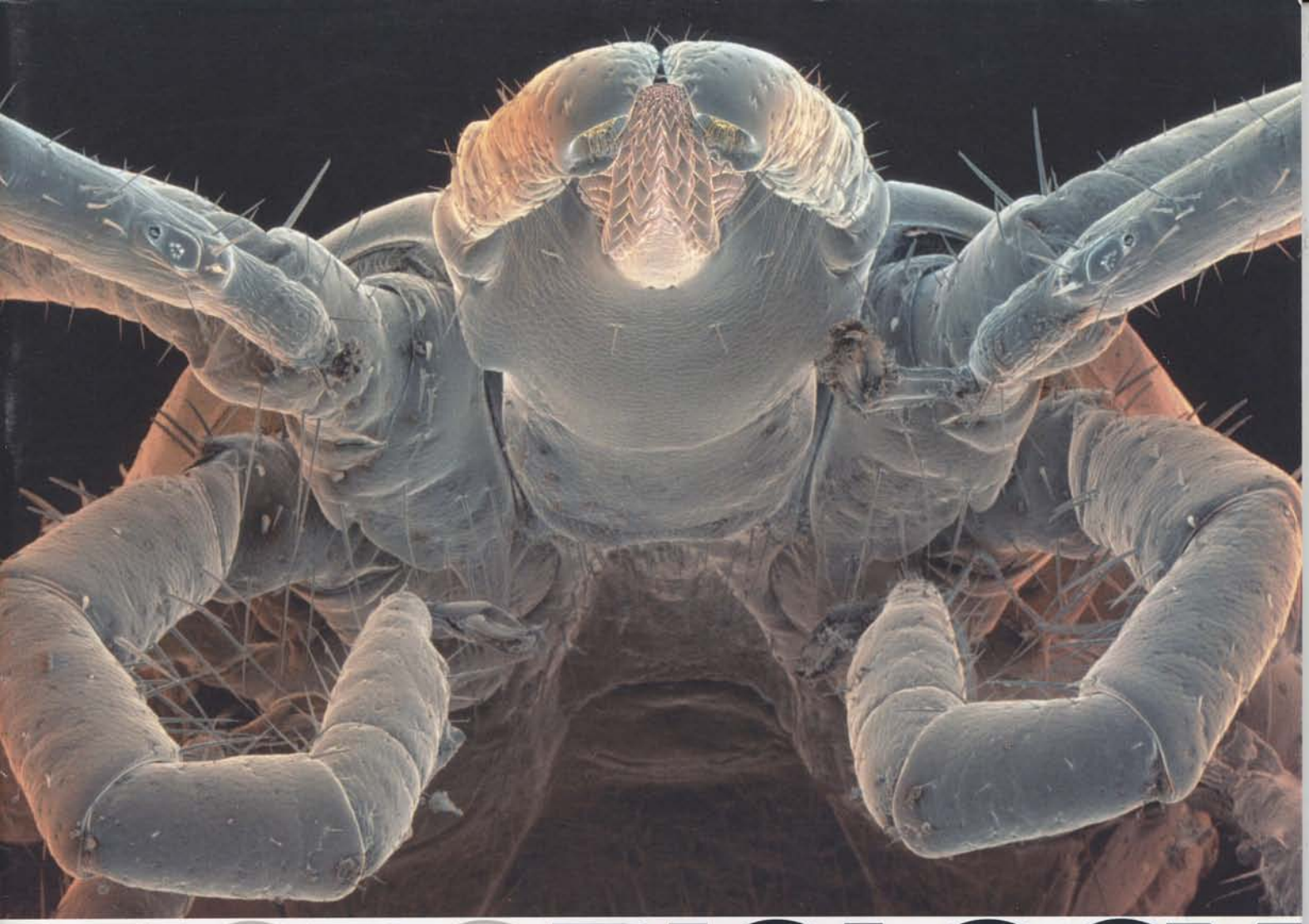


# MICROBIOLOGY TODAY

QUARTERLY MAGAZINE OF THE SOCIETY FOR GENERAL MICROBIOLOGY VOLUME 30 NOVEMBER 2003

Emerging infectious diseases of wildlife
Wildlife disease surveillance
The threat of West Nile virus in the UK
Seal distemper outbreak 2002
The mystery of Lyme disease
Tick-borne relapsing fever
Emerging bartonellosis
Genetic control in TSEs
Prions in the wild

# Contents

**SGM Headquarters**
Marlborough House,
Basingstoke Road, Spencers
Wood, Reading RG7 1AG
Tel. 0118 988 1800
Fax 0118 988 5656
email mtoday@sgm.ac.uk

**SGM Website**
http://www.sgm.ac.uk

**Editor**
Dr Gavin Thomas

**Editorial Board**
Dr Pauline Handley
Professor Tony Nash

**Managing Editor**
Janet Hurst

**Production Editor**
Ian Atherton

**Assistant Editor and Book Review Manager**
Janice Meekings

**Contributions**
These are always welcome and should be addressed to the Editor (c/o SGM Headquarters).

**Copy Dates**
Last dates for receipt of copy at Marlborough House are:
*General Copy*
February 2004 issue 8 December
May 2004 issue 8 March
*Advertisements (CRC)*
February 2004 issue 12 January
May 2004 issue 23 April

**Advertisements**
All enquiries should be sent to:
Peter Horncastle, Hardy Advertising, Granville House, 112 Bermondsey Street, London SE1 3TX
Tel. 0207 378 1579
Fax 0207 378 6421
email peter@hardyadvertising.co.uk

**Subscriptions 2004**
NON-MEMBERS
*Microbiology Today* £50.00 (US$85.00)
MEMBERS
All members receive *Microbiology Today*. In addition they may take any of the Society's journals.
*Ordinary Member*
Membership Subscription (inc. *Microbiology Today*) £45.00 (US$78.00)
*Microbiology/JGV/IJSEM* £85.00 (US$160.00)
*JMM* £45.00 (US$78.00)
*Student or Retired Member*
Membership Subscription (inc. *Microbiology Today*) £20.00 (US$35.00)
*Microbiology/JGV* £40.00 (US$75.00)
*IJSEM* £85.00 (US$160.00)
*JMM* £45.00 (US$78.00)
*Undergraduate or School Member*
Membership Subscription (inc. *Microbiology Today*) £10.00
*Corporate Member*
Membership Subscription (inc. *Microbiology Today*) £350/500
US Office of Publication: *Microbiology Today*, c/o Mercury Airfreight International Ltd, 365 Blair Road, Avenel, NJ07001, USA. Postmaster: send US address corrections to *Microbiology Today* c/o this address.

 ISSN: 1464-0570

**Above:** Coloured scanning electron micrograph of the deer tick, *Ixodes* sp. *David Scharf / Science Photo Library*

**Vol. 30, Part 4, November 2003**

In this issue we focus on emerging infectious diseases of wildlife. Some of these pathogens affect a wide range of animals, including domestic species and humans, whereas others are confined to their wildlife host and can devastate populations.

Sarah Cleaveland, in her overview, explains that the interaction between wildlife and human health is complex, with implications that pose real challenges to microbiologists and veterinarians (p. 155).

In the UK, the government is funding a project to survey the incidence of wildlife disease, as Paul Duff of the VLA describes (p. 157). Antibodies to West Nile virus, which is sweeping North America, have been found in UK wild birds. Ernie Gould assesses the risk to humans in Britain (p. 160).

Currently of no threat to humans, but deadly to seals, Phocine distemper virus caused the deaths of more than 22,000 animals in a recent outbreak off the UK East Coast, as Tom Barrett and his colleagues describe on p. 162.

Vectors have an essential role in many zoonoses, and ticks transmit a range of infections, such as Lyme disease, as Ruth Montgomery reports (p. 165), and Relapsing fever which as Sally Cutler and Alison Talbert explain, causes severe mortality in Tanzania (p. 167). Christoph Dehio and Anna Sander describe how Bartonellas are another rising cause for concern, spread by vectors ranging from the human louse to cat fleas (p. 168).

Prions may or may not be microbes, but TSE diseases are important worldwide. In the USA deer populations are affected by Chronic wasting disease, according to Beth Williams and Michael Miller (p. 172). On p. 170 Wilfred Goldmann considers whether or not wildlife species are more prone to TSE diseases than domesticated animals.

Finally, in Comment, Colin Howard and Geoffrey Schild stress the importance of veterinary and animal microbiology as exemplified by the other articles in the magazine, and ask, 'does the subject receive enough support?'

These articles appear in addition to all the regular features and reports of Society activities.


## Articles



## Regular Features



## Other Items

# Public Affairs

## Editorial

I am very honoured to be taking over as the new Editor of *Microbiology Today* and am relishing the challenge of keeping MT at the top of academic society publishing. Comparing MT to my first copies of the *SGM Quarterly* from 1995, the improvement in production quality is striking, with stylish layout and copious use of colour; it is no wonder the team has been winning so many awards recently! In my role I hope to maintain the high quality of the content that has been achieved by previous Editors, most recently Meriel Jones, especially in the production of interesting themed issues. One important function of MT is that it offers the opportunity to present microbiology to a larger community, including government and the general public, and I will strive to use this medium to improve the awareness of microbiology and the SGM to a larger audience, while retaining the appeal to members' interests.

On a much sadder note, we include in this issue a special obituary for long standing SGM member Dr David Kelly, who tragically took his own life earlier this year. Dr Kelly was a microbiologist who for many years had worked hard with the government in detection of bioweapons in Russia and Iraq and had the respect of all in this area. It is sad that a scientist, or any member of the public, can be put under such a level of intense public pressure that results in such tragic consequences. I only hope we can listen to the words of his daughter and learn to be more compassionate to those around us.

● ***Gavin Thomas***

*Public Affairs Administrator Faye Jones reports on some activities to raise the profile of science to government and opinion-formers.*

### Launch of the Biosciences Federation

The Biosciences Federation was officially launched at the House of Lords on Monday 15 September 2003. It was founded in December 2002 as an umbrella organization for the UK's biological sciences to speak with a stronger, more unified voice on matters of national importance and public affairs. Key aims of the Federation include promotion of liaison, dialogue and interactions within the diverse community of bioscientists on common issues that relate to teaching and research. The Federation also aims to provide opinion and information to assist the formulation of public policy and to promote wide and open debate about the practical and ethical issues surrounding developments in the biosciences. With 26 member societies, including SGM, the Federation represents thousands of life scientists.

About 200 parliamentarians, members of the press and representatives of the member societies attended the reception that was hosted by Lord Jenkin of Roding. Addressing the guests were Professor Colin Blakemore FRS, the Federation's current President, and Lord Sainsbury of Turville, Parliamentary Under-Secretary of State for Science and Innovation who spoke about the importance in Parliament of communicating with one strong voice. Sir John Sulston, Physiology/Medicine Nobel Prize winner in 2002, whose award was also celebrated at the event, also emphasized the importance of a body such as the Biosciences Federation.

Attending on behalf of SGM were David Hopwood, past President (and member of the Biosciences Federation Council), Hugh Pennington, incoming President, Geoffrey Schild, Professional Affairs Officer, and Peter Stanbury, Treasurer, plus several members of staff.

### Royal Society of Chemistry

#### Science and the Parliament Day
#### 12 November 2003, The Signet Library, Edinburgh

With the environment as its theme this year, the Royal Society of Chemistry (RSC), in association with other leading science organizations, will soon be holding its popular annual event to promote science to the Scottish Parliament and Executive.

Chairing will be Professor Wilson Sibbett, Chair of the Scottish Science Advisory Committee. Guest speakers include Sarah Boyack, Convener of the Environment and Rural Affairs Committee, Professor James Curran, Environmental Futures Manager at Scottish Environmental Protection Agency, Eleanor Scott, Environment spokesperson for the Green Party and Jim Wallace, Deputy First Minister. Participants will be invited to visit the Scottish Parliament to observe a specially planned science debate.

SGM will be attending as part of its Microbiology Awareness Campaign, with a display highlighting how and where microbiology and microbiologists can help in the protection and bioremediation of the Scottish environment.

Attendance at the event is by invitation only.

### Hitting the headlines

#### 153rd SGM Meeting, UMIST

For another successive year, the autumn meeting experienced an increase in attendance. A record number of nearly 800 delegates learned about some exciting recent findings and there was good coverage of the papers and posters in the media.

Belinda O'Grady from the University of Reading had her research into probiotics to combat *Helicobacter pylori* described on *BBC News Online*. This article also covered Dr Mark Stevens' research at the Institute for Animal Health into the identification of important colonization genes in *Escherichia coli* O157 and O26. Dr Stevens' work was the basis of a further *BBC News Online* article, as well as articles in the *Scotsman* and the *Edinburgh Evening News*. One final issue highlighted by *BBC News Online* was Dr Michael Prentice's research that failed to find traces of *Yersinia pestis* DNA in teeth taken from plague victims, throwing doubt on the cause of the Black Death. This story was also carried by the news agency Reuters and featured in the 13 September issue of *New Scientist*.

BioMedNet covered some of the presentations at the meeting, including research by Dr Bart Keijser, University of Amsterdam, into the survival of food-spoilage bacteria, such as *Bacillus*, during pasteurization, and Professor Brendan Wren's findings, London School of Hygiene and Tropical Medicine, that *Campylobacter jejuni* is unique amongst prokaryotes because it contains genes that encode for a general protein glycosylation system found in eukaryotes.

*New Scientist* also covered the Black Death story in their weekly science report television programme, shown 10–16 September on *The Discovery Channel UK* as part of 'Science Night'. *New Scientist* Reports can be seen every Tuesday at 2000, 2105, 2210 and 2315 and are repeated on Wednesday at 1200, 1305, 1410 and 1515.

To read some of the articles mentioned see:

- http://news.bbc.co.uk/1/hi/health/3084882.stm
- http://news.bbc.co.uk/1/hi/scotland/3088362.stm
- http://www.news.scotsman.com/archive.cfm?id=988532003
- http://www.news.scotsman.com/archive.cfm?id=990562003
- http://news.bbc.co.uk/1/hi/health/3097934.stm
- http://www.reuters.co.uk/newsArticle.jhtml?type=topNews&storyID=3423120
- http://www.newscientist.com/news/news.jsp?id=ns99994149

● ***Faye Jones, Public Affairs Administrator***

*Continued on p. 171*

# Emerging infectious diseases of wildlife

Sarah Cleaveland

Many pathogens are not fussy about their host species. As Sarah Cleaveland describes, this means that wildlife hosts can play a key role in the emergence of human and domestic animal diseases.

LEFT:
Rapid decomposition and scavenging of wildlife carcasses creates considerable difficulties for the collection of appropriate diagnostic samples. The development of robust and practical field techniques is required for effective wildlife disease surveillance.
PHOTOS SARAH CLEAVELAND (VULTURE) AND SARAH DURANT (LEOPARD)

We often think of human, veterinary and wildlife medicine as separate disciplines, but this distinction makes little sense when we consider the epidemiology of infectious diseases. Most pathogens that cause human and animal diseases are ecological generalists – they can infect and be transmitted by more than one host species – and for many, the spectrum of hosts includes wildlife species. This is true for almost half (44 %) of all pathogens that cause human diseases and 70 % of the pathogens that cause the most internationally important veterinary diseases (e.g. rinderpest, foot-and-mouth and avian influenza).

## Multi-host pathogens and emerging infectious diseases

Although we know very little about the dynamics of infectious agents in most wildlife populations, wildlife hosts appear to play a key role in the emergence of human and domestic animal diseases. A recent analysis has shown that pathogens that can infect wildlife are more than twice as likely to cause emerging diseases (in both humans and livestock) as those that do not affect wildlife. This includes newly recognized pathogens, such as HIV-1, HIV-2, BSE, Hendra virus, Nipah virus and the SARS coronavirus, as well as those that are spreading (re-emerging), such as rabies and West Nile virus.

Although we currently lack denominator data on wildlife diseases (and hence cannot quantify risk factors for emergence), pathogens that infect several host taxa have also been responsible for most emerging wildlife epidemics, including several that have threatened endangered species, such as rabies, which decimated populations of the Ethiopian wolf (one of the world's rarest carnivores) and canine distemper, which has caused major declines in Channel Island foxes and Lake Baikal seals. Generalism appears to be a common characteristic of emerging pathogens, whether in human or animal populations.

Several factors may underlie disease emergence, but in practice, most emerging human diseases are associated with ecological changes, such as changes in land-use, environment, climate, human demography or animal and human movement patterns. Many of these factors can lead to increased human–wildlife contact and thereby facilitate transmission of potentially pathogenic agents that are maintained in wildlife hosts. For example, deforestation and encroachment into new habitats have been linked with the emergence of California encephalitis virus, Ross River virus, Ebola and Marburg viruses (all of which co-infect wildlife).

## Human factors in emerging wildlife diseases

For emerging diseases of wildlife, similar ecological and anthropogenic factors come into play. For example, the

RIGHT AND BELOW:
Recent wildlife epidemics of rabies and canine distemper in wild carnivores in the Serengeti, Tanzania, have been associated with a rapid expansion of domestic dog populations.
PHOTOS SARAH CLEAVELAND

appearance of canine distemper as a devastating new disease in Serengeti lions has been associated with a rapid expansion of domestic dog populations living adjacent to the park; a marked increase in mycoplasmal conjunctivitis among passerine birds in north America has been attributed to habitat changes and artificial feeding; and the emergence of chytridiomycosis (a fungal skin disease), which has resulted in major population declines in amphibians in Australia and the Americas, has been linked to climate change and movements of captive amphibians.

