LETTERS TO THE EDITOR

Bacterial adhesion and *Helicobacter pylori*

SIR,—We read with great interest the important paper by Dr Hessey and colleagues (*Gut* 1990; 31: 134–8). Our ultrastructural studies^{1,2} also confirmed the close relation between the direct adhesion of *Helicobacter pylori* to gastric epithelium cells and epithelial degeneration, and we agree that this relation support a pathogenetic

role of *H. pylori* in chronic gastritis. We suspect, however, that the three categories of adhesion site observed by the authors may be conditioned by technical factors. When a bacterium is cut longitudinally all three of the described adhesive categories can be recognised (Fig 1): how the bacterial surface is transversally cut may affect how bacterium is classified.

An interesting question is whether *H. pylori* adhere before or after epithelial degeneration occurs, and we agree with Hessey *et al* that bacterial adhesion may play a part in cell degeneration. Our ultrastructural figures show the tendency of *H. pylori* to adhere to the cell surface already when primarily colonising the gastric epithelium: the large majority of bacteria (seen within the mucus layer) resting

on or are adherent to the short and irregular microvilli of an intact gastric epithelium, rather than suspended in mucus layer (Fig 2). Successively, possibly through the production of mucinase and cytotoxic activity,^{3,4} *H. pylori* may induce mucin depletion and microvillar and cellular damage. Damaged areas might be more susceptible to the acid attack. We also noted a decrease in the number of bacteria and in the extension of colonised areas when mucin depletion and cellular degeneration are present, as if the bacterial survival would be linked to a defined tissue background and the close adhesion to the epithelial wall would be the only alternative way of life.

Finally, our histological preliminary data seem to indicate that the type of chronic gastritis (superficial or atrophic chronic gastritis) and the presence of histological activity do not influence the modes of contact between *H. pylori* and gastric epithelium; the only relevant feature seems to be whether epithelial degeneration is present or not.

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- 1 Caselli M, Figura N, Trevisani L, *et al*. Patterns of physical modes of contact between *Campylobacter pylori* and gastric epithelium: implications about the bacterial pathogenicity. *Am J Gastroenterol* 1989; 84: 511–3.
- 2 Caselli M, Bovolenta MR, Alcott A, *et al*. Epithelial morphology of duodenal bulb and *Campylobacter*-like organisms. *J Submicrosc Cytol Pathol* 1988; 20: 237–42.
- 3 Slomiany BL, Bliski J, Sarosick J, *et al*. *Campylobacter pyloridis* degrades mucin and undermines gastric mucosa integrity. *Biochem Biophys Res Commun* 1987; 144: 307–14.
- 4 Figura N, Guglicmetti P, Rossolini A, *et al*. Cytotoxin production by *Campylobacter pylori* strains isolated from patients with peptic ulcers and from patients with chronic gastritis only. *J Clin Microbiol* 1989; 27: 225–6.
- 5 Leunk RD, Johnson PT, David BC, *et al*. Cytotoxic activity in broth-culture filtrates of *Campylobacter pylori*. *J Med Microbiol* 1988; 26: 93–9.

Reply

SIR,—Drs Caselli and Alvisi suggest that the three forms of adhesion we describe can be shown by the same organism and that their distinction is artificial. While it is true that an organism could in one plane of section be categorised as abutting and in another be seen to indent the plasma membrane, we feel confident that only a minority of organisms exhibit attachment by fully-formed pedestals. Hitherto, this latter feature has received much attention in the literature and our purpose in categorising the attachment sites was to emphasise the relative infrequency of adhesion pedestal formation compared to the number of organisms in intimate contact with the plasma membrane. Nevertheless, we did acknowledge that these various appearances were likely to be different stages in the process of adhesion which is why they were amalgamated for the purposes of analysis.

A further point at issue is the sequence of events leading to bacterial adhesion. Drs Caselli and Alvisi describe *H. pylori* first coming into contact with the microvillus border (primary colonisation?) where cytotoxin production and mucolytic activity cause cellular damage and disrupt the mucus barrier rendering the area more susceptible to acid attack. They have argued elsewhere that the bacteria then attach to the epithelium as a survival mechanism and that if it were not for bacterial adherence 'after degradation of the protective mucus layer, *C. pylori* would be rapidly washed out from the stomach.'¹

Figure 1: Gastric antral biopsy. Ultra thin section uranyl acetate-lead citrate stained. A longitudinally cut *Helicobacter*-like organism is recognisable. Original magnification $\times 15\,000$.

Figure 2: Gastric antral biopsy. Ultra thin section uranyl acetate-lead citrate stained. A large majority of *Helicobacter*-like organisms are resting on or are adherent to the microvillar surface of an intact epithelium. Original magnification $\times 12\,000$.