Letters

Colonisation of point of use water filters by silver resistant non-tuberculous mycobacteria

Point of use water treatment devices are often employed to eliminate the disagreeable taste that results from the use of chlorine to disinfect drinking water. These devices generally rely on granular activated carbon to remove chlorine and they contain silver as a bactericidal agent. This improves the taste of the water, so the consumer may perceive an improved water quality. However, previous studies have shown that bacteria present in tap water, including both innocuous and disease causing species, are able to multiply in carbon filters impregnated with silver and are released into the water passing through the filter.[1 2] The simplest devices available for home use are the "pour through" units: tap water is poured into the top of the unit, passes through the filter by gravity, and is stored in the bottom of the unit for use.

To date no one has reported the ability of non-tuberculous mycobacteria (NTM), including *M avium*, to colonise carbon filters. *M avium* often infects AIDS patients and there is evidence of waterborne transmission of *M avium* in such patients.[3 4] Accordingly we conducted several experiments to determine if NTM are able to colonise a commercial pour through device. Three NTM species, *M avium*, *M fortuitum*, and *M mucogenicum*, were tested in separate experiments. Filters were prepared as recommended by the manufacturer and placed in the filter container/storage unit. Suspensions of each mycobacterial species were diluted in 2 litres of tap water to a final concentration of 9 colony forming units (CFU)/ ml for *M fortuitum*, 24 CFU/ ml for *M mucogenicum*, and 145 CFU/ ml for *M avium*. No attempt was made to remove indigenous mycobacteria from the tap water. The tap water/ mycobacteria suspension was passed through the filter and the entire filter/storage unit was stored at room temperature overnight. The filtered water was then removed and 2 litres of fresh uninoculated tap water were filtered and the unit stored at room temperature. After one week, and each week thereafter for eight consecutive weeks, the filtered water was removed, analysed for mycobacteria, 2 litres of fresh uninoculated tap water were filtered, and the unit again stored at room temperature.

No mycobacteria were recovered at weeks 1 and 2 (< 1 CFU/ ml). *M avium* were detected at week 3 at 22 000 CFU/ml, peaked at week 5 at 47 000 CFU/ ml, and then decreased to 3 CFU/ml at week 8. All *M avium* isolates recovered had identical 16S rRNA gene sequences. *M fortuitum* and *M mucogenicum* were never detected in the filtered water (< 1 CFU/ ml, fig 1).

The three mycobacterial species evaluated were found to differ in sensitivity to silver by a disk diffusion assay. *M avium* was able to grow in the presence of 1000 µg/ml silver, whereas *M fortuitum* and *M mucogenicum* were inhibited at 50 µg/ml. A survey of 45 NTM drinking water isolates, representing 11 different species, revealed 26 isolates (57%) that were resistant to 1000 µg/ml silver, including all 20 *M avium* isolates tested.

These results suggest that drinking water containing silver resistant NTM, treated by point of use filtration that relies on the bacteriocidal effect of silver, could pose a health risk for immunocompromised consumers. For such consumers, boiling the filtered water might be the prudent option.

M R RODGERS
B J BLACKSTONE
A L REYES
T C COVERT
National Exposure Research Laboratory, US Environmental Protection Agency, Cincinnati, Ohio 45268, USA

1 Daschner FD, Ruden H, Simon R, *et al.* Microbiological contamination of drinking water in a commercial household water filter system. *Eur J Clin Microbiol Infect Dis* 1996;**15**:233–7.
2 Geldreich EE, Taylor RH, Blannon JC, *et al.* Bacterial colonization of point-of-use water treatment devices. *J Am Water Works Assoc* 1985;**77**:72–80.
3 Nightingale SD, Byrd LT, Southern PM, *et al.* Incidence of Mycobacterium avium-intracellulare complex bacteria in human immunodeficiency virus-positive patients. *J Infect Dis* 1992;**165**:1082–5.
4 von Reyn CF, Maslow JN, Barber TW, *et al.* Persistent colonisation of potable water as a source of Mycobacterium avium infection in AIDS. *Lancet* 1994;**343**:1137–41.