While medical attention may focus on zoonotic risks from wildlife infections, disease risks from humans are a growing concern for conservationists. Disease transmission from both local people and tourists has long been recognized as a threat for wild primates – outbreaks of measles in mountain gorillas, polio and pneumonia in chimps and, more recently, Ebola in great apes have all been associated with human contact and proximity. However, human diseases are also emerging as threats to non-primate vertebrates, and recent outbreaks of *Mycobacterium tuberculosis* (presumed to originate from humans) have led to high mortality in meerkats and banded mongooses in southern Africa. With HIV/AIDs causing widespread immunosuppression in the human population and enhancing the potential for pathogen transmission, the risk of future wildlife outbreaks from humans must surely be set to increase.

## ● Implications of wildlife infections in the control of human emerging diseases

The link between wildlife and human health poses several problems. First, the lack of knowledge of infection dynamics in wild animal populations limits the development of effective strategies for disease control. Even in the public health sector, disease surveillance is often not a high priority. In almost all wildlife populations, surveillance is rudimentary or non-existent – and this is particularly true in the developing world where many emerging diseases have originated. Detection of pathogens in free-living wildlife is notoriously difficult, hampered by the enormous practical problems of finding, collecting and storing appropriate samples under field conditions, as well as the lack of species-specific diagnostic tests. Even where considerable investment has been made (as with bovine tuberculosis and badgers in the UK), the complexities of infection dynamics in multi-host systems make it difficult to identify optimum methods of control.

Second, even where wildlife hosts and/or reservoirs have been identified, the options for control in wildlife are limited. Many strategies, such as culling and creation of barriers, invariably result in harm to wild animals. Conventional approaches to disease control in animals, such as vaccination or treatment have limitations in wildlife populations. Specific vaccines and treatments are often unavailable or untested for wildlife, and delivery in field settings is beset by logistic, financial and ethical considerations. The success of wildlife oral rabies vaccination campaigns in Europe and North America points a way to the future and has stimulated research into other non-lethal approaches, such as sterilization.

A third consequence of wildlife involvement in human diseases is the potential threat to the wildlife tourism industry. The economic damage caused by a decline in visitors to countries suffering from SARS and Ebola virus clearly highlights this problem. Equally clear is the important lesson learnt from the SARS epidemic about the need for open exchange and dissemination of epidemiological data. Balancing these needs presents a dilemma for wildlife managers, particularly in those countries dependent upon wildlife tourism for economic development. Additional dilemmas will invariably arise as molecular tools increasingly allow detection of pathogens in an expanding range of wildlife hosts. A major challenge for the future will be the epidemiological interpretation of these results and appropriate evaluation and management of potential disease risks.

In summary, the links between infectious diseases of wildlife and public health have far-ranging impacts and implications that pose considerable challenges to medical scientists, veterinarians and wildlife managers. To date, there has been little integration between these sectors – wildlife ecologists tend to show little interest in human health and public health scientists often have little knowledge of wildlife issues. But this interface provides exciting opportunities to develop innovative and collaborative approaches that will mitigate emerging disease risks for humans and minimize adverse impacts on wildlife.

● *Dr Sarah Cleaveland is a Lecturer in Tropical Animal Health at the Centre for Tropical Veterinary Medicine, University of Edinburgh, Easter Bush, Roslin, Midlothian, EH25 9RG, UK.*
*Tel. 0131 650 6404; Fax 0131 651 3903*
*email sarah.cleaveland@ed.ac.uk*

# Wildlife disease surveillance by the Veterinary Laboratories Agency

Paul Duff

From small beginnings, and although necessarily limited by its budget, the VLA Scheme, in collaboration with many organizations, has developed to become the first government-funded project for wildlife disease surveillance in England and Wales.

## Are diseases of wildlife important?

It is increasingly recognized, worldwide, that wildlife may be important hosts for diseases that are a risk to the health of man and his domesticated stock. Traditionally, we may have considered our wildlife to be a wholly beneficial resource and that diseases such as rabies, plague and anthrax occurred in other countries. But in fact, these three have occurred in England historically and it is certainly not out of the question that they may occur here again. The problems caused by emerging, or introduced, infectious diseases of wildlife will undoubtedly increase in the coming decades for a variety of reasons, not least of which is the increasing global movement and 'mixing' of organisms; from bacteria and viruses to birds, humans and other animals. As the physical barriers between potential pathogens and hosts are removed, the opportunities for infection and disease increase.

This article describes the VLA (Veterinary Laboratories Agency) Diseases of Wildlife surveillance scheme and how it investigates incidents of wildlife mortality and emerging disease. This work is mainly carried out on behalf of the government (specifically Defra: the Department for Environment, Food and Rural Affairs), and is done at 14 Regional Laboratories and two university surveillance centres in England and Wales, supported by two large laboratories at Weybridge and Lasswade, Scotland (Fig. 1). Surveillance in wild animals shares similarities with that for domesticated stock, and it can be described as the routine collection of data on disease in wildlife populations. It should be noted that non-infectious conditions, such as toxicities, are equally important and are also investigated by Regional Laboratories.

A fundamental question is how significant is wildlife disease? There is no concise answer to this question but some aspects that require consideration are listed below.

- Wildlife can be reservoirs of zoonotic disease, e.g. Lyme disease, leptospirosis.
- Wildlife can be reservoirs of disease of domesticated stock, e.g. bovine tuberculosis in badgers and deer.
- Exotic pathogens may be introduced to the country by migrating wildlife, e.g. *West Nile virus* by migrating birds.
- New and emerging diseases may first appear in wildlife species, e.g. European brown hare syndrome, the first recognized leporine caliciviral infection.
- Wildlife disease may be a sensitive indicator of underlying environmental pollution.
- Wildlife disease incidents, with mass mortality, may be of concern to the public.
- Wildlife disease may be of conservation importance, threatening endangered populations, e.g. squirrel parapox disease in English red squirrels.

Why is surveillance for wildlife disease important?

- To assess which diseases are present in the UK.
- To assess the risks of these diseases to human and livestock health.
- To assess how much disease is present and how this may vary.
- To assess new patterns of disease.
- To investigate potentially new and emerging conditions.

The VLA, and its predecessor organizations, have investigated wildlife disease for several decades, particularly new and emerging disease, and produced many of the first reports of 'new' disease in Britain. These include the first report of bovine tuberculosis in badgers in the late 1960s, the first identification and isolation in culture of the parapoxvirus and the iridovirus that cause squirrel parapox disease and 'red-legged frog disease', respectively, and the first reports of European brown hare syndrome (EBHS) and Rabbit haemorrhagic disease (RHD).

The VLA is well placed to carry out wildlife surveillance in England and Wales for three key reasons. It has:

- A national network of diagnostic laboratories (because dead and dying wildlife does not travel well; see Fig. 1).


LEFT:
**Fig. 1.** VLA Regional Laboratory network regional diagnostic laboratories are required for national coverage. COURTESY VLA

**Table 1. A selection of wildlife surveillance schemes in the UK**

| Project/scheme | Function/purpose | Principal organizations involved | Comment |
|---|---|---|---|
| Bovine tuberculosis in badgers | Investigation of the epidemiology of bovine tuberculosis in badgers, particularly as it affects the national cattle herd. | Principally government agencies, but also contracts to non-government organizations. | The budget for this work is greater than the budgets for the remaining projects combined. |
| Wildlife Incident Investigation Scheme | To investigate incidents of suspected pesticide poisoning in wildlife (and other) animal species. | Defra: (RDS – Rural Development Scheme; PSD – Pesticide Safety Division, Central Science Laboratory). | Field investigation, necropsies and toxicological analysis. The WII Scheme has operated for many years. |
| VLA Diseases of Wildlife Scheme (described in text) | Surveillance for wildlife diseases in England and Wales. | VLA contracted to Defra. | First scheme of its kind in Britain. |
| Rabies surveillance | Rabies checks in wildlife, especially bats. | VLA contracted to Defra. | Investigations, publications, advice to government. |
| Stranded Cetacean (marine mammal) | Project Investigation of causes of death/stranding in cetaceans. | British Museum, Institute of Zoology, Scottish Agricultural Colleges. | Scientific publications of on-going work. Advice to government. |
| Species Recovery Project | Promotion of endangered wild species. | English Nature, Institute of Zoology. | As above. |
| University projects | Range of projects of variable size investigating disease or specific aspects of disease. | Various universities, e.g. disease in wild rodents at Liverpool Veterinary School. | Scientific publications of on-going work. |
| RSPCA wildlife hospitals | Treatment and welfare of wild species. | Four wildlife hospitals in England. | Scientific papers on disease investigations and treatments. |
| Other wildlife hospitals | Treatment and welfare of wild species. | A vast range of sizes and capabilities from single individuals to organizations. | Represented by bodies such as the British Wildlife Rehabilitation Society. Publications. |
| Institute (independent and governmental) projects – usually as part of wider investigations | Investigation of wildlife disease, e.g. Louping-ill investigation by the Moredun Institute. | e.g. Game Conservancy – game species. Centre of Ecology and Hydrology – avian viruses. | Publications. Disease control advice. Centres of excellence for specific diseases. |
| Single projects, often undertaken by individuals | Often investigate diseases in one wild species or in particular regions. | Private or funded. | There is a history of individuals investigating wildlife disease in Britain. |

OPPOSITE PAGE:
**Fig. 2.** European brown hare syndrome (EBHS), an acute viral hepatitis of hares, was first reported from Europe and the UK in the late 1980s. However examination of UK archived hare tissues revealed that this disease had occurred in 1982 and our oldest hare sera, from 1963, contained EBHS antibodies. This suggested that EBHS was not a 'new' disease as was first thought, but that it had been present in the UK for some time before it was recognized. [Duff, P. *et al.* (1997). In *Proceedings of the 1st International Symposium on Calicivirus ESVV.* Edited by D. Chasey & others.]
COURTESY P. DUFF

- Diagnostic depth and expertise in the veterinary disciplines, for example pathology, histopathology, parasitology, bacteriology, virology, biochemistry.
- Experience in investigating wildlife disease.

These enable pathologists to do follow-up field visits and to select the most appropriate diagnostic tests when investigating new diseases. Further research on new diseases has frequently been done by non-government organizations in collaboration with the VLA. For instance with the Institute of Zoology, London, on squirrel parapox and red-legged frog disease.

Since 1998, the Agency has been contracted by Defra to provide general surveillance for wildlife diseases in England and Wales. The resulting VLA Diseases of Wildlife Scheme has several operating guidelines, but it particularly focuses on investigating wildlife diseases where there may be potential risks to human health and the health of domesticated stock. Providing that basic criteria for submission are met, then necropsy examinations of wildlife are undertaken, with the person making the submission receiving a copy of the resulting reports.

Data from these examinations appear in the *Wildlife Quarterly Reports*, available online from the VLA website (www.defra.gov.uk/corporate/vla/science/science-end-survrep.htm). Data are also presented in other forms, including an Annual Report which, since 1995, has collected information from many organizations. This report is then sent by Defra to the Office International des Epizooties (OIE) which in turn produces a synthesis of the wildlife diseases reported throughout the world. The Annual Report was the first attempt in Britain to collate wildlife disease data from different sources and it is hoped that it will be a source of information for other investigators. The VLA Scheme also shares diagnostic testing and a diagnostic database with the Scottish Agricultural Colleges (SAC) and can thus lay claim to national coverage. An essential provision of the Scheme is its links to other VLA, and non-VLA, wildlife disease projects (see Table 1).

In 2001, the Scheme began surveillance for *West Nile virus* in British wild bird casualties. In the intervening period, more than 800 birds of 78 species have been examined and although neither *West Nile virus* disease nor virus has been found (antibody, however, has been detected by a collaborating laboratory) it is clear that much work still has to be done to elucidate the epidemiology of this disease.

## The scope of surveillance – some wildlife disease investigation and surveillance projects in Britain

It should also be appreciated that there are many other organizations (both Government and non-Governmental) which make their own valuable contribution to the investigation of wildlife disease, and some of these are listed in Table 1.

The purpose of this table is not to provide specific details (the author apologizes for any inaccuracies), but

to indicate that there is a wide range of initiatives looking at disease in wildlife in Britain.

## Future directions

At present Defra is reviewing the animal health and welfare strategy to improve the health and welfare of animals kept by man and to protect the public from animal disease. Veterinary surveillance, including information on wildlife disease, is an integral part of that review. One reason for the inclusion of wildlife, is that diseases such as West Nile fever and European bat lyssavirus infection may pose risks to the national health. The investigation of these diseases is primarily a veterinary task and, as such, the surveillance of wildlife disease falls within the remit of the Defra veterinary strategy. In view of the wide scope in this field of study, collaboration between the government veterinary laboratories and other organizations is required. However, it is equally apparent that any national surveillance projects will need to utilize the VLA Regional Laboratories, and that frontline passive work of this kind should continue at these locations.

● *Paul Duff is Leader of the Diseases of Wildlife Project for the VLA. He is based at VLA Penrith, Cumbria CA11 9RR, UK.*
*Tel. 01768 885 295; Fax 01768 885 314;*
*email p.duff@vla.defra.gsi.gov.uk*

# SGM Prize Journals ALPSP/Charlesworth Awards 2003

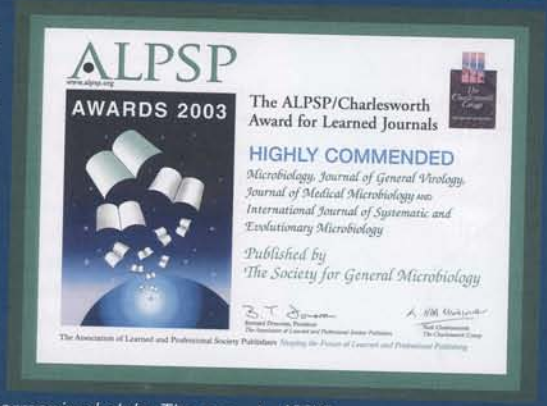
We are delighted to announce that the whole suite of SGM journals:

- *Microbiology*
- *Journal of General Virology*
- *Journal of Medical Microbiology*
- *International Journal of Systematic and Evolutionary Microbiology*

has been Highly Commended in the ALPSP/ Charlesworth Award for Learned Journals. This award recognizes outstanding journal design, including layout, cover and typography, with consideration for practical ease of use for readers and librarians. Journals must consist primarily of peer-reviewed scholarly research articles. The award is open to both commercial and not-for-profit publishers.

The judges of the SGM journals admired the overall branding and design of the collection, although considered that some individual issues had been allowed to be rather too thick and heavy for convenient library use.

Ian Atherton, Production Editor of *Microbiology Today*, who was responsible for the design, with help from his colleagues, and Executive Secretary, Ron Fraser, attended the ALPSP annual dinner on 18 September where they were presented with a certificate. Ron Fraser said, '*We are delighted to have received an ALPSP/ Charlesworth award for our four journals, recently re-designed in house to a harmonized style. The award joins the two we have received in previous years for our members' magazine Microbiology Today.*' The certificate will join the collection already adorning the wall of the conference room at Marlborough House.



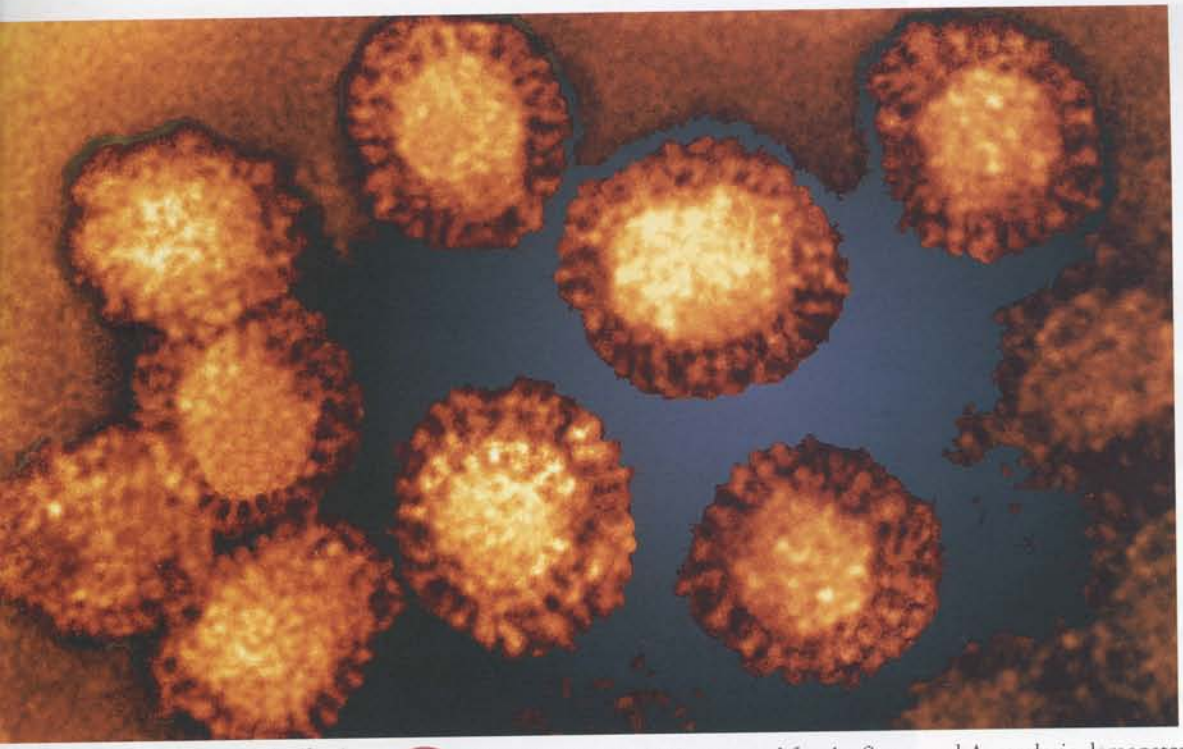

ABOVE:
Graham Lawley from Charlesworth (centre) presenting the certificate (shown below) to Ian Atherton (left) and Ron Fraser (right) at the ALPSP awards dinner held in the British Library, London, on 18 September 2003.

