## LETTERS TO THE EDITORS

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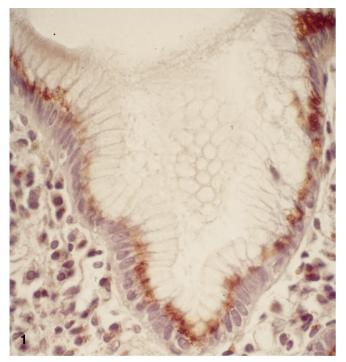
## Simple mucin-type carbohydrate antigens in *Helicobacter pylori*-positive chronic active gastritis

Sirs: We read with interest the excellent article entitled "Helicobacter pylori infection produces reversible glycosylation changes to gastric mucins" by Ota et al. [4], which was recently published in this journal. The authors, using monoclonal antibodies against simple mucin-type glycoproteins (Tn, sialyl Tn and Thomsen-Friedenreich antigen) and blood-group-related antigens bearing type 1 chain backbone, evaluated the immunoreactivity of gastric mucins in *H. pylori*-infected patients before and after cure and in normal uninfected volunteers. In particular, the authors encountered a significantly lower expression of Tn and sialyl Tn in antral mucosa of H. pylori-infected patients; after treatment for H. pylori the immunoreactivity for these antigens increased toward the normal pattern observed in antral mucosa taken from volunteers.

Utilising commercially available antibodies (HB-Tn1 and HB-T1, Dako, Copenhagen, Denmark; working dilution 1:200), we investigated the possibility that H. pylori might have a role in the production of changes in simple mucin-type carbohydrate antigen expression in chronic active antral gastritis. We selected 35 untreated patients who were to undergo endoscopic examination and subdivided them into two groups according to the Sydney system [2]: 25 were *H. pylori* infected (positive by culture, urease test and histological examination) and 10 were *H. pylori* negative. Immunohistochemical methods were applied as reported elsewhere [3]. The immunoexpression of Tn antigen (Fig. 1) localized at the supranuclear region of surface mucus cells was observed in all 35 samples with no significant quantitative differences in our two groups; these data may indicate that *H. pylori* infection does not influence Tn antigen expression in chronic active gastritis. In addition, T antigen immunoreactivity was found in the supranuclear region of surface mucus cells in only 16% of H. pyloripositive cases (Fig. 2), whereas in uninfected cases its staining was always negative; nevertheless, the low number of *H. pylori*-infected cases with T antigen reactivity did not allow us to retain this surface simple mucin-type antigen capable of specific binding to *H. pylori*.

In order to explain the expression pattern of simplemucin type antigens in chronic gastritis, Ota et al. [4] also characterise the normal pattern of positivity in gastric mucosa of volunteers. In detail, Tn and T antigens showed a diffuse cytoplasmic staining in the surface mucus cells in the antrum of 100% and 25% cases, respectively; in addition, Tn antigen was also detected in supranuclear regions of the same cells. Furthermore, Ota et al. [4] reported this latter pattern of Tn antigen exclusively expressed in pyloric mucosa of 21% of chronic active gastritis patients before treatment of their H. pylori infection; this finding is constantly observed in our *H. pylori*-infected cases. Analysis of the expression of T antigen performed by Ota et al. [4] showed a positive rate of 21.4% in antral mucosa of *H*. pylori-infected patients; a similar rate of T antigen positivity was also found in infected mucosa with a supranuclear localisation. Interestingly, in our group of patients with *H. pylori*-negative chronic active gastritis, we found a different pattern of Tn and T antigens from that reported by Ota et al. [4], in antral mucosa of volunteers as well as in that of patients after treatment for H. pylori after the disappearance of neutrophils. However, significant differences in the glycoconjugate patterns of the mucus cells of the antral surfaces were demonstrated by lectins between normal gastric mucosa and *H. pylori*-positive chronic active gastritis [1]; these differences in lectin conjugates have been attributed to bacterial factors or nonspecific reactions to the inflammatory processes [1]. In the light of our observations, we suggest the activity may itself act as a cofactor to explain changes in antral mucin-type carbohydrate anti-

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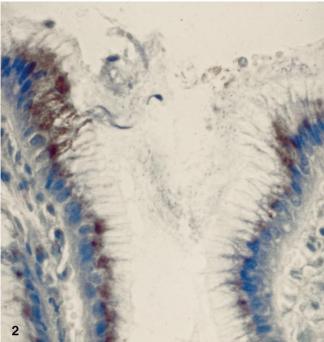


Fig. 1 Monoclonal antibody anti-Tn antigen. The immunoreactivity was evident in the supranuclear region. Mayer's haemalum counterstain,  $\times 50$ 

**Fig. 2** Monoclonal antibody anti-T antigen. *Helicobacter pylori* was seen in the glandular lumen while surface epithelial elements were reactive at the supranuclear region. Giemsa counterstain, ×50

## **References**

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