal mucosal samples.

G NARDONE
G BUDILLON
Cattedra di Gastroenterologia II, Università di Napoli Federico II, Via Pansino 5 80131 Napoli, Italy

1 Hills BA. Gastric mucosal barrier: evidence for Helicobacter pylori ingesting gastric surfactant and deriving protection from it. Gut 1993; 34: 588-93.