The winning journal in this category was *Feminist Review* (Palgrave Macmillan); other titles highly commended were *Nanotechnology* (Institute of Physics) and the *Journal of International Criminal Justice* (Oxford University Press).

The ALPSP represents the community of not-for-profit publishers and those who work with them to disseminate academic and professional information. See **www.alpsp.org** for further information.

# Does *West Nile virus* pose a threat to the UK?

Ernest Gould



Recent reports that antibodies to *West Nile virus* have been found in UK birds have caused concern. What are the risks of this potentially fatal disease to humans in Britain?

*West Nile virus* (WNV) appeared for the first time in North America in 1999, stimulating renewed interest in arthropod-borne diseases. The virus caused 62 human cases of encephalitis and six fatalities within a few months of its emergence in the New York area. Within 3 years, thousands of birds, horses and other wildlife species, including alligators and nearly 300 humans had died as the result of infection by WNV. The virus had also spread to Canada, the Caribbean and South America by early 2003. The perceived risk to humans increased when it was realized that the virus can be transmitted through blood transfusions, organ transplantations and even breast milk.

It is therefore not surprising that there was a significant reaction from the press and media when it was recently reported in the *Journal of General Virology* that scientists from CEH Oxford and Monkswood had found WNV-specific neutralizing antibodies in the sera of resident and migrant birds captured in Cambridgeshire, Dorset and South Wales. These results imply that the virus is circulating amongst the UK bird population. Ironically, evidence of the circulation of a non-pathogenic relative of WNV, *Usutu virus*, and another arthropod-borne virus, *Sindbis virus*, that causes polyarthritis in humans in Scandinavia, has been been almost totally ignored by the press.

## WNV before 1999

To put this into perspective we need to consider the history of the evolution, epidemiology and dispersal of WNV prior to 1999.

Since its emergence less than 20 years ago, WNV has circulated relatively harmlessly in Africa amongst mosquitoes and birds. Mosquitoes become infected when they feed on infected birds. They then reproduce and subsequently transmit the virus to other uninfected birds during the feeding process. These mosquitoes may infect humans or other vertebrates if they inadvertently feed on them, but human to human transmission is virtually unknown. Therefore, humans are dead-end hosts. Nevertheless, the presence of WNV-specific antibodies in humans, birds and horses throughout Europe, southern Asia and Australasia demonstrates the extent to which the virus has dispersed out of Africa, presumably carried by migratory birds which may have developed resistance to the virus. WNV has also been isolated from ticks, but their significance in its epidemiology has never been adequately investigated. Ticks could act as a secondary mechanism for survival of the virus through periods of low mosquito activity.

WNV is related to several other pathogenic viruses, including *Japanese encephalitis virus*, *St Louis encephalitis virus*, *Dengue virus* and *Yellow fever virus*, but prior to its emergence in North America in 1999, it was considered to be less important. However, with hindsight the observed bird die-off in Israel and the significance of outbreaks of West Nile encephalitis in Romania in 1996 and Russia in 1999/2000, involving several hundred human fatalities, were possibly underestimated. Recent evidence suggests that WNV in North America may be more virulent than some Old World strains due to mutational changes, but this remains to be confirmed. Regardless of this, it is now becoming clear that the combination of high-density human populations in rural and semi-urban areas, together with climatic and ecological conditions that favour mosquito breeding and bird conservation, provide ideal conditions for the efficient dispersal of viruses such as WNV.

## Symptoms of the infection

The great majority of infections are asymptomatic, nevertheless roughly 20 % result in the development of West Nile fever which has an incubation period of 2–6

ABOVE:
Coloured transmission electron micrograph of a group of West Nile viruses.
DR LINDA STANNARD, UCT / SCIENCE PHOTO LIBRARY

OPPOSITE PAGE:
A shop sign showing that the Canadians are doing their best to reduce the risk of exposure to mosquitoes!
PHOTO E. A. GOULD

days. Many patients experience a sudden onset of high fever with chills, malaise, headache, backache and pains in the joints, muscles and eyes when moved. A variety of other non-specific features such as anorexia, nausea, diarrhoea, coughing and sore throat may also occur. In some cases patients have a flushed face. A rash which appears 2–5 days after the onset of symptoms is seen in about 50 % of cases and is more common in children. The clinical symptoms of West Nile encephalitis, which is seen in less than 0·1 % of all infections, include aseptic meningitis, encephalitis, myelitis or combinations of the three. Only a small proportion of encephalitic cases are fatal and this is generally in old people.

### Future risks

What are the risks of exposure to WNV of humans in the UK? Although infectious WNV has never been isolated from birds in the UK, there is convincing serological evidence that it circulates amongst birds in the UK, Portugal, Spain, France and Poland. Moreover, infectious WNV has been isolated from horses in the Camargue region of France and also Italy. There is no record of birds dying from WNV infections in the UK. How can we explain this? First, since WNV is of relatively low virulence for humans even in epidemic situations, the disease may not be recognized in the UK where epidemics do not appear to occur. This is relevant because UK Public Health Laboratory Service figures show that 60 % of fatal encephalitic cases in the UK are undiagnosed! Second, the likelihood of exposure to the bite of a mosquito in the UK is significantly lower than in warmer parts of Europe. Third, the risk of infection would be highest amongst forestry workers, farm-workers, bird watchers, cross-country hikers, etc. Thus, the potential level of exposure to the bite of a WNV-infected mosquito for most individuals in the UK is extremely low. In summary, even if WNV is circulating annually amongst birds in the UK, the current risk to humans of developing West Nile encephalitis appears to be very low and since future prospects for vaccination against this virus appear promising, we probably do not need to live in fear of it in the UK.


● *Professor Ernest Gould, Centre for Ecology and Hydrology (CEH) Oxford, Institute of Virology and Environmental Microbiology, Mansfield Road, Oxford OX1 3SR, UK.*
*Tel. 01865 281630; Fax 01865 281696*
*email eag@ceh.ac.uk*

## Further reading

**Buckley, A., Dawson, A., Moss, S., Hinsley, S.A., Bellamy, P.E. & Gould, E.A. (2003).** Serological evidence of *West Nile virus*, *Usutu virus* and *Sindbis virus* infection of birds in the UK. *J Gen Virol* 84, 2807–2817.

**Gould, E. A. (2003).** Implications for Northern Europe of the emergence of *West Nile virus* in the USA. *Epidemiol Infect* 131, 583–589.

**Mackenzie, J.M., Barrett A.D.T. & Deubel, V. (2002).** Japanese encephalitis and West Nile viruses. *Curr Top Microbiol Immunol* 267, 1–10.

# Websites on infectious diseases of wildlife

Tony Nash

One of the leading centres for information on wildlife diseases is the National Wildlife Health Center (NWHC), an agency of the US Geological Survey, based in Madison, Wisconsin. This agency acts as a repository of information on wildlife diseases in North America. The NWHC hosts an excellent website (**www.nwhc.usgs.gov**) containing a comprehensive data base on wildlife diseases dating back to 1975 and features online publications and datasheets. You can download an entire *Field Manual of Wildlife Diseases*, a 51 chapter compendium dealing with diseases of birds or access current reports on emerging diseases, including the mysterious agent(s) associated with malformation of frogs and toads.

The NWHC site contains a wealth of information on two of the more recent threats to American wildlife, namely *West Nile virus* and Chronic Wasting Disease of deer and elk – a neurological disease related to BSE (see reports in this issue of *Microbiology Today*). The reader can find background information on these diseases and access more up-to-date epidemiological data charting the course of infection in the United States and, in the case of *West Nile virus*, the diversity of American wildlife infected.


PHOTO OXFORD SCIENTIFIC FILMS

In the UK the Veterinary Laboratories Agency (VLA) has responsibility for wildlife diseases. The VLA regional laboratories are responsible for investigating unusual wildlife mortality and have over the years a successful track record in identifying new diseases such as rabbit haemorrhagic disease, TB in badgers and red squirrel parapoxvirus infection. More details of the activities of the VLA can be found at **www.defra.gov.uk/corporate/vla**.

Additional information about wildlife issues can be found at the Defra website dealing with Wildlife and Countryside which lists the Government's programmes on wildlife and habitat conservation (**www.defra.gov.uk/wildlife-countryside/index**).

● ***Professor Tony Nash, Department of Veterinary Pathology, University of Edinburgh. email tony.nash@ed.ac.uk***

# Seal distemper outbreak 2002

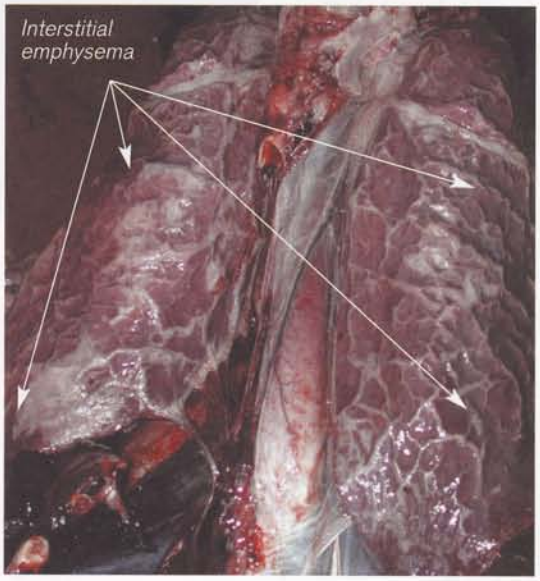
Tom Barrett, Pramoda Sahoo & Paul D. Jepson

Morbilliviruses, the group of viruses for which the human measles virus is the type virus, have recently been found to infect diverse species of marine mammals. Over the past 15 years epizootics of these viruses have caused mass die offs, mainly of seals and dolphins, in widely dispersed locations. The epizootic of phocine distemper virus (PDV) that occurred in northern European seals in April 2002 caused the deaths of more than 22,000 harbour and grey seals before finally burning out in late September.

## Phocine distemper virus

The past 15 years have witnessed the emergence of several newly recognized members of the genus *Morbillivirus* as significant causes of disease outbreaks and mortality in marine mammals belonging to the orders *Pinnipedia* (seals) and *Cetacea* (whales, porpoises and dolphins). The first morbillivirus with potentially severe ecological consequences for marine mammals was identified in harbour (*Phoca vitulina*; Fig. 1) and grey (*Halichoerus grypus*) seals which died in large numbers off the coasts of northern Europe in 1988. At first, based on clinical and antigenic similarities, the seal virus was thought to be canine distemper virus (CDV), the main pathological feature being acute pneumonia (see Fig. 2). The interest which resulted from finding morbillivirus in marine mammals for the first time meant that all available resources were quickly employed to characterize the new virus. These analyses soon showed that the seal virus was most closely related to, but distinct from, CDV and was classified as a new member of the genus *Morbillivirus* and named phocine distemper virus (PDV). The most severely affected species was the harbour seal from the North Sea and north-western Atlantic, with grey seals from the same areas proving more resistant to the virus.

TOP RIGHT:
**Fig. 1.** A harbour seal.
COURTESY DR PAUL THOMPSON, ST ANDREWS UNIVERSITY

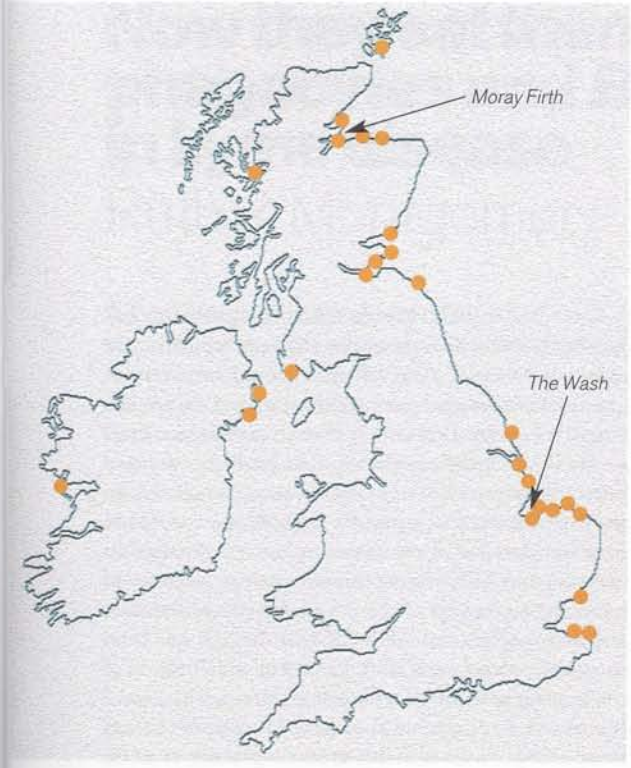



LOWER RIGHT:
**Fig. 2.** Infected seal lung showing severe interstitial pneumonia.
COURTESY T. BARRETT

Since then three other morbilliviruses have been found to infect various species of marine mammals: CDV itself has been found in seals and polar bears, a morbillivirus (PMV) has been isolated from porpoises and another from dolphins (DMV). The latter two viruses are genetically very closely related to each other, but they are not species-specific as the porpoise virus has been found in dolphins. They are best regarded as variants of a cetacean morbillivirus virus (CeMV). Many cetacean species worldwide have been reported to be seropositive for this virus. The pilot whale (*Globicephala* spp.), which has a worldwide distribution, appears to be the reservoir and vector for transmission of CeMV to other species as they move in large groups, known as pods, over great ocean distances. Over 90 % of pilot whales that were involved in mass strandings between 1982 and 1993 were morbillivirus-seropositive.

## Other morbilliviruses affecting seals

Apart from PDV infections, other morbilliviruses can also cause mortality in seal populations. In the winter of 1987–1988, just prior to the European seal epizootic, Lake Baikal seals (*Phoca sibirica*) suffered an unusual and severe mortality. Subsequently CDV, rather than PDV, was shown to have been the aetiological agent. Similarly, a mass die off that occurred in Caspian Sea seals (*Phoca caspica*) in 1997, and again in 2000, was also attributed to CDV infection. Although not proven, the source of the virus is likely to have been terrestrial carnivores, such as wolves, which prey on seal pups. Retrospective serological evidence also links this virus with deaths of thousands of crabeater (*Lobodon carcinophagus*) seals near a base with sledge dogs in the Antarctic in the 1950s. There are no terrestrial carnivores in the Antarctic and so it is likely that sledge dogs used at that time were the original source of CDV, or possibly the animals were infected by contact with carnivores in New Zealand or South America during migrations. The virus appears to have established itself in the crabeater seal population as a subsequent serological survey of Antarctic seals showed them to have a high prevalence of CDV-specific antibodies.




LEFT:
**Fig. 3.** Location of PDV outbreaks around the coasts of the British Isles during 2002.

BELOW:
**Fig. 4.** Seal mortality resulting from the European PDV epizootic in 2002. The numbers of dead seals in the different areas affected, excluding the Baltic Sea, are given for comparison. Kat, Kattegat; Ska, Skagerrak. The inset panel shows the weekly seal death figures in The Wash.

## Diagnosing morbillivirus infections

When wildlife species show clinical disease with signs resembling distemper, a morbillivirus infection may be involved. As CDV and PDV infections in seals are clinically similar, differential diagnosis has to be made between these two viruses and this is most easily done by RT-PCR using universal morbillivirus primer sets and primer sets specific for each morbillivirus species. Some evidence also links the CeMV with seal mortality, although not on the same massive scale seen with PDV or CDV. The rare monk seal population of the eastern Mediterranean was not severely affected during an outbreak of this virus which killed thousands of striped dolphins (*Stenella coeruleoalba*) around the Mediterranean coasts in 1990–1991. However, CeMV was found in carcasses of monk seals that died in large numbers off the coast of Mauritania in 1997. At the time an algal bloom was also considered to be a primary or an exacerbating factor which may have contributed to the very high mortality among adult seals.

## Recent outbreaks of PDV

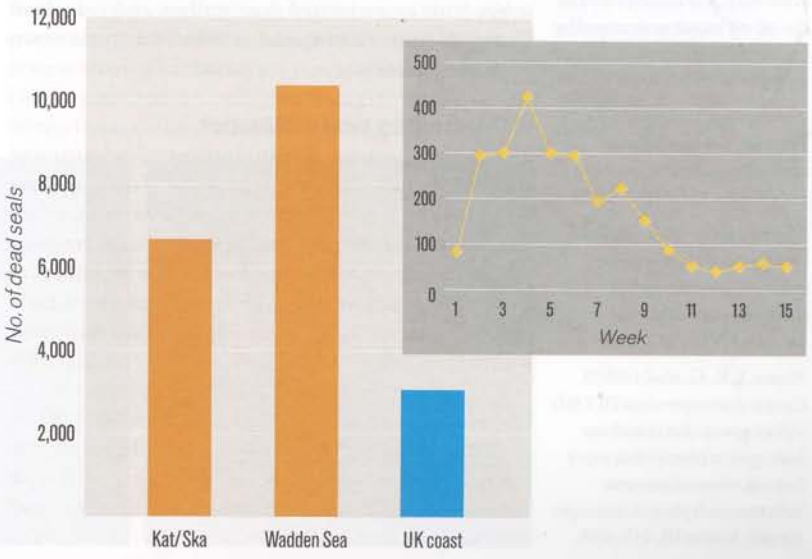
Until last year the most devastating virus-induced mass mortality event in European seals occurred in 1988, when PDV killed more than 18,000 seals around northern European coasts, the vast majority being harbour seals. That epizootic began on the Danish island of Anholt in the Kattegat and then spread to Sweden, the Netherlands, Norway, Germany, the UK and Ireland. Since 1989 there has been no convincing serological evidence for persistence of PDV in European seals. A minor outbreak of disease in seals occurred in 1998 along the Belgian and northern French coasts and morbillivirus antigen and nucleic acid were detected in tissues from sick animals. However, most of the viruses involved were genetically closely related to either CeMV or CDV.

