Correspondence

Neomycin blood agar as a selective medium for vancomycin resistant *Enterococcus faecium*

We read with interest the article by Chadwick and Oppenheim[1] regarding the selective isolation of *Enterococcus faecium* and agree that further comparative studies of screening media are required for detection of vancomycin resistant enterococci (VRE) from clinical and environmental sources. Furthermore, we believe that an overall strategy for the isolation of these increasingly prevalent nosocomial pathogens should be developed.

Using cephalexin aztreonam arabinose agar (CAA),[2] a medium developed for the selective isolation of *E faecium*, in association with a broth enrichment technique, we examined 92 swabs from 70 environmental sites and 22 rectal swabs during the investigation of a nosocomial outbreak on a renal unit. All swabs were plated directly onto CAA, and CAA containing 4 mg/l vancomycin (Eli Lilly). The swab was then placed into cephalexin aztreonam (CA) broth prepared by the addition of cephalexin and aztreonam to one litre of sterile brain heart infusion broth (Unipath). Plates were examined for *E faecium*, following 24 and 48 hours' incubation at 37°C in air. CA broth was subcultured onto both of the above media following enrichment for 24 hours.

Thirty eight *E faecium* strains were isolated from 92 environmental and patient samples. Of these, 28 (74%) were vancomycin sensitive and 10 (26%) were vancomycin resistant. When the isolation of *E faecium* from direct culture and broth enrichment was compared, 16 strains (42%) were isolated on direct culture, and the remaining 22 strains (58%) were isolated from broth enrichment only. Of the 10 vancomycin resistant strains, only two (20%) were isolated on direct plating. It was interesting to note that vancomycin resistant strains often required 48 hours' incubation to produce typical colonies. This delayed growth was presumably because of the time required for the induction of the Van B resistance phenotype typical of our outbreak strains.

Our investigations show that the isolation rate of *E faecium* during nosocomial outbreaks may be seriously underestimated if a broth enrichment procedure is not used, as only 16 (42%) of 38 *E faecium* strains were isolated on direct culture. Moreover, only 20% of strains of VRE were isolated on direct culture. It is likely that the additional strains detected after broth enrichment were present in low numbers, which might easily have been missed if the broth enrichment step was not used, regardless of the type of selective media used. This effect might, however, be compounded if a selective medium inhibitory to VRE[1] was used without an enrichment stage.

In order to implement a successful infection control strategy it is essential that accurate information is available about the numbers of cases of clinical infection or colonisation, and the extent of any environmental contamination with VRE. Our study suggests that outbreak management based on results of screening exercises using only direct culture techniques may be inappropriate.

We agree with Chadwick and Oppenheim[1] that comparative studies of screening media are warranted, but also recommend the use of a broth enrichment step in association with an appropriate selective medium such as CAA for the isolation of VRE during the investigation of nosocomial outbreaks.

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1 Chadwick PR, Oppenheim BA. Neomycin blood agar as a selective medium for vancomycin resistant Enterococcus faecium. *J Clin Pathol* 1995;**48**:1068–70.
2 Ford M, Perry JD, Gould FK. Use of cephalexin aztreonam arabinose agar for the selective isolation of Enterococcus faecium. *J Clin Microbiol* 1994;**32**:2999–3001.

Coronary artery dissection

Bateman *et al*[1] describe an interesting spectrum of clinical presentation of spontaneous coronary artery dissection. Despite its rarity, the entity shows a striking constancy in the vessels involved and the presence of an inflammatory infiltrate rich in eosinophils. These features were also seen in a recent necropsy in our department. The patient, an obese 43 year old woman with no recent pregnancy, had a background of mild hypertension not requiring medical therapy. She complained of severe back pain one evening, and died the following morning. At necropsy, the heart weighed 400 g. The left anterior descending coronary artery was occluded by thrombus from its origin, and a dissection, clearly visible grossly, extended the length of the artery. There was no atheroma, and histologically, no abnormal accumulations of mucin and no evidence of systemic vasculitis. No intimal tear was identified. Like cases 2 and 3 reported by Bateman *et al*, in which there was an interval between onset of symptoms and death, there was an adventitial infiltrate with prominent eosinophils. The dissection in our case was mostly between the media and adventitia, internal to the external elastic lamina, with small foci in the outer media. We have seen dissection in this location in a previously reported case,[2] as have others.[3] It seems likely that it overlaps with the dissection in the outer third of the tunica media, and does not justify the description of "unusual" as suggested.[1] Finally, increased awareness of this entity may mean that early presentation may result in salvage of some cases.

