Correspondence

Safer staining method for acid fast bacilli

I read with interest the article by Ellis and Zarbrowany on the use of a non-phenolic staining solution for acid fast bacilli and can report that the technique works well in our hands. Any modification of standard staining techniques that can reduce the use of hazardous chemicals is to be welcomed and to that end we substituted phenol with the LOC High Suds in several other methods used in this department.

The following techniques were tried:
(a) Long Ziehl-Neelsen stain, for lipofuchsin2
(b) Lendrum’s carbol chromatope, for eosinophil granules3
(c) Gram’s stain (where dilute carbol fuchsin is used to stain Gram negative organisms) for bacteria4
(d) Modified Ziehl-Neelsen stain, for cryptococcosis.

I can report, in each case, that the results obtained were comparable with those obtained with the original techniques. I would recommend the use of LOC High Suds in all of these techniques as a safer, cheaper substitute for phenol.

B KELLY
Department of Veterinary Pathology, Royal (Dick) School of Veterinary Studies, University of Edinburgh, Summerhall, Edinburgh EH9 1QH

3 Lendrum CC. The staining of eosinophil polymorphs and enterochromaffin cells in histological sections. J Pathol Bacteriol 1944;56:3.

Dr Ellis comments:
We also have had excellent results with the long Ziehl-Neelsen stain for lipofuchsin, but have not tried to substitute LOC High Suds in the other methods mentioned mainly because they are not methods we commonly use. Our primary aim is to investigate and develop safer methodology for those techniques used in this department.

The method currently under development is a modification of Fite and Faracco’s method for Mycobacterium leprae. We have developed a staining solution to replace carbol fuchsin which contains LOC High Suds, with which we have had considerable success. I hope that the method will be published after further trials in other laboratories and after staining a wider range of tissues.

Seroconversion for Helicobacter pylori

Kuipers et al5 recently reported a very low seroconversion for Helicobacter pylori infection in an adult population. They measured H pylori immunoglobulin G (IgG) antibodies in two serum samples taken from each of 115 patients, obtained with a mean interval of 11-5 years, and found that only two patients became infected during follow up. From their data, the authors suggested that the age related increase in H pylori prevalence was due to a dominant infection rate in childhood. Data on seroconversion in an untreated population are quite scarce. We report our data on 207 asymptomatic Italian children (aged 4-15 years) and 1010 blood donors (aged 18-65 years) who have been assessed serologically for both IgG and IgM (by in-house enzyme linked immunosorbent assay (ELISA)), with a specificity and sensitivity of 93%.6

Our results show that the prevalence of H pylori IgG antibodies increases with age, both in children and in adult blood donors, but that the prevalence of H pylori IgM antibodies is highest in the 18-25 year age group and that it decreases with age (fig.1). Concentrations of IgG or IgM antibodies in H pylori positive patients (measured by optical density at 470 nm) did not change with age. Our data strongly support their hypothesis of an age-cohort effect, with the acquisition of most H pylori infection during youth (below the age of 20 years).

High IgM titres consistent with a first contact with the infection associated with low IgG titres, that consistently correlate with active H pylori gastritis, strongly support the hypothesis of a spontaneous elimination of the infection in young patients. A spontaneous elimination of the first infection was shown in 33 out of 134 Gambian children aged 1-15 months by measuring serum antibodies and performing a 14C urea breath test every month over a period of 2 years. Most contact with H pylori infection occurs in childhood, but the majority of younger subjects will spontaneously eliminate it. In Italians this occurs mostly during the second or third decade and in Gambians in the first 5 years of life; the difference is probably related to either hygiene conditions or the nutritional status of the population.

G ODERDA
Paediatric Gastroenterology, University of Torino, Piazza Polonia 94, 10126 Torino, Italy
D VAIKA
1st Medical Clinic, University of Bologna, Bologna, Italy
J HOLTON
Department of Microbiology, University College and Middlesex School of Medicine, London


Tissue artefacts caused by sponges

Following the recent correspondence by Platt and Newman regarding the use of tea-bags or synthetic Shandon bags in the processing of small biopsy specimens, we wish to draw attention to a tissue artefact which may occur when such specimens are processed in synthetic bags.7

Following the discovery that triangular shaped defects in renal and liver biopsy specimens were due to the use of foam sponges in embedding cassettes,2 we changed our procedure and processed all such specimens wrapped in perspex. Recently, however, our laboratory ran out of perspex and for a few weeks we processed renal biopsy specimens in Shandon bags. We soon noticed a small angular elliptical defect (fig 1) was occurring in tissue sections. Close inspection of the bag

Figure 1 Elliptical effect in tissue sections.
production. Recent evidence indicates that, as in the mouse, human T cells of the Th1 subset synthesise and secrete interleukin-2 (IL-2) and γ-interferon (IFN), whereas those of the Th2 subset produce interleukin-4 (IL-4) and IL-5 but not IL-2 and γ-IFN. Further work is required to clarify this issue. In particular, the relationship between Th1 and Th2 subsets and the presence of the Th3 subset, which is characterised by the presence of IL-10, remains to be elucidated.

Dr Metz comments:
We thank Dr Slatter for his comments, and agree that the prominence of eosinophils in conditions like HES has distracted attention from the possible cell of origin: a T cell. We would agree also that the T cells in our patient were probably of the Th2 phenotype, and we in fact demonstrated detectable concentrations of IL-5. However, although the Th2 cell class is increasingly employed in murine cell lines, its clinical relevance in human disease remains to be fully documented.

With regard to the clonality of the lymphocytic proliferation studies on bone marrow, as we reported, failed to demonstrate any cytogenetic abnormality and genotypic studies on blood showed a polyclonal pattern. We did not consider it warranted to repeat the bone marrow biopsy for the sole purpose of obtaining material for genomic Southern blot analysis. Although it remains possible that a clonal population of T cells was not detected, this would mean that the patient had had a clonal T cell proliferation for 15 years, which is rather unlikely.

We look forward to the results of Dr Slatter’s investigations to ascertain whether these conditions characterised by eosinophilia represent Th2 diseases.

Book reviews


The title promises much, but in fact this volume in the Cancer Surveys series aims to illustrate a series of models for selected cancers, and comprises a mixture of short articles, review articles, and reports on cell biology, and molecular pathology. Interpretation of “model” includes animal, cell culture, and theoretical systems, so that the collection is little heterogeneous. Of the 16 chapters, four are concerned with breast cancer (the article on human breast cancer by Drs Walker and Varley is particularly well presented) and two with colorectal cancer. The other half of the book deals with some molecular aspects of thyroid and pancreatic cancers, lymphomas, tumour metastasis and, slightly curiously, prospects for cervical cancer vaccines. There is also a brief review of oncogenes, growth factors, and control of the cell cycle, and a short biography of each of the 26 authors, many of whom are from the Imperial Cancer Research Fund Laboratories. There is a certain amount of repetitiveness and, curiously, the nomenclature for p53 has been changed to TP53 throughout, except in the references. References are mostly up to 1991, with a few from 1992. The book is well produced but sparsely illustrated, mostly with line diagrams and a few photographs of cell cultures or gels. It is difficult to know who buys this sort of book, but the reference lists could be useful to workers in the relevant fields.


It is with mixed feelings that I review this book. Having had the privilege of being trained by Dr Stokes, I cut my microbiological teeth on the fourth edition, and have frequently referred to it and subsequent editions. The changes in microbiology in the 90s and the role of the routine clinical laboratory have been addressed in the seventh edition.

The details of a busy routine microbiology laboratory are helpful, particularly in the prevailing climate. A clearer understanding of “value for money” is now