Unfortunately, in 2002 we again witnessed the recurrence of a major PDV outbreak, again mainly affecting harbour seals. The epizootic followed an almost identical course to that of 1988, starting at the end of April, also originating from the island of Anholt (population ~12,000 harbour seals). Through May the virus spread from the initial focus in the Kattegat and spread through The Skagerrak and then into the Dutch Wadden Sea and the North Sea. In the UK, as in 1988, the first PDV-positive harbour seal was identified in The Wash in April. By mid-August an epizootic-type increase in harbour seal mortality began in the immediate area, peaking in mid-September when more than 400 seals were washed up dead each week. By then PDV had reached Scotland and was first identified in a harbour seal found in Dornoch in the Moray Firth. Shortly afterwards a small number of dead seals was found in Northern Ireland and along the west coast of Ireland and PDV infection was confirmed. No PDV-positive seals were found along the Welsh coast (see Fig. 3 for location of UK outbreaks). Although further PDV-positive harbour and grey seals were identified in east and west Scotland (including Orkney) in late 2002, unlike 1988, no noticeable increase in Scottish seal mortality was apparent.

The total death toll in European waters eventually reached over 22,000 based on dead seal counts, approaching 60 % of harbour seals in the worst affected areas, although these figures may be revised following analysis of aerial survey data. Approximately 10 % of these were found in The Wash, the vast majority being harbour seals (see Fig. 4).

## Transmission of PDV

The small size and scattered nature of European seal populations makes it unlikely that they could maintain this virus in circulation as morbilliviruses need a constant supply of new susceptible hosts since, following recovery, infection induces life-long immunity to the



virus. The curious course of the epizootic on both occasions, starting from the Danish coast, moving first to the Dutch and then on to English coasts and finally ending in Scotland, may be explained by the migratory and breeding habits of the seals. There is growing consensus among some seal biologists that grey seals may act as vectors for the transmission of PDV between harbour seal colonies in European waters, especially where large geographical jumps are made. Harbour seals usually return to the same haulout, whereas grey seals do not and move much greater distances between haulout sites. Since it is likely that most virus transmission occurs on land, their mixing at haulout sites may be the key factor. Another may be the relative resistance of this species to the virus, enabling it to transmit without necessarily showing severe clinical disease.

### The original infection source

The most likely source of the original infection for the European seals was contact with seal species from the Arctic. This has been proposed on the grounds that morbillivirus, and more specifically PDV antibodies, were found in archival sera obtained from Arctic seals long before the first known European outbreak in 1988. Arctic harp seals (*Phoca groenlandica*) were noticed to have migrated much further south to reach northern European waters in the year prior to the outbreak, probably as a result of climatic changes and overfishing around Greenland. One possible scenario is that harp seals, at least in 1988, infected grey seals, which then introduced the virus into harbour seals at Anholt. Grey seals are known to haulout in small numbers in Anholt alongside the large harbour seal population there. This may explain why both 1988 and 2002 epizootics started on Anholt. The full host range of PDV has not been determined, but it can infect many species of seal. The harp seal population is extremely large, with an estimated four million individuals in Canadian waters alone, and is sufficient to maintain PDV in circulation.

### Controlling seal distemper

The various animal health institutes, government agencies, private rescue charities and welfare organizations dealing with marine wildlife diseases were well prepared to deal with the 2002 British epizootic once the seal deaths on Anholt were confirmed as being due to PDV. Organization and co-ordination of the various official bodies concerned and the hundreds of volunteers who helped locate the dead seals was easier than in 1988 as the systems set up to deal with that event were re-activated and a single hotline number for the UK was set up for reporting sightings of dead seals. However, the main difference was the wide availability of electronic communication, which made co-ordination on a national and European scale much easier.

Control of wildlife diseases is extremely difficult and vaccination of wild animal populations, which in any case is logistically very difficult, would raise ethical questions concerning uncontrolled spread of the vaccine virus. Protection of vulnerable animals in seal sanctuaries would be acceptable as the animals could be confined until the excretion of any vaccine virus had ceased. In any event, there is no currently licensed vaccine for use in seals. Because of the close antigenic relationship between morbilliviruses, strong cross-protection to infection is given by vaccines for other morbilliviruses and an experimental CDV-ISCOM vaccine has been shown to protect seals from PDV, but the duration of the immunity induced remains uncertain. This, and a live attenuated vaccine that is used to protect dogs from CDV, could also be used to protect seals in sanctuaries during virus epizootics. However, there are no commercial sources of the CDV-ISCOM vaccine, and the commercially available CDV vaccines have not been tested for safety in seals and have been shown to cause death in some wild species. During the 2002 epizootic a small trial of a commercially available CDV vaccine was carried out and the results are still awaited. Following the 1988 epizootic the affected seal populations recovered their normal (pre-1988) population levels within a few years and, it is hoped, the same will happen this time round. A full report on the outbreak is scheduled to be published by Defra in 2004.


● *Tom Barrett and Pramoda Sahoo, Division of Molecular Biology, Institute for Animal Health, Pirbright Laboratory, Ash Road, Pirbright GU24 0NF, UK.*

● *Paul D. Jepson, Institute of Zoology, Regent's Park, London NW1 4RY, UK.*



### Acknowledgements

*The authors thank Dr Ailsa Hall of the Sea Mammal Research Unit (SMRU), Gatty Marine Lab, St Andrews University, for advice and comments on the manuscript.*


## Further reading


**Barrett, T. *et al.* (1993).** Dolphin and porpoise morbilliviruses are genetically distinct from phocine distemper virus. *Virology* 193, 1010–1012.

**Bengtson, J.L. *et al.* (1991).** Antibodies to canine distemper virus in Antarctic seals. *Mar Mamm Sci* 71, 85–87.

**Dietz, R. *et al.* (1989).** Clue to seal epizootic? *Nature* 338, 627.

**Duignan, P.J. *et al.* (1995).** Morbillivirus infection in two species of pilot whales (*Globicephala* sp.) from the western Atlantic. *Mar Mamm Sci* 11, 150–162.

**Duignan, P.J. *et al.* (1997).** Epizootology of morbillivirus infection in harp, hooded and ringed seals from the Canadian Arctic and Western Atlantic. *J Wildl Dis* 33, 7–19.

**El Mjiyad, N. *et al.* (2003).** Cases of morbillivirus infections among seals (*Phoca vitulina*) and fin whales (*Balaenoptera physalus*) stranded on the Belgian and Northern French coast from 1997 until 2002. In *Abstracts of the European Cetacean Society Conference, Las Palmas de Gran Canaria, 9–13 March, 2003*.

**Harwood, J. (1998).** What killed the monk seals? *Nature* 393, 17–18.

**Kennedy, S. *et al.* (2000).** Mass die-off of Caspian seals caused by canine distemper virus. *Emerg Infect Dis* 6, 637–639.

**Thompson, P.M. *et al.* (2002).** Prevalence of morbillivirus antibodies in Scottish harbour seals. *Vet Rec* 151, 609–610.

**Van Bressem, M.-F. *et al.* (2001).** An insight into the epidemiology of dolphin morbillivirus worldwide. *Vet Microbiol* 81, 287–304.

**Visser, I. K. G. *et al.* (1992).** Canine distemper virus ISCOMs induce protection in harbour seals against phocid distemper but still allow subsequent infection with phocid distemper virus-1. *Vaccine* 10, 435–438.

# Now they eat them, now they don't: phagocytes and *Borrelia burgdorferi* in Lyme disease

Ruth R. Montgomery

Lyme Disease is an important spirochaete infection which is transmitted by ticks. Ruth Montgomery describes the complex, and still mysterious, interaction between the host's phagocytes and these bacteria.

## The course of natural infection

Lyme disease, the most common vector-borne disease in North America, is caused by infection with the tick-transmitted spirochaete *Borrelia burgdorferi* (*Bb*) and is characterized initially by the skin lesion, erythema migrans. Subsequent disease reflects the *in vivo* migration of the spirochaete in its host and includes arthritis, neurological symptoms and carditis. Even though most patients are readily cured by antibiotic therapy, rare patients have persistent or recurring illness.

Skin is the initial site of entry of spirochaetes into the host, and in established disease, the skin can provide a haven to *Bb* for extended periods without eliciting an immune response. *Bb* can be recultured from non-lesional human and murine skin and, although dormant in the original host, are still infectious and pathogenic when transferred to a naïve animal. In both human and murine Lyme borreliosis, intact spirochaetes persist in infected tissues and are not completely eliminated by phagocytic innate immune cells.

A vaccine for Lyme disease was developed in mice, targeting the prominently *in vitro*-expressed outer-surface protein A (OspA). The vaccine was effective by blocking transmission from the tick vector to the host and the protective immunity waned rapidly. Notably, OspA is down-regulated in the vertebrate host. Patients required three vaccinations and annual boosters; despite several years of successful use, it was withdrawn from the market by the manufacturer.

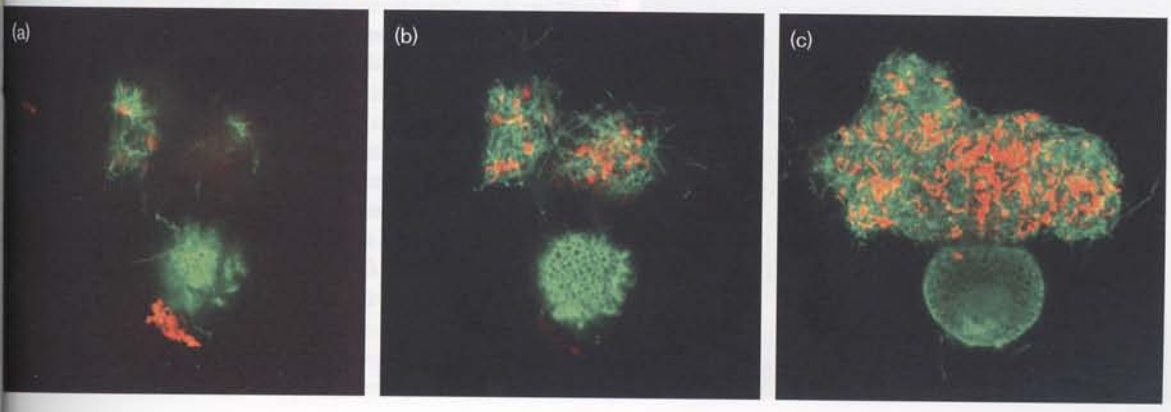
## Innate immune cells and spirochaetes *in vitro*

Mature macrophages *in vitro* ingest and kill spirochaetes avidly and in large numbers. Spirochaetes attach at their ends, independently of the Fc receptor, and are delivered to lysosomes for degradation with a $T_{1/2}$ of about 20 minutes. Nearly all of the ingested spirochaetes are killed, but occasional cell-associated spirochaetes persist. Spirochaete stimulation of macrophages initiates a cascade of pro-inflammatory signals leading to the production of potent inflammatory mediators, including reactive oxygen and nitrogen intermediates, arachidonic acid metabolites, proteases, and in addition, cytokines and chemokines that elicit adaptive immune responses. *Bb* lipoproteins are highly inflammatory and incite this response via pattern recognition receptors CD14 and Toll-like receptors (TLR) 1 and 2; the latter are from a family of highly conserved transmembrane receptors that have an essential role in the innate immune defence against pathogens. Macrophages are the most efficient cellular defence against spirochaetes during the initial innate immune response; monocytes are less effective.

## Importance of antibody for clearance by polymorphonuclear leukocytes (PMN)

PMN *in vitro* bind *Bb* by conventional phagocytosis as well as via tube and coiling phagocytosis. Antibody is critical for the efficient clearance of *Bb* by PMN: while unopsonized spirochaetes are poorly eliminated, PMN clear opsonized spirochaetes rapidly. There is no intracellular co-localization of PMN granule products

TOP LEFT:
*Ixodes dammini* tick. Ticks were microinjected in the anal aperture with fluorescent dyes to label surface structures and midgut cells. The ticks were imaged live 24 h after injection by laser scanning confocal fluorescent microscopy.
COURTESY R. MONTGOMERY

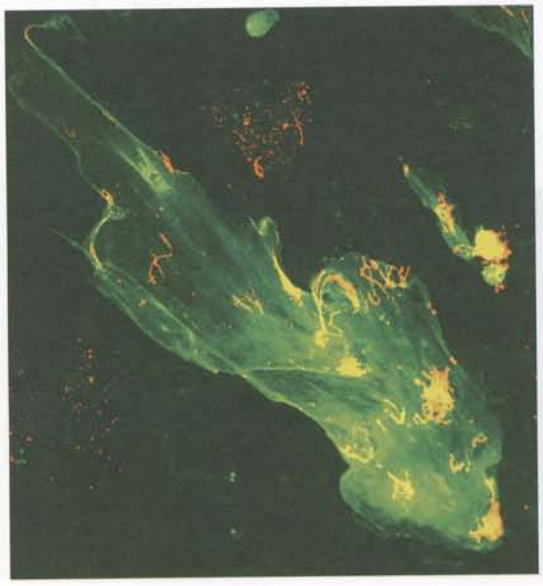



LOWER LEFT:
Killing of spirochaetes by macrophages. Human macrophages were incubated with unopsonized *Bb*. After 60 min incubation at 37 °C, samples were stained with live/dead dye, red indicating killed organisms. Note the extensive killing by macrophages evident at the top surface of the cells (a), the middle level (b) and adherent to the coverslip (c).
COURTESY R. MONTGOMERY

with spirochaetes; significant killing of spirochaetes by PMN occurs extracellularly. PMN have abundant mechanisms for killing spirochaetes and eliminate *Bb* using both oxidative and non-oxidative killing mechanisms. PMN lysates kill *Bb* as effectively as do intact PMN given opsonized spirochaetes, highlighting the importance of granule proteins. In addition, the abundant cytosolic protein calprotectin is also bacteristatic, probably through chelation of $Zn^{2+}$, an essential cation for *Bb*. In contrast to the granule components, which may require the environment of the phagolysosome, calprotectin can function as an extracellular anti-microbial factor. Indeed, elevated levels of calprotectin (also known as S100, MRP 8/14) are noted in arthritic joints.

## Arthropod saliva modulates infection

In natural infection, spirochaetes are delivered via an Ixodid tick vector in the presence of saliva, which forms a plug at the inoculation site. The local concentrations of saliva in the skin may be quite high. The saliva of hematophagous arthropods is a pharmacological arsenal to aid in blood feeding by the vector (such vectors have been called 'invertebrate syringes'). Saliva contains potent activities that both reduce clotting and increase vasodilation, as well as potent anti-inflammatory activities that inhibit cellular immune responses, including antigen presentation and IFNγ-stimulated $H_2O_2$ release by human macrophages. The efficacy of the arthropod strategy can be seen as sandfly saliva allows a smaller inoculum of infecting *Leishmania* organisms to establish infection, encompassing a larger lesion in the mouse. Similarly, in early Lyme disease, there is a boost to infection via tick delivery (over syringe inoculation), altering the initial antibody response, presumably due to the presence of saliva.



BELOW:
PMN myeloperoxidase does not co-localize with spirochaetes. Fresh human PMN incubated with opsonized *Bb* were fixed after 60 minutes and double-labelled with antibodies specific for myeloperoxidase (green) and spirochaetes (red). Images obtained using confocal microscopy demonstrate the spread of granule components over an area far exceeding that of two neighbouring intact cells (top and right), consistent with the extracellular killing of spirochaetes.
COURTESY R. MONTGOMERY

## Tick saliva alters phagocyte function

PMN are the first cells of the innate immune system to arrive at the site of spirochaete deposition in the skin. The saliva of *Ixodes* ticks is known to inhibit in some way phagocytosis, granule release and superoxide production by PMN, as well as aggregation of platelets *in vitro*. *Ixodes* tick saliva also inhibits T cell proliferation and reduces the production of cytokines and nitric oxide and the killing of spirochaetes by macrophages. Inhibitory bioactivities of *Ixodes* tick saliva include an anti-coagulant, prostaglandin E2, kininase, an antioxidant and an anticomplement peptide. These salivary proteins are now being thoroughly characterized by several groups for potential therapeutic advantage.

## The conundrum of persistent infection

The ease of elimination *in vitro* belies the persistence of spirochaetes in the infected host. We have at present no explanation for the persistence of spirochaetes in the host in prolonged disease and the apparent immune invisibility of *Bb* in tissue. Macrophages *in vivo* in infected animals display no global inhibition of function; rather they are appropriately activated *in situ* and remain in a resting state in unaffected tissues. In Lyme carditis, they produce increased levels of mRNA for pro-inflammatory cytokines, reflecting appropriate macrophage activation and display no evidence of pressure toward immune down-regulation.

