CBS, was incorporated in brain heart infusion agar plus 7% horse blood plus 1% IsoVitalex to achieve final concentrations of 0-25–64 mg/l, with appropriate controls. The inoculum was 0·1 ml of an undiluted broth culture shaken for 18 hours. MICs of CBS for C. pylori have been found to be 8–32 mg/l by us and others.6–7

Fifteen isolates of C. pylori obtained from patients unsuccessfully treated with CBS were studied and the MICs were found to be 16–32 mg/l. In a pair of isolates before and after treatment, the MIC of CBS seemed to drop from 32 mg/l to 16 mg/l, but this result is within experimental error. Thus all pairs of isolates showed the same MIC before and after treatment. There was no development of resistance to CBS.

C. pylori was also inoculated into liquid medium consisting of brain heart infusion broth plus 10% horse serum plus 0·25% yeast extract with final concentrations of CBS of 4, 8, 16 and 32 mg/l. The broth was shaken overnight in a microaerophilic atmosphere. Subcultures to CBS concentrations twofold higher in each case, up to 16 and 32 mg/l, yielded no growth in the latter concentrations. Thus we were unable to train C. pylori to become resistant to bismuth.

These results are encouraging for the clinical use of CBS to treat C. pylori, but we have already shown that CBS must be combined with at least one other antibacterial agent to achieve more than 30% eradication of C. pylori in the stomach. This is probably due to the fact that C. pylori can be found deep in mucus glands and would often be sequestered from the intraluminal action of CBS.8

Enterotoxin production by strains of Staphylococcus aureus isolated from clinical specimens

Certain strains of Staphylococcus aureus are known to produce different enterotoxins designated as A, B, C, D and F (TSST-1). These enterotoxins have an important role in the pathogenesis of staphylococcal infections, mainly in food poisoning outbreaks and in the toxic shock syndrome (TSS). Between 25–50% of the population are carriers of S. aureus and 15–20% of the strains are enterotoxigenic.6 This is probably due to the fact that C. pylori can be found deep in mucus glands and would often be sequestered from the intraluminal action of CBS.6

References


Table Enterotoxin production by strains of Staphylococcus aureus isolated from patients

<table>
<thead>
<tr>
<th>Enterotoxin type (%)</th>
<th>Site of isolation</th>
<th>A</th>
<th>B</th>
<th>C</th>
<th>D</th>
<th>AC</th>
<th>AD</th>
<th>BC</th>
<th>BD</th>
<th>BCD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Skin</td>
<td>17 (11·1)</td>
<td>7 (1·1)</td>
<td>17 (26·9)</td>
<td>25 (39·6)</td>
<td>4 (6·3)</td>
<td>6 (6·3)</td>
<td>2 (1·5)</td>
<td>1 (4·7)</td>
<td>1 (1·5)</td>
<td></td>
</tr>
<tr>
<td>Respiratory tract</td>
<td>14 (2·1)</td>
<td>3 (1·1)</td>
<td>7 (33·3)</td>
<td>4 (19·0)</td>
<td>6 (28·5)</td>
<td>1 (1·5)</td>
<td>1 (4·7)</td>
<td>1 (1·5)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Blood</td>
<td>6 (6·3)</td>
<td>4 (33·3)</td>
<td>6 (50·0)</td>
<td>2 (16·6)</td>
<td>2 (16·6)</td>
<td>1 (12·5)</td>
<td>3 (60·0)</td>
<td>1 (20·0)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bone</td>
<td>3 (3·7)</td>
<td>2 (25·0)</td>
<td>1 (20·0)</td>
<td>1 (50·0)</td>
<td>1 (30·0)</td>
<td>1 (100)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Urine</td>
<td>1 (0·9)</td>
<td>6 (5·4)</td>
<td>5 (4·5)</td>
<td>7 (6·2)</td>
<td>2 (1·8)</td>
<td>1 (0·9)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Total 17 (16·1) 32 (28·5) 32 (28·5) 10 (8·9) 6 (5·4) 5 (4·5) 7 (6·2) 2 (1·8) 1 (0·9)
Letters to the Editor

resistant of the six staphylococcal enterotoxins and has always been associated with enterocolitis.1 The prevalence of enterotoxin B production by the strains studied in the present series shows that these strains have the capacity to produce enterocolitis. The absence of enterotoxin F in our series contrasts with the high percentage of F producing strains (42%) reported by DeNoij et al.2 This author found that many strains isolated from clinical specimens not associated with the TSS produced enterotoxin F.