Laboratory diagnosis of vaginal discharge (ACP Broadsheet No 153)

This ACP Broadsheet[1] (was known as Best Practice—ED) is a useful document, which is likely to become the gold standard for the laboratory investigation of patients presenting with vaginal discharge. However, the authors also attempt to deal with clinical situations in which vaginal discharge is unlikely to be the presenting complaint and here their advice is contentious. They recommend that vaginal swabs submitted from patients with pelvic inflammatory disease (PID) should undergo "full culture" with special media for the isolation of coliforms and anaerobes in addition to routine investigation for *N gonorrhoeae*, bacterial vaginosis, *Trichomonas vaginalis*, and *Candida* spp. Coliforms and anaerobes are indeed implicated in PID, possibly as secondary invaders from the vagina, but the temporal association and pathogenesis are unclear. Culture of a vaginal swab from a patient with PID is analogous to culture of a throat swab from a patient with pneumonia. Full culture of a vaginal specimen is not generally recommended in the investigation of PID[2] as it does not aid in diagnosis or determine the choice of therapeutic antimicrobial agents.

"Full culture" is also recommended when a vaginal swab is submitted in clinical situations such as "premature labour, prolonged rupture of membranes, spontaneous rupture of membranes, antepartum haemorrhage, and threatened abortion." In these circumstances, the diagnosis of infection (amnionitis) and the timing of antimicrobial therapy are based on pre-agreed clinical criteria and empiric antimicrobial therapy is directed at a range of organisms implicated in the condition. Gram stain and culture of amniotic fluid have been recommended but even these are of limited value in individual patients.[3]

In PID and the other clinical situations mentioned full culture of vaginal specimens for coliforms and anaerobes is unwarranted and therefore an unnecessary expense.

M NOONE
North Tees General Hospital, Hardwick, Stockton on Tees, UK

1 Macsween KF, Ridgway GL. The laboratory investigation of vaginal discharge. ACP Broadsheet No 153. *J Clin Pathol* 1998;**51**:564-7.
2 McCormack WM. Pelvic inflammatory disease. *N Engl J Med* 1994;**330**:115–19.
3 Newton ER. Chorioamnionitis and intraamniotic infection. *Clin Obstet Gynecol* 1993; **36**:795–808.

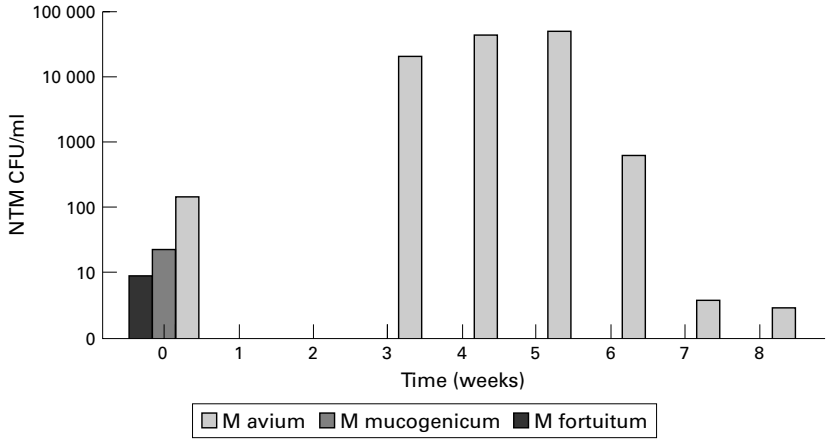

Figure 1 Non-tuberculous mycobacteria colonisation of point of use filter systems.

Book reviews

Color Atlas of Differential Diagnosis in Exfoliative and Aspiration Cytopathology. By S R Kini. ($150.00.) Lippincott Williams & Wilkins, 1998. ISBN 0683 30675 8.