Tick saliva contributes to the failure of phagocytes to clear organisms from the skin in initial infection. Saliva's inhibition of phagocyte function gives the infecting spirochaetes an initial advantage in evading the sentry phagocytes.

Persistence may depend on the spirochaetes themselves: there is evidence that spirochaetes down-regulate their antigenic surface proteins or coat them with host proteins. Freeze fracture electron microscopy of *Bb* reveals fewer surface-expressed proteins than on other spirochaetes, and an mRNA array analysis of *Bb* lipoprotein genes during the course of murine infection shows a reduction from 116 genes expressed to fewer than 40 genes remaining at 30 days of infection, after induction of adaptive immunity. The paradoxical persistence of spirochaetes in the host, despite appropriate activation (or non-activation) of phagocytes, remains mysterious.

● *Ruth Montgomery is a Senior Research Scientist in the Department of Internal Medicine at Yale University School of Medicine, 333 Cedar Street, New Haven, CT 06520-8031, USA.*
*Tel. +1 203 785 7039; Fax +1 203 785 7053*
*email ruth.montgomery@yale.edu*

## Further reading

Barthold, S.W., de Souza, M.S., Janotka, J.L., Smith, A.L. & Persing, D.H. (1993). Chronic Lyme borreliosis in the laboratory mouse. *Am J Pathol* 143, 959–972.

Hirschfeld, M., Kirschning, C.J., Schwandner, R., Wesche, H., Weis, J.H., Wooten, R.M. & Weis, J.J. (1999). Cutting edge: inflammatory signaling by *Borrelia burgdorferi* lipoproteins is mediated by Toll-like receptor 2. *J Immunol* 163, 2382–2386.

Lusitani, D.L., Malawista, S.E. & Montgomery, R.R. (2003). Calprotectin, an abundant cytosolic protein from human polymorphonuclear leukocytes, inhibits the growth of *Borrelia burgdorferi*. *Infect Immun* 71, 4711–4716.

Montgomery, R.R., Lusitani, D.L., de Boisfleury Chevance, A. & Malawista, S.E. (2002). Human phagocytic cells in the early innate immune response to *Borrelia burgdorferi*. *J Infect Dis* 185, 1773–1779.

Ribeiro, J.M. & Francischetti, I.M. (2003). Role of arthropod saliva in blood feeding: sialome and post-sialome perspectives. *Annu Rev Entomol* 48, 73–88.

# Tick-borne relapsing fever in Tanzania

Sally J. Cutler & Alison Talbert

The 2nd International Conference on tick-borne relapsing fever took place recently in Africa. Sally Cutler and Alison Talbert describe some of the latest findings on this life-threatening disease.

TOP LEFT:
The semi-arid savannah of Central Tanzania.

BELOW:
Chicken roosting in the kitchen area inside a traditional house.

PHOTOS S.J. CUTLER

Scientists and healthcare specialists working towards the control of tick-borne relapsing fever (TBRF) met recently in Mvumi, near Dodoma, Central Tanzania. This disease, caused by the spirochaete *Borrelia duttonii* and transmitted by *Ornithodoros moubata* tick vectors, remains a significant cause of mortality and morbidity throughout much of Tanzania. The traditional 'tembe' houses are frequently infested with the tick vector and the disease particularly affects young children and pregnant women, resulting in foetal loss and neonatal deaths (the perinatal mortality rate is 436 per 1,000).

Delegates were updated on the number of cases in the Mvumi and Mwanza districts where the disease is endemic. The annual incidence in children in Mvumi under a year is 384 per 1,000, and 163 per 1,000 in children under 5 years. However, little data are collated on the local levels on disease for the remainder of Tanzania.

Recent work on the tick vectors suggests that *O. porcinus domesticus* may be the principal vector in Tanzania. Phylogenetic analysis of ticks and patient samples has indicated the presence of a novel borrelial species showing a closer resemblance to New World species than those present in Africa. The role of this spirochaete in clinical disease has yet to be determined.

Other studies on the possible extended reservoir for this disease beyond man are currently underway. Certainly, most other borrelial species exist with their natural reservoir in either rodents or birds, the exceptions being *B. duttonii* and *B. recurrentis*. Both rats and chickens frequently share traditional tembe accommodation and consequently will have close proximity to the tick vectors. For man to serve as the sole reservoir, it would be expected that these spirochaetes should persist at a site where they can be readily acquired by their arthropod vectors. Evidence of spirochaetes present in the blood of asymptomatic villagers was presented; however, only 11·1 % (6/54) of febrile and 4·2 % (13/307) of afebrile children under 5 years of age were positive. Whether this is sufficient to maintain the infectious cycle remains an open question. Certainly, these spirochaetes are able to undergo antigenic variation, presenting different variable membrane proteins to the hosts' immune system.

Molecular mechanisms of antigenic variation are also being studied, and different variable membrane protein genes were reported. Furthermore, these spirochaetes have recently been shown to bind soluble immune mediators such as factor H and factor H-like protein.

A clear need for accurate surveillance to determine the size and extent of TBRF was demonstrated. Although funds for this are currently unavailable, data collection from selected regions could be collated. This, together with further work to understand both spirochaete–vector and host–spirochaete relationships, will allow investigations on possible intervention strategies. Full details of the meeting can be viewed at **www.mvumi.org**.

● *Dr Sally J. Cutler is research team leader in the Bacterial Zoonoses section of the Department of Statutory and Exotic Bacterial Diseases, Veterinary Laboratories Agency, Woodham Lane, New Haw, Addlestone, Surrey KT15 3NB, UK. Tel. 01932 357807 Fax 01932 357423 email s.cutler@vla.defra.gsi.gov.uk*

● *Dr Alison Talbert is a paediatrician in Mvumi Hospital, Mvumi, PO Box 32, Dodoma, Tanzania.*

# Emerging bartonellosis

Christoph Dehio & Anna Sander

Bartonellae are arthropod-borne pathogens of growing medical importance. Until the early 1990s, only two species of this bacterial genus, *B. bacilliformis* and *B. quintana*, were recognized as causing disease in humans. In addition to re-emergence of the human-specific *B. quintana*, a number of zoonotic *Bartonella* species have now been recognized as causative agents for a broadening spectrum of diseases that can be transmitted to humans from their animal hosts. Most prominently, *B. henselae* is an important zoonotic pathogen that is frequently passed from its feline reservoir to humans.

Bacteria of the genus *Bartonella* are Gram-negative, pleomorphic, fastidious bacilli that belong to the α-2 subclass of *Proteobacteria*. All *Bartonella* species appear to have a specific mammalian species as a host, in which they cause a long-lasting infection within the red blood cells (intraerythrocytic bacteraemia). Blood-sucking arthropod vectors transmit the bacteria from this reservoir to new hosts. Incidental infection of non-reservoir hosts (e.g. humans by the zoonotic species) may cause disease, but does not result in intraerythrocytic infection.

**Table 1. *Bartonella* species, their natural reservoir, mode of transmission, and human diseases**

| *Bartonella* species | Reservoir | Vector | Human diseases |
|---|---|---|---|
| **Human-specific species:** | | | |
| *B. bacilliformis* | Human | Sandfly | Carrión's disease: Oroya fever and verruga peruana |
| *B. quintana* | Human | Body louse | Trench fever, endocarditis, bacillary angiomatosis peliosis |
| **Zoonotic species:** | | | |
| *B. clarridgeiae* | Cat | Cat flea | CSD |
| *B. elizabethae* | Rat | | Endocarditis, neuroretinitis |
| *B. grahamii* | Mouse, vole | | Neuroretinitis |
| *B. henselae* | Cat | Cat flea | CSD, endocarditis, bacillary angiomatosis peliosis, neuroretinitis |
| *B. vinsonii* subsp. *arupensis* | Mouse | Ticks | Fever, bacteraemia |
| '*B. washoensis*' | Ground squirrels | | Myocarditis |
| **Animal-specific species:** | | | |
| *B. alsatica* | Rabbit | | - |
| *B. birtlesii* | Mouse | | - |
| *B. bovis* (='*B. weissii*') | Cattle / cat | | - |
| *B. capreoli* | Roe deer | | - |
| *B. chomelii* | Cattle | | - |
| *B. doshiae* | Vole | | - |
| *B. koehlerae* | Cat | | - |
| *B. peromysci* | Deer, mouse | | - |
| *B. schoenbuchensis* | Roe deer | Deer ked | - |
| *B. talpae* | Mole | | - |
| *B. taylorii* | Mouse, vole | | - |
| *B. tribocorum* | Rat | | - |
| *B. vinsonii* subsp. *berkhoffii* | Dog | Ticks | - |
| *B. vinsonii* subsp. *vinsonii* | Vole | Vole ear mite | - |

## Natural history and epidemiology

Humans are the only known reservoir for two *Bartonella* species, *B. bacilliformis* and *B. quintana*.

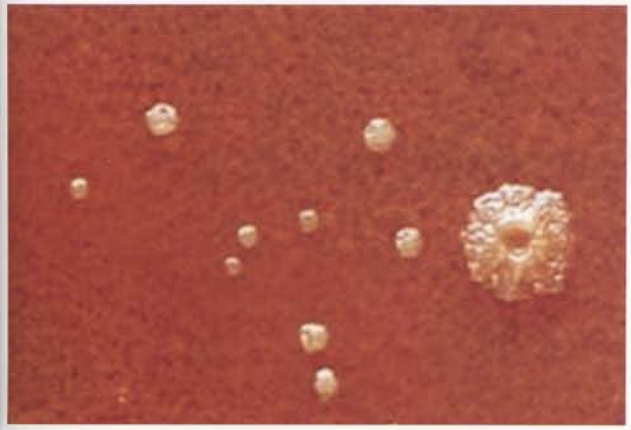
*B. quintana* was a leading cause of infectious morbidity among soldiers during World War I, and recurred on the East European front in World War II. The disease, Trench fever, is rarely fatal and is characterized by an intraerythrocytic bacteraemia with recurrent, cycling fever. It is transmitted among humans by the human body louse *Pediculus humanus*. Although almost forgotten by medical science since the end of World War II, *B. quintana* re-emerged as an agent of disease among homeless people and those with a lowered immune response towards the end of the 20th century, exhibiting novel symptoms.

*B. bacilliformis* was described by the Peruvian physician Alberto Barton in 1909. He observed the presence of intraerythrocytic bacilli in blood smears of patients suffering from Carrión's disease. This biphasic disease is transmitted by the sandfly *Lutzomyia verrucarum* and occurs endemically in the valleys of the South American Andes of Peru, Columbia and Ecuador. The infection can manifest as the life-threatening condition called Oroya fever, where the patient suffers from anaemia and a high temperature. If the patient survives, this may be followed by a condition called verruga peruana, which is a less dangerous chronic illness characterized by benign wart-like vascular lesions on the skin.

In recent years, the appreciation of the widespread occurrence of *Bartonella* species in the bloodstream of mammals has resulted in the description of several new *Bartonella* species. In several instances this has allowed researchers to connect the presence of these bacteria with human diseases of previously unknown origin. Table 1 lists the 20 species of the genus *Bartonella*. So far, eight of them are known to be zoonotic species that can act as pathogens in humans, although the preferred host is another mammal.

## *B. henselae*: a major zoonotic pathogen

*B. henselae* (Fig. 1) is the most important zoonotic species to cause human disease. This pathogen is distributed worldwide and causes intraerythrocytic bacteraemia in the feline reservoir host. Between 40 and 70 % of cats living in warm, humid geographic regions have either an active infection within their bloodstream, or have antibodies, indicating that they have been infected in the past. Feral cats are more likely to be infected than pet cats



from the same region. Although some of these animals may be bacteraemic over a period of more than 1 year, cats usually appear healthy and asymptomatic. Transmission to humans occurs by the cat flea *Ctenocephalides felis*, or directly by cat scratch or bite.

*B. henselae* can cause a variety of clinical manifestations. Cat-scratch disease (CSD) was first described in 1950 by the French physician Debré in patients suffering from inflamed lymph nodes following cat scratches. CSD occurs worldwide in all age groups. In the US, *B. henselae* causes 22,000 diagnosed cases of CSD per year, requiring hospitalization of about 10 % of the patients. Most cases occur in October to March, when a closer and more prolonged contact with cats occurs indoors. Regional swelling of the lymph nodes (Fig. 2) is the most common clinical manifestation of CSD, but atypical symptoms with involvement of the eyes, liver, spleen, central nervous system, skin, bones or other organs may occur, particularly in immunocompromised individuals.

In recent years a large number of *Bartonella* infections of the heart (endocarditis) have been reported in the literature, indicating that it may cause 3–4 % of all cases. Homeless and chronically alcoholic individuals are particularly susceptible to the disease. In the immunocompromised, especially HIV-infected, patients bacillary angiomatosis peliosis is the most common clinical manifestation of an infection with *B. henselae*. The symptoms are red, blood-filled lesions on the patient's skin due to proliferation of surface blood vessels, and resemble pyogenic granulomas, haemangiomas or Kaposi's sarcoma.

Laboratory diagnosis of *B. henselae* and other *Bartonella* infections uses the most recent developments in serological testing [indirect immunofluorescent antibody assay, enzyme-linked immunosorbent assay (ELISA), Western blot analysis], histopathological investigation of lymph node specimens, skin biopsies, or other clinical material, and PCR amplification of *Bartonella* DNA from human specimens.

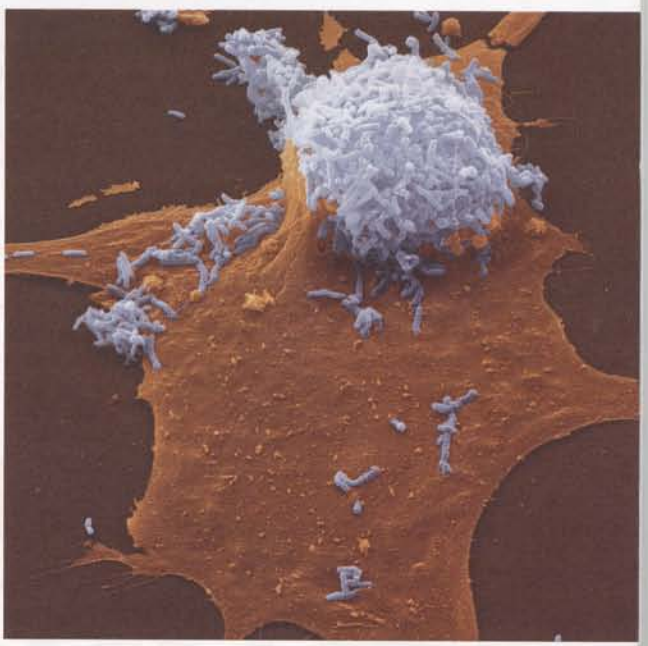
## Pathogenesis

During infection of their mammalian hosts, bartonellae migrate towards two cell types, the red blood cells (erythrocytes) and the cells lining the blood vessels (vascular endothelium). The invasion and long-lasting intracellular colonization of erythrocytes occurs exclusively in the normal reservoir host (i.e. humans for *B. bacilliformis* and *B. quintana*; other mammals for other *Bartonella* species). In contrast, vascular endothelial cells are targets in both reservoir and incidental hosts, and their infection by bartonellae may result in the formation of proliferative tumours. This ability to stimulate uncontrolled growth of vascular tissue is unique to bartonellae. Cultured human vascular endothelial cells provide an *in vitro* system to study the interaction of *B. henselae* with the human vascular endothelium. The bacteria are internalized by endothelial cells and activate a proinflammatory phenotype. Moreover, the infection inhibits programmed cell death (apoptosis) and stimulates multiplication of the endothelial cells (Fig. 3), thereby facilitating the formation of vaso-proliferative lesions. Recent evidence has demonstrated that most of these dramatic changes in endothelial cell physiology are mediated by bacterial proteins that are delivered into infected endothelial cells by the bacterial type IV secretion system VirB. Since animal models of *Bartonella* infection have shown that the VirB system is essential for establishing an infection, and many of its components are on the bacterial cell surface, it is a promising target for vaccine development. One component of VirB, the 17 kDa antigen, has already been shown to be immunogenic, and indicates a way forward.

UPPER LEFT:
**Fig. 1.** Colonies of *B. henselae* (large) and *B. clarridgeiae* (small) isolated from cat blood.
COURTESY A. SANDER

LOWER LEFT:
**Fig. 2.** Regional swelling of the lymph nodes is a common clinical manifestation of CSD.
COURTESY A. SANDER

BELOW:
**Fig. 3.** Coloured scanning electron micrograph of *B. henselae* bacteria (blue) infecting a human cell (brown).
REPRODUCED FROM DEHIO *ET AL.*, *J CELL SCI* 110, 2141–2154 WITH THE KIND PERMISSION OF THE COMPANY OF BIOLOGISTS LTD.




