LETTERS TO THE EDITOR

Serum peptidoglycan in blood transfusion donors

EDITOR.—We read with great interest the paper by Veennedael et al in Gut.1 In this study the authors showed that blood donors positive for H pylori antibodies had a higher serum peptidoglycan (NUD) and proved active helicobacter associated gastritis was significantly higher than in healthy seropositive blood donors (mean 3.63 (1-24) range 1-06-6-45 v mean 2-32 (0-47) range 1-51-3-43 respectively, p<0.001). Although considerable overlap was present in the lower ranges, it was concluded that high P/N ratios occur with active inflammation and that lower P/N ratios can reflect a serological scar of past infection as well. It was also shown that inflammation with polymorphonuclear cells invading the mucosa causes a higher P/N ratio, hence antibody response, compared with a milder degree of inflammation.1

The Table

<table>
<thead>
<tr>
<th>Age (yr)</th>
<th>Blood donors</th>
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<th>Patients with non-ulcer dyspepsia</th>
</tr>
</thead>
<tbody>
<tr>
<td>21-30</td>
<td>3:34 (0-93)</td>
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Figure: Data from the table shown graphically.

and Figure the mean P/N ratio and the standard deviation of the healthy seropositive blood donors and the patients with NUD are plotted against age. It is clear that the findings of Veennedael et al cannot be confirmed as no increase in P/N ratio with rising age is seen in the healthy blood donors. On the other hand, a slight increase in antibody response is seen among the patients with non-ulcer dyspepsia. Comparison of age cohorts 21-30 and 31-40 with age cohort 61-70 revealed a p value of 0.08 and 0.02 respectively (Student’s t test). The other cohorts showed no differences in the height of the P/N ratio. In our opinion, this does not indicate that the antibody titre in an individual patient increases with rising age. Longitudinal serological follow up has not been done to our knowledge. The only data reported in previous studies show a decrease in antibody titre after therapeutic intervention aimed at suppression or eradication of H pylori, and an increase in the incidence of recrudescence or reinfection. From the paper of Veennedael et al it cannot be concluded that the antibody titre from an infection with H pylori acquired at an early age rises when the patients get older.

The statement that chronic active gastritis becomes worse with rising age can be considered correct if the development of intestinal metaplasia and glandular atrophy are considered a part of the deterioration of gastric mucosal histology. As reported in previous studies, however, the role of H pylori during the course diminishes and it is more logical to expect that the antibody response in the individual patient does not rise during the course of helicobacter gastritis. Serological follow up in gastritis patients is necessary to learn more of the natural history of the antibody response.

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Reply

EDITOR.—We thank Loffeld et al for their interest in our paper in which showed that the age related increase in serum peptidoglycan A and C occurring in healthy populations, is caused by Helicobacter pylori infection.1 As Loffeld et al do not mention data regarding P/N ratio with rising age. Longitudinal serological follow up has not been done to our knowledge. The only data reported in previous studies show a decrease in antibody titre after therapeutic intervention aimed at suppression or eradication of H pylori, and an increase in the incidence of recrudescence or reinfection. From the paper of Veennedael et al it cannot be concluded that the antibody titre from an infection with H pylori acquired at an early age rises when the patients get older.

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Faecal pH and colon cancer

EDITOR.—I would like to comment on the excellent letter1 from Johannesburg reporting on the low faecal pH levels in African subjects and their possible connection with the low incidence of colorectal cancer.

My own experience in India supports the concept of a possible relation between faecal pH and the incidence of large bowel cancer.1 A group of 60 patients with colorectal cancer, principally south Indians, had a median faecal pH of 6-8-9-0, compared with a median pH of 6-1-6-5 in 120 matched healthy Indian subjects. The large differences in pH values between patients with colorectal cancer and the control group appear to be dependent on diet. The former ate non-masticatory meals of boiled refined rice, which were low in dietary fibre, and low in fermented milk products. The latter ate high fibre meals of thick whole wheat chapatties, vegetable and gurme curries, and their diets were also high in fermented milk products such as yoghurt, yoghurt drink, white cheese, and ghee – made from fermented milk and rich in short chain fatty acids. Fibre fermenters in the gut lumen liberating large quantities of acetic acid (precursor of short chain fatty acids). This augments the H ion in the case of the controls.

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Letters

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