The title of this book accurately reflects the objectives of the text. The concept is based around teaching methods used by the author for postgraduate training. However, this book is clearly not aimed solely at trainees in pathology, and much of the information would be of benefit to anyone who routinely reports cytopathological material. The format of the book consists of text, tables listing features that may be of use in differential diagnosis, and numerous illustrations.

The text is well written and it is gratifying to see that the gynaecological cytology section does not restrict itself solely to

Bethesda terminology. The illustrations are of very high quality throughout and the use of colour is helpful.

The book, while overall of high quality, does have two major limitations. There are very few illustrations of Giemsa stained material. The author makes this clear from the beginning, stating a strong preference for Papanicolaou stained slides, but this does limit the usefulness of the images for those of us who use Giemsa stained material extensively in routine practice. The other major limitation is the manner of presentation of the illustrations at the end of each chapter. This makes flicking back and forward from text to illustrations necessary, although after a while this ceases to be a major irritation.

Overall, I would recommend this book to practising cytopathologists as there are many useful lessons presented, although I think that most general trainees would find this text quite heavy going.

NEIL ANDERSON

The Prothrombin Time. World Health Organisation. (Free to laboratories.) Geneva: WHO, reference No WHO/Lab/98.3.

Dr Poller has produced, on behalf of the World Health Organisation, a monograph on the prothrombin time (used synonymously with thromboplastin time or Quick test). This is a technical document and does not intrude on clinical or therapeutic grounds nor does it concern itself with any aspect of oral anticoagulation other than monitoring.

This brief publication (32 pages in all) provides all the information on the prothrombin time (historical, manufacturing, technical, and scientific) that one could possibly need—and probably a lot more than most require. For the latter, interested only in a particular issue, the index is clear and thorough.

The author has had a long and distinguished association with this coagulation test and its technical ramifications. This shows in the loving and exquisite detail of the practical instructions. Although not a rollicking good read it is written clearly and is easy to understand. These views apply equally to the higher mathematical hieroglyphics of ISI calibration and the cookery class homeliness of tissue thromboplastin extract preparation.

If you need to know anything about the prothrombin time you should read this monograph.

P KESTEVEN

Mucosal T Cells. Edited by T T MacDonald. ($104.50.) Karger, 1998. ISBN: 3 8055 6722 7.

This is a timely, detailed, up to date reference work on the key roles played by T cells in different compartments of the mucosal immune system. From a potentially vast subject, the editor has been sensible in selecting the more pertinent topics on which to focus. The book will prove popular both with those involved in basic research and with others in more applied human clinical research, from asthma to inflammatory bowel disease. In general, the data are clearly presented, and the areas where our knowledge is currently deficient are sensibly discussed, with pointers towards areas where future progress is likely.

A wide range of fascinating topics is dealt with clearly and concisely. To provide but a small sample, these include the basic biology of γδ T cells, TH1 and TH2 subdivisions within the mucosal environment, the role of T cells in oral tolerance, and data on how mucosal T cells bias mucosal B cells towards IgA responses.

The book will be most useful as a detailed reference source, both for students in the field and also for more seasoned researchers, whether they be interested particularly in the mucosal system or have a more global interest in the immune system. There is still plenty to learn and this volume will excite much new interest in mucosal immunity.

D J UNSWORTH

Cell Death and Diseases of the Nervous System. Edited by V E Koliatsos and R R Ratan. ($145.00.) Humana Press, 1998. ISBN 0 896 03413 5.

This multiauthor textbook presents a comprehensive overview of mechanisms of cell death in the brain. The book is divided into four general sections which cover the cellular and molecular mechanisms of cell death, animal models, nerve cell death in human diseases, and approaches to treatment. The authors cover a wide range of disciplines from basic and applied neuroscience to pathology, neurology, and therapeutics. Both apoptotic and non-apoptotic mechanisms of cell death are considered for neurones and glial cells, and this appears to be the first book which concentrates on these mechanisms in diseases of the nervous system. As a neuropathologist I was particularly interested in the large central section on nerve cell death in human disease, which covers a wide range of topics from mitochondrial disorders to infectious and transmissible diseases, hypoxia/stroke, trauma, and neurodegenerative diseases including Alzheimer's disease, Huntington's disease, Parkinson's disease, and motor neurone disease.