● *Dr Christoph Dehio is Assistant Professor for Molecular Microbiology at the Biozentrum of the University of Basel, Switzerland.*
*Tel. +41 61 267 2140; Fax +41 61 267 2118;*
*email christoph.dehio@unibas.ch*
● *Dr Anna Sander is Assistant Professor in Medical Microbiology, Department of Medical Microbiology and Hygiene, University of Freiburg, Germany.*
*Tel. +49 761 203 6529; Fax +49 761 203 6529;*
*email sander@ukl.uni-freiburg.de*

# The significance of genetic control in TSEs

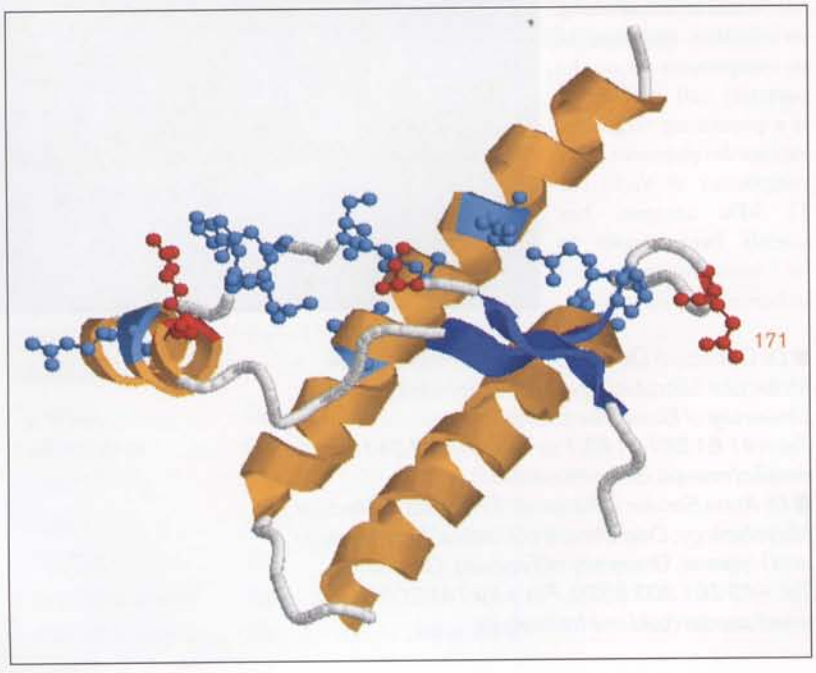
Wilfred Goldmann

Are wildlife species less prone to TSE diseases than domesticated animals? Wilfred Goldmann assesses the latest findings on PrP gene dependent susceptibility.

Transmissible spongiform encephalopathies (TSEs), also called prion diseases, are unconventional diseases. The agent is not a standard micro-organism, incubation periods can be several years and the most characteristic pathology is the accumulation of the prion protein, PrP. TSEs are endemic in domestic sheep (scrapie) and have been naturally transmitted to cattle (bovine spongiform encephalopathy, BSE) and experimentally transmitted to laboratory rodents. In humans they occur sporadically as Creutzfeldt–Jakob disease (CJD).

When BSE began to infect first hundreds and then thousands of cattle in the UK, the question arose why this new form of TSE had affected cattle to this extent, but not other farmed species such as pigs. Why did almost a hundred domestic cats develop a feline TSE, but no dog was ever diagnosed with a TSE? Is there a method to predict the TSE susceptibility of any mammal, whether domesticated or wildlife? Research into scrapie and CJD is helping us to answer these important questions. Crucially, it is now known that the susceptibility to disease and the long incubation periods are under strong genetic control of the PrP gene.

PrP gene dependent susceptibility operates at two levels. Primary susceptibility demands that the organism produces the PrP protein. It was shown that transgenic mice without functional PrP gene cannot develop experimental disease and it is reasonable to assume that this resistance would apply to scrapie and other natural TSEs. Are there any mammalian species that do not have a PrP gene? The answer is no for all 120 mammals of many genera that have been analysed so far. Whether functional PrP protein is always present in the relevant tissues is currently not known. Secondary susceptibility stipulates that PrP protein has to have a susceptible amino acid sequence. The PrP protein gene contains about 255 codons. It is well conserved with common motifs found in PrP of very divergent genera. Amino acid variations are, however, common which makes it difficult to predict changes relevant to TSEs. Despite this, definitions of susceptible sequence motifs have emerged.

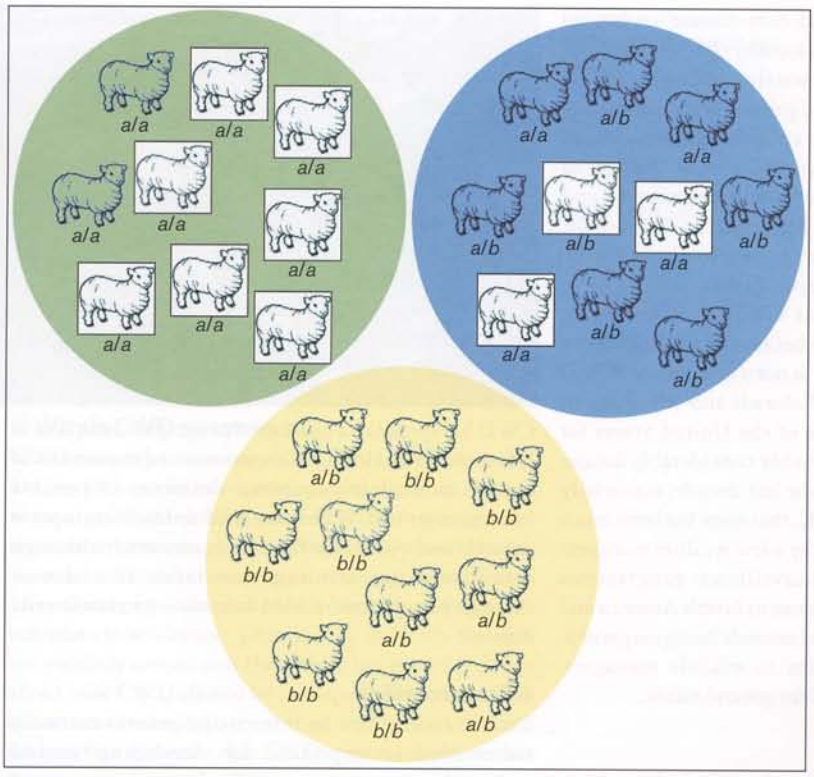



BELOW:
The PrP protein structure is partly described which helps to link the position of amino acid changes associated with disease to protein structure. Amino acid positions genetically variable in sheep are highlighted in colour.
COURTESY W GOLDMANN

Fortunately, sheep represent an excellent model for studying the link between genetic control of disease and the many variants of the PrP gene (PrP alleles). More than 20 ruminant PrP alleles have been described; several show major, some very subtle, effects on susceptibility. They are almost all associated with single amino acid substitutions. For example, the amino acid glutamine is encoded at codon 171 of sheep PrP and can be linked to susceptibility. If however arginine is encoded at position 171, as found in sheep, resistance is conferred. Most importantly, this effect is dominant, so that, i.e. heterozygous glutamine/arginine genotypes are less susceptible than glutamine/glutamine genotypes. Higher resistance of heterozygous PrP genotypes has also been shown for codon 129 in human TSEs. These sequence positions and genotypes appear to be of fundamental importance for assessing or predicting susceptibility.

Having demonstrated in sheep that the amino acid at codon 171 is crucial for the development of scrapie, it is of great interest to analyse which other species are polymorphic at or near to the 171 position. Arginine at 171 has not yet been found in wildlife, but significantly humans encode a different amino acid in this position (glutamic acid). Whether this is a result of genetic selection, which makes us less susceptible but not quite resistant to TSEs, remains to be investigated. The PrP genetics in wildlife are mostly unknown as often only one or very few samples are analysed. From the few available data it appears however that genetic variation of PrP in wildlife species is common. Keeping in mind that heterozygosity in the PrP locus is advantageous with respect to TSEs, one may speculate that a lower susceptibility is a consequence.

Many years ago it was proposed that the TSE agent is ubiquitous, consequently every mammal may be exposed to it, but only a few are susceptible to disease. If this were the case we might expect to see the PrP gene sequence evolved into a semi-resistant form, which remains only susceptible to new strains of the TSE agent. Most species may be genetically resistant to the extent that epidemic outbreaks are less likely, although individual cases may still occur. If this resistance is overcome by the change of environmental factors (i.e. feeding practice), epidemics such as BSE in cattle result. Except for chronic wasting disease in American deer, TSE-like diseases have so far not been observed in




ABOVE:
Genetic diversity in the PrP gene protects populations from TSEs. In the green population all individuals are of the same PrP genotype (*a*/*a*) and many will succumb to disease (white sheep). In the blue population heterozygous genotypes (*a*/*b*) decreases the overall disease rate. The yellow population is free of disease as the resistant allele (*b*) dominates the genepool.
COURTESY W GOLDMANN

wildlife. Based on our experience with domestic animals the main reason may well be strong genetic resistance due to the prevalence of highly resistant PrP sequences. This could have come about due to the fact that an animal with a neurological disease becomes easier prey. It would represent a stronger selection pressure on the genetic background than exists in domesticated species.

But more subtle genetic modulations may be equally effective. PrP variants may have evolved in wildlife that led to an extension of incubation periods beyond the normal lifespan. This effect would certainly be more evident in wildlife than in domestic or laboratory animals, which have a longer life expectancy. What is the likelihood that wildlife represents a reservoir of new TSE agent strains? We cannot answer this with our current scientific knowledge, but research into the significance of non-clinical carriers of infection in sheep is underway and will provide important answers in the future.

● *Dr Wilfred Goldmann is a Senior Research Scientist at the Neuropathogenesis Unit, Institute for Animal Health, Edinburgh, EH9 3JF, UK.*
*Tel. 0131 6675204*
*email wilfred.goldmann@bbsrc.ac.uk*

## Science Policy

SGM has been responding to consultation documents and helping to shape science policy in the UK. Reports have now appeared on the following topics.

In Spring 2003, the Department for Environment, Food and Rural Affairs (DEFRA), in collaboration with the Scottish Executive and the Welsh Assembly Government, consulted on preparing an *Animal Health And Welfare Strategy for Great Britain*. The resultant strategy is intended to reduce the economic, social and environmental impact of animal diseases and improve the welfare of all animals kept by man (www.defra.gov.uk/animalh/ahws/default.htm).

SGM also contributed to the Royal Society consultation on *Measuring Biodiversity* for conservation (www.royalsoc.ac.uk/templates/statements/StatementDetails.cfm?statementid=232). The loss of species has accelerated over the last 200 years and effective methods of measuring biodiversity, based on sound science, are urgently needed to monitor changes in the state of life on earth. The main recommendation of the report is the routine application of a framework, developed for selecting and undertaking appropriate ways of measuring biodiversity. The report also calls for the crucial merging of biodiversity information to make scattered data, held globally in museums, libraries and informal records, more readily available and useful.

The House of Lords Select Committee on Science and Technology held a consultation on *Fighting Infection* (www.publications.parliament.uk/pa/ld200203/ldselect/ldsctech/138/13801.htm). Infectious disease is a significant cause of human illness and death and the emergence of new infections, such as SARS, causes widespread anxiety and affects international travel and trade. Services expected to protect the population from both common and more unusual infections are under-resourced and over-stretched. Recommendations from the report include improving collaborative relationships across the various health services (including international services), ensuring there are sufficient, well-trained health professionals, funding for research to provide an evidence base for improving the diagnosis, treatment, prevention and control of infections, secure supplies of vaccines in case of epidemics and the provision of clear advice and information to the public.

SGM has also submitted responses to two further consultation documents; the final reports are pending.

The Royal Society called for evidence on the detection and decontamination of chemical and biological weapons. The working group set up for this study will use the evidence submitted to identify cutting edge science and technology that might practically be applied to detection and decontamination in the future.

The Food Standards Agency (FSA) consulted on the strategy for the control of *Campylobacter* in chickens. The FSA has a target to reduce the incidence of human food-borne disease by 20% by April 2006. *Campylobacter* is the greatest challenge as far as this target is concerned and there is strong evidence that the most significant route by which people are exposed to *Campylobacter* is through chicken.

● ***Faye Jones, Public Affairs Administrator***

# Prions in the wild: CWD in deer and elk

Elizabeth Williams & Michael Miller

Chronic wasting disease (CWD) is a TSE that affects members of the deer family in North America. It is the only TSE known to occur in free-ranging animals. Beth Williams and Michael Miller describe the epidemiology of CWD, how it spreads and why it is so unusual.

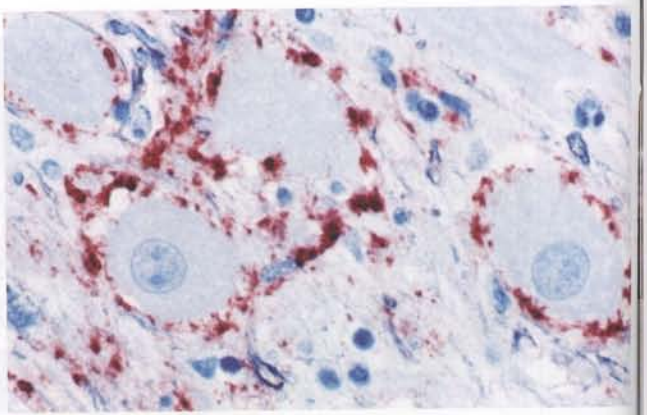
No – it is not 'mad deer disease' or bovine spongiform encephalopathy (BSE) in deer and elk. But chronic wasting disease (CWD) belongs to the same group of prion diseases known as the transmissible spongiform encephalopathies (TSEs). CWD is a prion disease that seems to be specific to cervids (members of the deer family) in western and midwestern North America. Although the origin of the disease is not known it appears to be related to a disease called scrapie of domestic sheep and goats.

While it may appear that CWD is an emerging infectious disease in wildlife because of all the concern about the TSEs worldwide, it is not a new disease. CWD has been present in parts of Colorado and Wyoming in the Rocky Mountain region of the United States for more than 30 years and probably considerably longer than that. It is only within the last decade, essentially since BSE was found in the UK, that there has been much interest in CWD other than by a few wildlife managers and scientists. But with surveillance programmes detecting CWD in new locations in North America and increasing numbers of affected animals being diagnosed, CWD has become a concern to wildlife managers, hunters, conservationists and the general public.

## Epidemiology

Like all of the TSEs, CWD is a disease of adults. Most clinically affected deer and elk are 3–7 years of age; both males and females become ill, although the males (buck deer and bull elk) are more likely to be affected in free-ranging populations than are the females (doe deer and cow elk). Both native species of deer in North America, mule deer (*Odocoileus hemionus*; they are called mule deer because they have big ears) and white-tailed deer (*Odocoileus virginianus*; called white-tailed deer because they have distinct white tails that they 'flag' when they are running) are susceptible and seem to be the primary hosts. Elk (*Cervus elaphus nelsoni*; also called 'wapiti', a name given to them by native Americans) are also susceptible. Elk are closely related to the red deer of Europe (multiple subspecies of *Cervus elaphus*). CWD is not seasonal and affected animals may appear at any time of the year. However, in the wild, cases are more often observed during the fall and winter due to an increase in visibility and possibly the stressful effects of winter weather and shortage of forage.

Because of the way that cervids are managed in North America there are two related but different on-going epidemics of CWD. First, free-ranging deer and elk in parts of Wyoming, Colorado, and Nebraska maintain CWD in their populations. The disease is probably slowly spreading through natural movements of animals as they migrate and disperse. The other CWD epidemic, which may have a common origin with the disease in free-ranging deer and elk, is occurring in privately owned game-farmed animals (primarily elk).



CWD has spread to a number of geographic locations in North America through human-assisted movement of captive animals in commerce. Instances of possible transmission of CWD from wild animals to captive animals and vice-versa have been observed, although definitively documenting movement of a disease through populations of wild animals is extraordinarily difficult.

## Transmission

Understanding how an infectious disease is naturally transmitted is important for developing control and eradication strategies. Tracking the routes of transmission of two similar animal TSEs, scrapie and CWD, has been a challenge to scientists. Scrapie has been known for centuries and it is only within the last few years, with improved diagnostic techniques, that contact with placenta from a scrapie-infected ewe has been found to be important for transmission within flocks. The contribution of direct (animal to animal) or indirect (environmental contamination) horizontal routes of transmission in scrapie is not clear. In contrast, CWD appears to move between animals differently. Transmission from doe to fawn (maternal transmission) does not drive CWD epidemics, but rather horizontal transmission appears to be critical. This observation suggests that control of CWD epidemics will be difficult both in captive and free-ranging situations.

But exactly how does the CWD agent get from one animal to another? Based on the presence of agent in lymphoid tissues lining the alimentary tract, excretion in saliva and/or faeces seems a likely route to the external environment where a susceptible deer or elk could become infected. In free-ranging herds, it is also possible that decomposing carcasses of CWD-affected deer and elk, containing high titres of CWD agent in brain and spinal cord, could serve as a source of agent for other animals. We are studying these possibilities; but as with most TSE animal research, patience is necessary due to the very long incubation periods characteristic of these diseases.

TOP RIGHT:
A section of brain from a mule deer with CWD. The red-staining material around the large neurons is disease-associated PrP.
COURTESY E. WILLIAMS

TOP LEFT:
Healthy free-ranging mule deer bucks.

LEFT:
A subclinically CWD-affected captive elk.

PHOTOS E. WILLIAMS

### Why is CWD so unusual?

CWD is a unique member of the TSEs. It is the only TSE known to occur in free-ranging animals. That leads to some significant difficulties in trying to manage and eradicate it. Free-ranging deer and elk in western North America are found at relatively low densities spread over large tracts of private and public land which vary from suburban to wilderness. Deer and elk in North America are publicly owned and the public has mixed feelings about how CWD should be managed. Thus there is no consensus among all concerned entities on how to address this disease or that CWD is even a problem that requires active management.