The incidence of S aureus enterotoxigenic strains varies according to the clinical origin of specimen. In our series the strains isolated from skin were the most enterotoxigenic; 61-7% of these strains produced one or more enterotoxins. This result is higher than that obtained by Melconian et al.,3 who reported an incidence of 48.8% in their series. On the other hand, the highest percentage was obtained in strains isolated from the respiratory tract (58.8%), while our incidence was 53.8% for these specimens. In the strains isolated from blood, six (50.0%) produced enterotoxin B and six (50.0%) enterotoxin A; four (33.3%) strains produced only enterotoxin A, and two (16.6%) also produced enterotoxin C. In the rest of the clinical specimens we were only able to isolate a reduced number of S aureus strains, so the results were not significant.

Our results are different in many respects from those published. This discrepancy may depend on epidemiological factors, or it may be the result of different methods of enterotoxin detection used. Further studies are required in this field.

References


Nucleolar organiser regions in squamous tumours of the pharynx and larynx

Squamous tumours of the pharynx and larynx, including benign papilloma, verrucous carcinoma, and invasive carcinoma, may be difficult to distinguish histologically. We applied the recently introduced silver colloidal technique used to identify nucleolar organiser regions (AgNORS) to examples of these tumours in an attempt to facilitate diagnosis.

Ten cases of each of benign squamous papilloma, verrucous carcinoma, and invasive squamous carcinoma (including well, moderately, and poorly differentiated tumours) of the pharynx and larynx were examined. Sections were stained by the usual AgNOR method solution1 and the AgNORS, which were seen as intranuclear dots, were counted using a x100 oil immersion lens. One hundred cells from all epithelial layers in random fields from each section were examined and the mean number of AgNORS in each cell calculated.

Squamous papilloma had a mean AgNOR count of 6-9 (range 4.5-9.2; SD 1.50). The mean value for verrucous carcinoma was 7.3 (range 5.5-10.7; SD 1.55) and for invasive squamous carcinoma 7.4 (range 4.5-10.9; SD 2.49). There was no significant difference between the mean AgNOR counts for the different tumour types.

AgNORS are loops of DNA in the cell nucleus which contain genes coding for ribosomal RNA, and some of the associated proteins (NORAPs) are stained with the AgNOR staining solution. Differences in mean AgNOR count have been shown to be useful in distinguishing between various conditions which may be difficult to differentiate on routine histological examination. These include high and low grade non-Hodgkin's lymphoma,1 melanocytic lesions of skin,2 and low grade infantile fibrosarcoma and fibrous proliferations of childhood.3 In other cases the AgNOR technique has been disappointing—for example, when applied to making the distinction between various types of disease in endocrine tissue.4 Hall et al. have shown a linear relationship between AgNOR count and Ki-67 immunoreactivity, suggesting that AgNOR numbers may be related to cell proliferative activity.5 This has been confirmed in non-Hodgkin's lymphomas by DNA flow cytometry. In tissues in which malignant tumours have a significantly higher mitotic rate than their benign counterparts the AgNOR technique would then be expected to be a useful diagnostic indicator. In other tumours, including those of endocrine tissue, the situation may be less well defined as benign lesions may have a relatively high mitotic rate. Hence the AgNOR count may possibly be as high as a result of stimulation by trophic hormones. Hormones may affect NOR numbers and sizes by means of gene amplification.6

Similarly, viral stimulation may result in considerable proliferative activity in benign lesions of the pharynx and larynx. Hence there may be little difference between the AgNOR counts in these lesions and those of malignant tumours. Again, it has been shown that gene amplification may occur in virally infected cells.

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References