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1 Bateman AC, Gallagher PJ, Vincenti AC. Sudden death from coronary artery dissection. *J Clin Pathol* 1995;**48**:781–4.
2 McDonald GSA. Spontaneous dissection of the coronary artery. *Ir J Med Sci* 1989;**158**:304–6.
3 Glasgow BJ, Tift JP, Alexander CB. Spontaneous primary dissecting coronary artery aneurysm. *Am J Forensic Med Pathol* 1984;5:155–9.

Drs Bateman and Gallagher comment:
We read Drs Mooney and McDonald's further report on spontaneous coronary artery dissection with interest. The intimal dissection in our first case remains unusual.[1] However, their comments regarding the plane of dissection in our second and third cases are entirely justified. We accept that coronary artery dissection has been observed both within the outer tunica media and between the media and adventitia.[1–4] Differences in the reported plane of dissection may result from sampling error. It is widely believed that spontaneous coronary artery dissection results from luminal blood entering the arterial wall via an intimal tear, although this feature is rarely identified at postmortem examination.[3] Histological examination of coronary artery segments may therefore reveal dissection at varying levels within the tunica media as the blood flows towards the relatively impervious tunica adventitia.

Adventitial inflammatory cell infiltrates containing a high proportion of eosinophils remain a common feature of spontaneous coronary artery dissection, and their role has yet to be elucidated. Drs McDonald and Mooney suggested an intriguing association between autoimmune endocrine disease and coronary artery dissection in a previous report, although the composition of the inflammatory cell infiltrate differed between the involved coronary artery and the autoimmune thyroiditis.[2] The pathogenesis of spontaneous coronary artery dissection, particularly in patients in whom no associated factors are identified, remains an enigma.

1 Bateman AC, Gallagher PJ, Vincenti AC. Sudden death from coronary artery dissection. *J Clin Pathol* 1995;**48**:781–4.
2 McDonald GSA. Spontaneous dissection of the coronary artery. *Ir J Med Sci* 1989;**158**:304–6.
3 Glasgow BJ, Tift JP, Alexander CB. Spontaneous primary dissecting coronary artery aneurysm. *Am J Forensic Med Pathol* 1984;5:155–9.
4 Robinowitz M, Virmani R, McAllister HA. Spontaneous coronary artery dissection and eosinophilic inflammation: a cause and effect relationship? *Am J Med* 1982;72:923–8.

Effects of interleukins on the proliferation and survival of chronic lymphocytic leukaemia cells

Mainou-Fowler *et al*[1] report in their interesting study of the in vitro response of B chronic lymphocytic leukaemia (B-CLL) cells to interleukins that the effects of interleukin-4 (IL-4) are heterogenous. They show that "IL-4 enhanced cell proliferation by . . . 235% (123–400%) in four of 12 B-CLL cases" and they propose that this variability in response is a result of variable B-CLL cell maturity and defective expression of receptors for growth factors.

We suggest that their observations may be a result of heterogenous expression of the IL-4 receptor (IL-4R), as we have shown that B-CLL cells express IL-4R and have presented evidence of the expression of two species of high affinity receptor by these cells.[2] Briefly, the presence of high affinity IL4-R was determined by ^{125}I labelled IL-4 binding and Scatchard analysis using MLA-144 cells as a positive control.[3] While a high affinity IL-4R was detected in all six samples examined, there was evidence in some cases of expression of a distinct, and previously unreported, high affinity IL-4R. Thus, four of six samples expressed the conventional[4] high affinity IL-4R, K_D 17-95 pM, which was of similar affinity to the IL-4R expressed by MLA-144, K_D 22-188 pM, but two of six samples expressed a high affinity receptor, K_D 293-549 pM. The IL-4R was initially thought to be composed