Transmission of CWD appears to be highly efficient in comparison with other animal TSEs, resulting in prevalences of greater than 90% in some densely confined deer and elk populations. Another important difference is that susceptibility of sheep to scrapie is modulated by genetic factors; these do not seem to play a very significant role in epidemics of CWD in deer and elk.

CWD presents many challenges to wildlife managers, scientists, regulatory agencies, and the general public. As with any disease that has potential negative implications for populations of wild animals or for human or domestic animal health, developing approaches to management or eradication is complex. Many agencies and scientists have recently turned their attention to the study of CWD; no doubt important insights into the epidemiology, transmission and control of this disease will be forthcoming.

Stay tuned.


● *Dr Elizabeth Williams is a diagnostic veterinary pathologist and wildlife veterinarian at the University of Wyoming, Laramie, Wyoming, USA.*
*email storm@uwyo.edu*
● *Dr Michael Miller is a wildlife veterinarian with the Colorado Division of Wildlife, Fort Collins, Colorado, USA.*
*email mike.miller@state.co.us*


## Further reading


**Miller, M.W. & Williams, E.S. (2003).** Prion disease: Horizontal prion transmission in mule deer. *Nature* 425, 35–36.

**Williams, E.S., Yuill, T., Artois, M., Fischer, J. & Haigh, S.A. (2002).** Emerging infectious diseases in wildlife. *Sci Tech Rev Int Off Epizootics* 21, 139–157.

**Williams, E.S., Miller, M.W., Kreeger, T.J., Kahn, R.H. & Thorne, E.T. (2002).** Chronic wasting disease of deer and elk: a review with recommendations for management. *J Wildl Manag* 66, 551–563.


# Micro Shorts

## Biology of Living Fungi CD-ROM

The Fungal Cell Biology Group at the University of Edinburgh, in association with the British Mycological Society, has developed on CD-ROM a compilation of movies that illustrates key aspects of the cell biology of living filamentous fungi. The movies have been obtained using confocal microscopy and show time-lapse sequences and three-dimensional reconstructions of fungal cells stained with fluorescent dyes and/or expressing GFP.

The aim of this publication is to provide a valuable resource and powerful educational tool showing the dynamic nature of fungal cells. The target audience is anyone interested in fungal biology, particularly students and those teaching mycology. The movies can be readily imported into Microsoft Powerpoint® presentations. Click on **www.fungalcell.org** to preview the package, which costs £20 (incl. UK p& p). Order via the website.

## An Introduction to Practical Microbiology

This DVD has been produced at Manchester Metropolitan University by SGM Education Group Convener, Professor Jo Verran, with the assistance of some of her students and Dean Madden of the NCBE. It includes a 30 minute film with commentary describing basic laboratory skills in microbiology such as aseptic technique, making streak plates and Gram staining. Also playable in chapters, it is accompanied by a 36-page colour text in PDF format which can be printed off.

This package is suitable for all beginners in practical microbiology, including school students. The price is £30. Contact Jo Verran for further information: **j.verran@mmu.ac.uk**

## Mushrooms in Art Registry

Anyone interested in the connection of people of different cultures with mushrooms learns that, until modern times, there are many gaps in our knowledge. On the assumption that paintings may yield useful information a *Registry of Mushrooms in Art* has been compiled. Its purpose is *'to contribute to the understanding of the relationship between mushrooms and people as reflected in works of art from different historical periods, and to provide enjoyment to anyone interested in the subject.*

The Registry includes about 600 entries, mainly European paintings and a few tapestries. It is subdivided into art periods, a taxonomic listing, works depicting vendors and kitchen scenes, and people collecting mushrooms. The Registry, which is sponsored by the North American Mycological Association, can be found at **http://members.cox.net/mushroomsinart/** For many entries there are links to websites where reproductions can be found. Suggestions for further entries are welcome. Contact the curators: Elio Schaechter (mschaech@sunstroke.sdsu.edu), Tjakko Stijve (tjakko.stijve@bluewin.ch) or Hanns Kreisel (Hanns.Kreisel@gmx.de).

## July Council Meeting

### Editor of *Microbiology Today*

● Council welcomed **Dr Gavin Thomas**, who takes up his post as Editor from 9 September and appointed him as a member of Council. The outgoing Editor, Dr Meriel Jones, said she had enjoyed her time in the job and wished her successor well for his future term.

### Attendance at CRAC Graduate Schools

● Council approved a recommendation from Treasurer's Committee that Postgraduate Student Members, who are ineligible for funding from Research Councils or other studentship-awarding bodies to attend CRAC Graduate Schools, could in future apply to the President's Fund for support. This would be in addition to the member's entitlement to a normal grant (see p. 177 for details).

### HEFCE Consultation on Postgraduate Research Degree Programmes

● Council agreed that the HEFCE document encapsulated common practice, but noted that there was need for a good balance between postgraduates and postdoctoral fellows in any research grouping, and the need for flexibility of interpretation of the 4-year completion rule. Some concern was expressed that there was no mention of outputs, such as the expectation that students should publish research papers from their work.

### SGM and the media

● Council was pleased to hear that Council and Society members had received extensive coverage on Slovenian television during the recent FEMS Congress in Ljubljana.

### Enhancements to the SGM journals website

● Council noted that a number of enhancements had been ordered for our journals at the HighWire site and these would be installed in due course. They include enabling Google to search full-text articles and enhancements to the home and archive pages.

### Retiring members of Council

● Council was chaired for the last time by **Professor Sir David Hopwood**, who thanked the retiring elected members, **Professor Colin Howard** and **Professor Dave Kelly**, for their contributions during their terms. He also thanked the outgoing Editor of *Microbiology Today*, **Dr Meriel Jones**, for her excellent work leading the in-house team to produce a most attractive magazine, which brought great credit to SGM. Council members expressed their appreciation for Professor Hopwood's contribution over his 3 years of office by a round of applause.

● *Alan Vivian, General Secretary*

## Jobs on the Web

● Do you have a post to fill?

● Are you seeking a job or a PhD studentship?

SGM can help! Microbiology job vacancies, studentships and postdocs are now advertised on the SGM website. This new service is currently free for SGM members. See the SGM 'Jobs' page to browse the latest vacancies or find out how to advertise a post. Follow the links from the 'Noticeboard' page at **www.sgm.ac.uk/noticeboard.cfm**. For further information contact Faye Jones (f.jones@sgm.ac.uk).

## New Members of Council and Group Committees 2003

### Council

Following the call for nominations to fill three vacancies for elected members of Council and a ballot of all ordinary members of the Society, the following have been elected to serve for 4 years from 9 September 2003:

| | |
|---|---|
| Professor Lorna A. Casselton | University of Oxford |
| Professor Nicholas H. Mann | University of Warwick |
| Professor Tony C. Minson | University of Cambridge |

Biographies of the new Council Members appear on p. 175.

A profile of Dr Gavin Thomas, new Editor of *Microbiology Today* can also be found on p. 175.

### Groups

New Committee Members elected by postal ballot (Clinical Microbiology and Virus Groups) or elected unopposed (all other Groups) to serve for 3 years from 9 September 2003 are as follows:

**Cells & Cell Surfaces**

| | |
|---|---|
| K.A. Homer | King's College London |
| M.P. Stevens | IAH, Compton |
| I.C. Sutcliffe | University of Northumbria |

**Clinical Microbiology**

| | |
|---|---|
| M.R. Barer | University of Leicester |
| S.C. Clarke | Meningococcus/Pneumococcus Lab, Glasgow |

**Clinical Virology**

| | |
|---|---|
| J. Breuer | St Bartholomew's Hospital, London |
| P. Mackie | Royal Hospital for Sick Children, Glasgow |
| P.S. Rice | St George's Hospital, London |
| S.J. Skidmore | Princess Royal Hospital, Telford |

H.J. O'Neill has taken over as Group Convener

**Education & Training** – *No vacancies*

**Environmental Microbiology**

| | |
|---|---|
| I.M. Head | University of Newcastle |
| G.I. Paton | University of Aberdeen |
| D.A. Pearce | British Antarctic Survey, Cambridge |

**Eukaryotic Microbiology** – *No vacancies*

**Fermentation & Bioprocessing** – *1 vacancy*

| | |
|---|---|
| R. Dennett | Eden Biopharm, Ellesmere Port |
| P.A. Hoskisson | John Innes Centre, Norwich |

**Food & Beverages** – *No vacancies*

**Irish Branch**

| | |
|---|---|
| C.L. Lowery | University of Ulster, Coleraine |
| S.G. Smith | Trinity College Dublin |

**Microbial Infection**

| | |
|---|---|
| N.P. Minton | Queen's Medical Centre, Nottingham |
| K. Robinson | University of Nottingham |

**Physiology, Biochemistry & Molecular Genetics**

| | |
|---|---|
| G.W. Black | University of Northumbria |

**Systematics & Evolution**

| | |
|---|---|
| S.P. Cummings | University of Sunderland |
| N.A. Logan | Glasgow Caledonian University |
| J.O. McInerney | National University of Ireland, Maynooth |
| H.J. Rolph | Glasgow Dental School |

**Virus**

| | |
|---|---|
| N.M. Almond | NIBSC, Potters Bar |
| D.J. Blackbourn | Institute of Virology, Glasgow |
| L.K. Dixon | IAH, Pirbright |
| G.W.G. Wilkinson | University of Wales College of Medicine, Cardiff |

# New Elected Members of Council

## Professor Lorna Casselton

Lorna graduated in Botany at University College London and continued in the same department to do her PhD and postdoctoral research in fungal genetics. After 25 years teaching genetics at Queen Mary College London, she decided in 1991 to devote her time to research and moved to Oxford where she held a Research Fellowship from BBSRC for 10 years. She is now Emeritus Professor of Fungal Genetics at Oxford and was awarded a Leverhulme Emeritus Fellowship this year.

There has only been one fungus in Lorna's life – *Coprinus cinereus*. Her research in recent years has focused on dissecting the complex mating type system of this fungus and unravelling the molecular basis of mate recognition. With its genomic sequence now available, her current interest is in developing the necessary tools for genome analysis. She is doing this in collaboration with the mushroom research group at Horticultural Research International, Wellesbourne.

## Professor Nick Mann

Nick Mann trained initially as a biochemist at the University of Liverpool and became interested in cyanobacteria through a final year research project on cyanobacteria (then known as blue-green algae) in the laboratory of Noel Carr. This led on to PhD research in the same laboratory.

The first postdoctoral position was as a Guinness Fellow in the Chemical Microbiology Unit at Oxford University working with Professor Joel Mandelstam FRS on *Bacillus subtilis* sporulation. This was followed by a spell at the University of Leeds in the laboratory of Dr Simon Baumberg, at the time when gene cloning was just starting, expressing *B. subtilis* genes in *E. coli*.

Finally, a permanent position arrived in the form of a lectureship in Biological Sciences at Warwick. Despite temporary excursions into areas such as plasmid biology, the research returned to the primary interest in cyanobacteria and has remained there ever since.

## Professor Tony Minson

Tony Minson graduated in Microbiology at the University of Birmingham in 1965 and, as a post-graduate, studied the control of histidine synthesis in *Neurospora Crassa* at the John Curtin Medical School, Australian National University. Interactions with members of Frank Fenner's department at the John Curtin stimulated his interest in virology and in 1970 he returned to Birmingham and joined Peter Wildy's department to initiate a programme of research on tobacco rattle virus in collaboration with Graham Darby.

In 1976 he moved to Cambridge with Peter Wildy and began work on the biology and pathogenesis of herpes simplex viruses, the subject which has remained the focus of his research. In 1991 he was appointed Professor of Virology and has recently been appointed Pro-Vice Chancellor. He has served as a member of SGM Council, as a member of the Governing Body of the Institute for Animal Health and as a member of the Technical Sub-committee of the ACGM. He was elected a Fellow of the Academy of Medical Sciences in 2002.

# New Editor of *Microbiology Today*

## Dr Gavin H. Thomas

I became interested in microbiology at school on reading John Postgate's *Microbes and Man*. This resulted in me moving from my native North Yorkshire to Bristol to read Microbiology.

After enjoying a short molecular microbiology project with Peter Bennett, I decided to study for a PhD. This prompted a move to Birmingham where I started working with Jeff Cole on my favourite organism, *Escherichia coli*. We characterized the periplasmic nitrate reductase of *E. coli* and did some early work on the Tat protein export system.

Continuing to work with *E. coli*, I moved to the John Innes Centre to postdoc with Mike Merrick. Our work on ammonium transport started my interest in membrane transport proteins, which I continued with Dave Kelly in Sheffield working on TRAP transporters.

In 2002 I moved to York to a lectureship in the Biology department to work on membrane transport proteins and *E. coli* bioinformatics.

## News of Members

The Society notes with regret the death of **Professor E. Glyn V. Evans** (Member since 1986).

## Staff News

Congratulations to Professional Affairs Administrator, **Faye Jones**, who married Neil Stokes in Aberdeen on 30 August. The happy couple spent their honeymoon cruising the Mediterranean and will make their home in Reading.

We are sorry to say goodbye to two members of the JGV editorial team who have done sterling work to contribute to the success of the journal. Deputy Managing Editor **Catherine Tarbatt** is moving on to a post in medical communications and staff editor **Lucia Primavesi** has decided to go back to the bench. She will be working as a postdoctoral researcher at IACR Rothamsted.

## SGM Membership Subscriptions 2004

*The following rates were agreed at the AGM of the Society on 9 September 2003.*

| **Ordinary Member** | £ | US$ |
|---|---|---|
| ■ Membership subscription (including *Microbiology Today*) | 45.00 | 78.00 |
| Additional concessionary subscriptions for publications: | | |
| ■ *Microbiology* | 85.00 | 160.00 |
| ■ *Journal of General Virology* | 85.00 | 160.00 |
| ■ *Int J Syst Evol Microbiol* | 85.00 | 160.00 |
| ■ *Journal of Medical Microbiology* | 45.00 | 78.00 |

| **Postgraduate Student or Retired Member** | £ | US$ |
|---|---|---|
| ■ Membership subscription (including *Microbiology Today*) | 20.00 | 35.00 |
| Additional concessionary subscriptions for publications: | | |
| ■ *Microbiology* | 40.00 | 75.00 |
| ■ *Journal of General Virology* | 40.00 | 75.00 |
| ■ *Int J Syst Evol Microbiol* | 85.00 | 160.00 |
| ■ *Journal of Medical Microbiology* | 45.00 | 78.00 |

| **Undergraduate Member (UK and Republic of Ireland)** | £ | US$ |
|---|---|---|
| ■ Membership subscription (including *Microbiology Today* – no concessionary subscriptions to journals are available to Undergraduate Members) | 10.00 | NA |

| **School Member (UK and Republic of Ireland)** | £ | US$ |
|---|---|---|
| ■ Membership subscription (including *Microbiology Today* – no concessionary subscriptions to journals are available to School Members) | 10.00 | NA |

| **Corporate Member** | £ | US$ |
|---|---|---|
| ■ Membership subscription (Tier 1/Tier2) (including *Microbiology Today* – no concessionary subscriptions to journals are available to Corporate Members) | 500.00/350.00 | NA |

Members are reminded that their 2004 subscriptions are due for payment by **1 December 2003**.

As in previous years, no journal or meetings information will be despatched to members who are in arrears, and there will be no guarantee of provision of back numbers of journals for members who pay their subscription late.

### Payment by direct debit or continuous credit card

Subscription notices were despatched recently to all members paying by direct debit or by continuous credit card arrangement. To continue your present status and journal requirements, no further action is necessary. However, if you pay by continuous credit card, you should check that the card number and expiry date on the subscription notice are correct. To change your membership status or journal requirements for 2004, or your credit card details, you should have amended your subscription notice and returned it to the membership office by **14 November 2003**. However, if you have missed this deadline, your amended notice will be accepted if it is submitted immediately.

### Payment against invoice

Invoices were despatched recently to all members who pay by this method. If you did not receive one, please inform the Membership Office.

### Subscriptions waived for unemployed members

As in previous years, subscriptions may be waived at the discretion of the Society for unemployed members under the age of 35 who are resident in the UK. If you are eligible and wish to benefit in this way in 2004, you should send a signed statement that you are currently unemployed to the Membership Office before **30 November 2003**. (Please note that no increase in journal requirements will be permitted.)

### Income tax relief on membership subscriptions

Members who are liable for UK income tax are reminded that their annual subscriptions to the Society have been approved by the Inland Revenue as qualifying for income tax relief. Any member who would like further information or has difficulty in obtaining this relief should contact the Executive Secretary.

# New Group Conveners

## Clinical Virology

## Dr Hugh O'Neill

Hugh O'Neill is a consultant clinical scientist in the Regional Virus Laboratory at The Royal Hospitals Trust in Belfast. He was first employed as a medical laboratory technician and became a fellow of the Institute of Biomedical Sciences in 1967 after specializing in bacteriology and virology.

He obtained an Open University degree in 1976 and a PhD in 1985 after studying antibody responses in CMV infection in transplant patients. In 1994 he became a member of the Royal College of Pathologists by examination. He has worked on various clinical research projects on various aspects of virus infection and diagnosis of disease. He is an enthusiastic advocate of real-time molecular diagnostics. His current specific interests are in the diagnosis of infectious disease of the respiratory tract, virus load quantitation and the molecular epidemiology of rotaviruses circulating in the community.