I found this an interesting work which, because of its focus on the central nervous system, would be of particular interest to neuropathologists and both clinical and applied neuroscientists. However, there is much here to interest others who are working in the area of cell death, since there are few if any competitors.

The book is well produced and helpfully illustrated by line diagrams and monochrome prints, with occasional colour illustrations, the references are as up to date as is reasonable, and the index is helpful. It is self recommending for those working on diseases involving the central nervous system, but should also be considered as a library purchase for those interested in the general field of cell death.

J W IRONSIDE

Transplantation Pathology. Edited by C L Berry. (£96.00.) Springer, 1999. ISBN 3 540 64096 7.

It is now likely that many diagnostic histopathologists will fairly regularly encounter specimens derived from transplant patients. A good overview of the relevant pathology, which is very wide ranging, is therefore highly desirable and represents a stated aim of this book on transplantation pathology.

The first two chapters set the scene with overviews of transplantation immunology and infection. Thereafter chapters on renal, liver, bone marrow, heart, and lung transplant pathology form the core of the book, with a final chapter on CNS pathology acting to illustrate potential ways forward. Each chapter is a detailed distillation of knowledge, with a laudable lack of typographical errors, the most obvious being the inversion of figures 8 and 9 in the liver chapter. Variability in the writing style and use of diagrams, tables, and photomicrographs is a result of the multiauthor nature of the book. In general, it does not attempt to be a diagnostic bench book but provides a solid theoretical background to the areas covered. However, the chapters on liver transplantation and, to a lesser extent, heart and lung transplantation provide a greater degree of diagnostic guidance.

Overall, this volume succeeds in its aim to be a useful reference source for the specialist and it will inform the generalist. Its price may be slightly high for some budgets, considering its relatively slim profile.

A R McPHADEN

Notices

Melanoma '99

Brighton Conference Centre, Brighton, UK

29 and 30 September 1999

The Melanoma Study Group and the Association of Clinical Pathologists announce the fourth Brighton Melanoma Conference, Melanoma '99, which is being held jointly with the annual national scientific meeting of the Association of Clinical Pathologists. Speakers include Lorenzo Cerroni, Alistair Cochran, Kerry Crotty, and Sabine Kohler.

Further information: Melanoma '99 Secretariat, Association of Clinical Pathologists, 189 Dyke Road, Hove, East Sussex BN3 1TL, UK; tel +44 (0)1273 775700; fax +44 (0)1273 773303; email: melanom99@pathologists.org.uk

British Division of the International Academy of Pathology

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Further details from: Mrs C Harris (Administrative Secretary), PO Box 73, Westbury on Trym, Bristol BS9 1RY; tel +44 (0)117 907 7940; fax +44 (0)117 907 7941; email: bdiap@cableinet.co.uk

Correction

We are informed that in the paper entitled "How many lymph nodes to stages colorectal carcinoma?" (February 1998, vol 51, pages 165-6), the author list should have included **H Kulacoglu** as second author.

Instructions for Authors

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References

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1 Fletcher CDM, McKee H. Sarcomas - a clinicopathological guide with particular reference to cutaneous manifestations. I. Dermatofibrosarcoma protuberans, malignant fibrous histiocytoma and the epithelial sarcoma of Enzinger. *Clin Exp Dermatol* 1984;**9:**451–65.
2 Washington JA. Conventional approaches to blood culture. In: Washington JA, ed. *The detection of septicemia*. West Palm Beach, Florida: CRP Press, 1978:41–87.

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1 International Committee of Medical Journal Editors. Uniform requirements for manuscripts submitted to biomedical journals. *BMJ* 1991;**302:** 338–41.
2 International Committee of Medical Journal Editors. Protection of patients' rights to privacy. *BMJ* 1995;**311:**1272.

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Revised January 1999

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