β-lactamase production by Campylobacter jejuni

There is very little information now available about the β-lactamase producing strains of Campylobacter jejuni among human isolates. Severin et al. in Holland reported β-lactamase production by 57 (92%) of 62 human isolates by the chromogenic cephalosporin method. We therefore decided to undertake a retrospective study of the incidence of β-lactamase production among strains of C. jejuni, isolated over a 12-month period from human stools. Two simple laboratory methods were used, the starch-iodine paper and the chromogenic cephalosporin substrate. In addition, minimum inhibitory concentrations (MIC) of ampicillin for each of the strains were determined.

Identical strains isolated from family outbreaks were included only once. A total of 76 strains of C. jejuni were studied, of which 73 (96%) had been stored in liquid nitrogen for varying periods not exceeding one year. Strains were inoculated on to Oxoid blood agar base with 7% lysed horse blood and incubated under appropriate conditions.

Minimum inhibitory concentrations of ampicillin for all 76 strains were determined by 2 μl spot-inoculation on Oxoid Diagnostic Sensitivity Test agar incorporating doubling dilutions of ampicillin. These plates were then incubated for 48 hours under identical conditions. The inoculum was derived from an overnight culture of the test strain suspended in Bloodgrow (Medical Wire and Equipment Co (Bath) Ltd, Potley, Corsham, Wilts, UK) and incubated in 10% CO₂ atmosphere at 37°C and diluted so that there were 10⁸ to 10⁹ organisms/ml.

Of the 76 strains of C. jejuni tested for β-lactamase production, four (5.3%) gave positive results; identical results were obtained by both methods. The Figure shows the MIC of ampicillin of 72 strains was 25.0 μg/ml or less; all these strains were negative when tested for β-lactamase. The MIC of the four β-lactamase producing strains was 50-0 μg/ml or more.

Our finding of an incidence of 5-3% from human stools differs markedly from the 92% of Severin's study. There may thus be a much higher prevalence of β-lactamase producing strains of C. jejuni in Holland. Severin also reports that 30 (48%) of 62 strains were sensitive to 5-0 μg ampicillin/ml, this figure being similar to our own findings, but sensitivities to higher concentrations of ampicillin were not given.

We noticed that it was difficult to interpret the colour change with the chromogenic cephalosporin substrate method because of the pigmented nature of the colonies when picked off agar

Sensitivity of 76 strains of C. jejuni to ampicillin.
medium containing blood. For this reason we preferred to test our strains after growth on blood agar base. Precise details of Severin’s chromogenic cephalosporin substrate method were not stated. To the best of our knowledge, antibiotic therapy had not been given to the four patients from whom β-lactamase producing strains were isolated. Clearly, the possibility of transfer of plasmids determining β-lactamase production between campylobacters and other intestinal bacteria deserves consideration.

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References


Reference standard for packed cell volume

In a recent issue (J Clin Pathol 1980;33:1) the International Committee for Standardization in Haematology presents a recommendation for a reference method for determining packed cell volume (PCV) of blood. I feel that there has been a significant omission in that the committee does not specify the proportion and type of anticoagulant to be used. Although trapped plasma may increase PCV, the effect of the anticoagulant may offset this by decreasing the volume of the individual red cell.

Brittin et al.1 studied the effect of excess disodium EDTA and demonstrated that excess EDTA shrinks red cells in proportion to the excessive concentration of anticoagulant. However, this error, due to excess anticoagulant, was not produced when the haematocrit was determined by the Coulter Counter Model S.* It has been our experience, in an unpublished study comparing 1500 duplicate pairs of haematocrit values done by the microhaematocrit technique and by the Coulter Counter Model S, that the microhaematocrit was one unit lower than the haematocrit as determined by the Coulter Counter Model S. We feel that this is probably due to excess EDTA, which overcompensates for the increased PCV created by excess plasma.

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Pseudoleptospira in blood culture

We noted with interest the observation by Rahman and Macis1 that pseudoleptospira could be identified when blood cultures from healthy humans were examined under dark-ground microscopy. We have observed the presence of artefacts similar in all respects to those described by these authors when whole blood samples from normal, healthy guinea-pigs, hamsters, mice, and chickens have been submitted to direct dark-ground examination. Furthermore, the same type of spiral filaments have invariably been observed when fluid from freshly prepared or incubated suspensions of liver and kidney tissue from these same animals have been similarly examined. It would thus seem likely that such artefacts would be found in corresponding preparations from other animal species as well as man. Although these pseudoleptospira can usually be fairly easily differentiated from the true leptospire by an experienced worker, we concur wholeheartedly with the view that a diagnosis of
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