## Food & Beverages

## Dr Bob Rastall

Bob Rastall is Senior Lecturer in Food Biotechnology in the School of Food Biosciences at the University of Reading. He obtained his degree in Applied Biology at the University of Greenwich and stayed at that institution for his PhD on 'The cell surface biochemistry of *Erwinia amylovora*'.

He then carried out postdoctoral research at the University of Westminster on enzymatic oligosaccharide synthesis prior to joining the University of Reading. His research interests are in the area of biomanufacture of food ingredients, particularly functional food ingredients. Specific areas include carbohydrate metabolism in probiotic bacteria, enzymatic manufacture of novel prebiotic oligosaccharides and enzymatic manufacture of bacterial pathogen receptor sequences.

# Grants

## President's Fund

The President's Fund offers financial support to younger members of the Society for one of the following:

1. Travelling to present a paper or a poster on a microbiological topic at a scientific meeting
2. Attending a short course (up to two weeks) including UK GRADschools
3. Making a short research visit – larger awards are available for short research visits

### 1 & 2 – Smaller Awards

Maximum grants are:

- £125 for attendance at meetings/courses in the country of residence
- £200 for travel to another European country
- £300 for travel outside Europe
- £300 for attendance at a UK GRADschool

### 3 – Larger Awards (research visit)

Up to £2,000 is available for making a short research visit of up to two months. The host institution may be overseas or in the country of residence.

All applicants must be resident and registered for a higher degree, or in a first postdoctoral position, in a country in the European Union. Only one application may be made to the fund during the term of a studentship or fellowship. The full rules of the scheme are published on the SGM website, from which application forms may be downloaded.

### New!

Grants of up to £300 are now available from the President's Fund to attend a UK GRAD School. See Gradline (p. 182) for details of these useful courses.

Details of all grant schemes are available on the SGM website at **www.sgm.ac.uk**. Please consult these before making an application. You can download the application forms for schemes where these are required. Click on the 'Grants and Funding' button for details.

Any enquiries should be made to the Grants Office, SGM, Marlborough House, Basingstoke Road, Spencers Wood, Reading RG7 1AG [Tel: +44 (0)118 988 1821; Fax: +44 (0)118 988 5656; email: grants@sgm.ac.uk].

## Vacation Studentships 2004

The Society offers a limited number of awards to enable undergraduates to work on microbiological research projects during the summer vacation. The purpose of the awards is to provide undergraduates with experience of research and to encourage them to consider a career in scientific research. The studentships provide support at a rate of £160 per week for a period of up to 8 weeks. An additional sum of up to £400 for specific research costs may also awarded. Applications on behalf of named students are now invited from SGM members in higher education institutions and research institutes. Details and application forms are available on the website.

The closing date for applications is **27 February 2004**.

## Postgraduate Conference Grants

Postgraduate Student Members of SGM currently resident and registered for a higher degree in the UK or another European Union country are eligible for a grant to cover the costs of accommodation and travel in attending ONE of the following Society meetings in 2004: University of Bath, March/April; Trinity College Dublin, September; or any other SGM Group or Branch meeting. Application forms giving full details of the scheme were sent to all Postgraduate Student Members in the EU with their subscription invoices. A form can also be downloaded from the SGM website.

## Undergraduate Microbiology Prizes

The scheme to encourage excellence in the study of microbiology by undergraduate students continues to be well received in universities in the UK and Republic of Ireland. Institutions offering an appropriate microbiology course were invited to nominate a student for an SGM prize, based on good performance in microbiology in the penultimate year of study for a BSc. The department was able to choose the type of assessed work for which the prize was awarded. Of the 64 departments circulated, 53 made nominations. Each prizewinner will receive a certificate, a cheque for £100 and a year's free Undergraduate Membership of the Society.

Undergraduate Microbiology Prizes will be awarded annually and the invitations for nominations in 2004 will be circulated next May. Details are also available on the SGM website.

## Seminar Speakers Fund 2003/2004

The purpose of the Seminar Speakers Fund is to promote talks on microbiological topics in departmental seminar programmes. Applications are invited from Higher Education Institutions where microbiology is taught for grants of up to £200 towards the travel, and if necessary, accommodation, expenses of an invited speaker. See website for full rules. Applications will be dealt with on a first come, first served basis during the academic year, which is defined as running from September 2003 to June 2004. Written submissions should be sent to the Grants Office at SGM HQ.

## Public Understanding of Science Awards

Are you planning any projects to promote the public understanding of microbiology? Have you got a National Science Week event in mind? SGM can help. Grants of up to £1,000 are available to fund appropriate activities. Applications are considered on a first come, first served basis throughout the calendar year.

## Retired Members Grants

These cover the costs of accommodation and the Society Dinner for Retired Members of the Society in attending one SGM meeting each year. An application form is on the website.

# Meetings

## Meetings on the web

For up-to-date information on future Society meetings and to book on-line see: **www.sgm.ac.uk**

## Meetings organization

The SGM meetings programmes are organized by the committees of the special interest groups, co-ordinated by the Scientific Meetings Officer, **Professor Howard Jenkinson**. Suggestions for topics for future symposia are always welcome. See p. 203 for contact details of Group Conveners.

Administration of meetings is carried out by **Mrs Josiane Dunn** at SGM Headquarters, Marlborough House, Basingstoke Road, Spencers Wood, Reading RG7 1AG (Tel. 0118 988 1805; Fax 0118 988 5656; email meetings@sgm.ac.uk).

## Offered papers and posters

Many Groups organize sessions for the presentation of short oral papers or allow intercalated papers within their symposia. Offered posters are welcome at all Society meetings.

### Offered posters

Each poster should be associated either with the Plenary Session topic or with a Group. The subject content of the latter should be relevant to the remit of a Group (see website for details); it does not have to relate to the topic of the Group Symposium taking place at a particular meeting. General Offered Posters will not be accepted.

## Abstracts

Titles and abstracts for all presentations are required in a standard format and should be submitted through the SGM website. Deadlines for submissions are published in *Microbiology Today* and on the web. For further information contact the Events Administrator.

## Abstracts Book

**153rd Meeting**
**UMIST, Manchester, 8–11 September 2003**

The full text of the abstracts book is now available as a PDF file on the SGM website.

## Future Meetings

**SPRING 2004 – 154th Meeting**
**University of Bath**
**29 March–2 April 2004**

### ● Plenary: Microbe–vector interactions in vector-borne diseases

29–30 March

Organizers: S.H. Gillespie, H.F. Jenkinson, A. Osbourn, P.C.F. Oyston & R.E. Randall

### ● Speakers

29 March

B. MAHY (NCID, USA)
*Vector-borne diseases*

S. RANDOLPH (Oxford)
*Evolution of tick-borne diseases*

S. BLANC (Montpellier, France)
*Insect transmission*

S.W. DING (Riverside, USA)
*Interactive silencing of host gene expression*

A.G. BARBOUR (Irvine, USA)
*Reducing the prevalence of Borrelia in ticks*

R.M. ELLIOTT (Glasgow)
*Bunyavirus/mosquito interactions*

S. HIGGS (Texas, USA)
*How do vectors live with their viruses?*

S. WEAVER (Texas)
*Induction of vector competence*

30 March

P. MELLOR (Pirbright)
*Climatic change*

N. RATCLIFFE (Swansea)
*Immune systems in vectors*

S. MACFARLANE (SCRI, Dundee)
*Nematode transmission*

M.J. TAYLOR (Liverpool)
*Wolbachia host–symbiont interactions*

E. FIRKRIG (New Haven, USA)
*Human granulocytic ehrlichiosis*

J. HINNESBUSCH (NIAID, USA)
*Plague in fleas*

P.W. ATKINSON (Riverside, USA)
*Transgenic malaria*

G. TARGETT (London SHTM)
*Vaccines targeting vectors*

### ● Other symposia and workshops

● Surface mediators
**Cells & Cell Surfaces Group**
30 March
Organizers: N.J. High (nicky.high@man.ac.uk) & D.G.E. Smith (dgesmith@vet.ed.ac.uk)

● Imported infections
**Clinical Microbiology Group/ British Infection Society**
31 March–1 April
Organizer: S.H. Gillespie (stepheng@rfc.ucl.ac.uk)

● Healthcare-associated virus infections
**Clinical Virology Group**
30 March
Organizer: B. Cohen (bernard.cohen@hpa.org.uk)

● Bored with the same old culture? Inoculate new life into your classroom!
**Education & Training Group**
31 March
Organizers: M.R. Adams (m.adams@ surrey.ac.uk), H.J. Sears (h.j.sears@leeds.ac.uk) & J. Verran (j.verran@mmu.ac.uk)

● The role and impact of fungi on biogeochemical cycles
**Environmental Microbiology and Eukaryotic Microbiology Groups/ British Mycological Society**
31 March–1 April
Organizer: G.M. Gadd (g.m.gadd@dundee.ac.uk)

● Meeting the challenges of producing proteins and antibodies
**Fermentation & Bioprocessing Group**
29 March
Organizers: M.G. Duchars (matthew.duchars@avecia.com) & J. Miller (julie.miller@mhra.gsi.gov.uk)

● Fundamental and applied aspects of bacterial toxins
**Microbial Infection Group**
31 March–1 April
Organizers: O. Sparagano (olivier.sparagano@ncl.ac.uk) & N. Fairweather (n.fairweather@ic.ac.uk)

● RNA–protein interactions in the regulation of gene expression
**Physiology, Biochemistry & Molecular Genetics Group**
31 March–1 April
Organizer: I. Stansfield (i.stansfield@abdn.ac.uk)

● Clinical diagnostics: current applications and future prospects
**Systematics & Evolution and Clinical Microbiology Groups**
29–30 March
Organizers: R.A. Whiley (r.a.whiley@qmul.ac.uk), S. Clarke (stuart.clarke@northglasgow.scot.nhs.uk), S.H. Gillespie (stepheng@rfc.ucl.ac.uk) & G. Saddler (gerry.saddler@sasa.gsi.gov.uk)

● Viruses and signalling (Symposium 1)
**Virus Group**
29–30 March
Organizers: W.S. Barclay (w.s.barclay@reading.ac.uk ) & K.N. Leppard (keith.leppard@warwick.ac.uk)

● Viral hepatitis (Symposium 2)
**Virus Group**
1–2 April
Organizers: M. Harris (mharris@bmb.leeds.ac.uk), D.J. Rowlands (d.j.rowlands@bmb.leeds.ac.uk) & J. McLauchlan (j.mclauchlan@vir.gla.ac.uk)


*A leaflet about the meeting is enclosed with this issue. An A3 poster (above) is also available from the Events Administrator if you would like to help publicize the meeting.*

### Workshops

**Virus Group**

**31 March**

*Plant viruses* (half day)

Organizers: S.A. MacFarlane (s.macfarlane@scri.sari.ac.uk) & J.P. Carr (jpc1005@hermes.cam. ac.uk)

*Retroviruses* (half day)

Organizers: J.C. Neil (j.c.neil@vet.gla.ac.uk) & N.M. Almond (nalmond@nibsc.ac.uk)

*DNA Viruses* (1 day)

Organizers: S. Efstathiou (se@mole.bio.cam.ac.uk), M.A. Skinner (michael.skinner@bbsrc.ac.uk) & D.J. Blackbourn (d.blackbourn@vir.gla.ac.uk)

*RNA virus positive sense viruses* (half day – a.m.)

Organizers: M.D. Ryan (martin.ryan@st-and.ac.uk) & S.G. Siddell (stuart.siddell@bristol. ac.uk)

*RNA virus negative sense/ dsRNA viruses* (half day – p.m.)

Organizers: W.S. Barclay (w.s.barclay@reading.ac.uk) & P.E. Digard (pd1@mole.bio.cam. ac.uk)

**1 April**

*Human papillomaviruses* (one day)

Organizers: S. Graham (s.v.graham@bio.gla.ac.uk), I.M. Morgan (i.morgan@vet.gla.ac.uk) & J. Doorbar (jdoorba@nimr.mrc.ac.uk)

### Special Events

**SGM Postgrad Career Development Workshop**

**Evening, 1 April**

A talk on presentation skills will be followed by a session on CV writing and career planning with Sara Shinton (the Career Doctor from *Science*'s Nextwave). A buffet and drinks reception will close the workshop, giving postgrad delegates the opportunity to practise networking skills and chat informally to the speakers, staff from UK Grad and other careers specialists.

**CV clinic:** feedback will be offered on ~20 individual CVs submitted on a first come, first served basis. Please contact Jane Westwell (j.westwell@sgm.ac.uk) for further information.

**Welcome Drinks Reception**

**Sports Hall, 29 March**

**Society Dinner & Disco**

**Assembly Rooms, Bath, 30 March**

**Roman Baths evening**

**31 March**

Drinks around the pool with a difference (togas optional).

Deadline for the receipt of titles and abstracts: **28 November 2003**

## AUTUMN 2004 – 155th Meeting

**Trinity College, Dublin**

**6–9 September 2004**

### ● Plenary: Novel anti-microbial therapies

### ● Other symposia:

Lactic acid bacteria
**Irish Branch/ Food & Beverages/ Systematics & Evolution Groups**

Secreted effectors
**Cells & Cell Surfaces Group**

Antibiotic resistance
**Clinical Microbiology Group**

E-learning and distance learning
**Education & Training Group**

Epigenetic resistance
**Eukaryotic Microbiology Group**

Gram-positive cell factories
**Fermentation & Bioprocessing Group**

Zoonotic infections
**Microbial Infection Group**

Molecular chaperones and protein folding
**Physiology, Biochemistry & Molecular Genetics Group**

## Irish Branch

**An update on bacterial molecular pathogenicity**

**National University of Ireland, Galway**
**15–16 April 2004**

Organizer: Cyril Carroll (cyril.carroll@unigalway.ie

Invited speakers include:
C. HILL (University College Cork)
C.D. O'CONNOR (University of Southampton)
S. PARK (University of Surrey)
A. MORAN (National University of Ireland, Galway)

*For details of Irish Branch activities contact the Convener, Catherine O'Reilly (coreilly@wit.ie)*

## Other Events

### ● SfAM/SGM One-day Regional Meetings

A joint initiative to sponsor one-day regional meetings in the UK and Ireland has been launched by the SGM and the Society for Applied Microbiology. See the website of either society for the full rules and to download an application form: **www.sfam.org.uk** or **www.sgm.ac.uk**

### ● Infection and Immunity

**University of Bristol**
**27–28 November 2003**

The session will cover exogenous and endogenous regulation of host cell immunity: from fundamental research to clinical applications.

For further information, contact Professor Stuart Siddell, University of Bristol (Tel. 0117 928 7889; email stuart.siddell@bristol.ac.uk).

# SchoolZone

Schools Membership costs only £10 a year. For this, a named teacher representative will receive *Microbiology Today* each quarter, advance notification and copies of new microbiology teaching resources and discounted fees for attendance on SGM training courses and workshops. Application forms are available at **www.sgm.ac.uk**

Enquiries:
education@sgm.ac.uk

Website:
www.microbiology online.org.uk

## MISAC 2004 Schools Competition

### Composting: Not just a load of old rot, but a way to save the planet

*Sponsored by*

society for applied sfam microbiology

This year's secondary schools competition will focus on the value of composting and the role that micro-organisms play in this fascinating process, both in the heaps in our gardens and in commercial systems. School pupils in two age groups (Key Stage 3 and GCSE) are being asked to produce an illustrated information leaflet which encourages the use of composting as a contribution to the recycling of waste materials. This should be suitable for distribution by a local authority to the general public.

Cash prizes are up for grabs as follows

| | School | Pupil |
|---|---|---|
| 1st | £200 | £30 |
| 2nd | £100 | £20 |
| 3rd | £50 | £10 |

In addition every school entering the competition receives a pack of microbiology teaching resources and each student submitting an entry of scientific merit is sent a certificate.

Entry forms can be downloaded from **www.microbiologyonline.org.uk**

The closing date is **31 March 2004**.

MISAC wishes to express its sincere thanks to the Society for Applied Microbiology for sponsoring the 16th competition.

## More dishes on the microbial menu

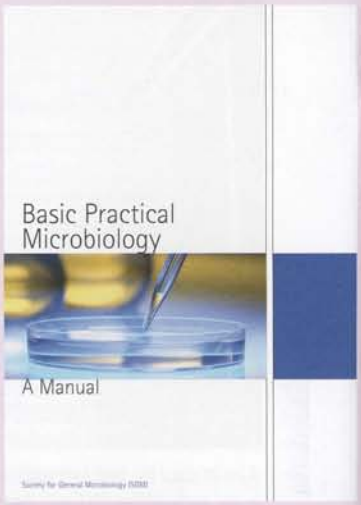
Dave Roberts of the Natural History Museum has come up with two new recipes which rely on the activities of microbes.

**Bagels** are made from a yeast dough, but why is their appearance and texture so different from that of bread? The hole in the middle is down to mechanical action, but bagels have a smooth surface and dense inside because they are cooked twice. First they are boiled, then they are baked.

If you fancy a drink with your bagel, why not try your hand at making **elderflower champagne**? The fermentation apparently relies on the natural flora of the plant material, but Dave tells us that the yeasts in the sediment in the bottom of the jar at the end of the process are not the same as the ones that can be isolated from the flowers at the start. Can anyone explain this apparent 'spontaneous generation'?

Further information on bagels and elderflower champagne is to be found on **www.microbiologyonline.org.uk**

## Basic Practical Microbiology: A Manual




The book to accompany our popular training course for schoolteachers and technicians has been revised and is now available in a glossy format illustrated with colour photographs. School Members only (or SGM members who do outreach work in schools) who would like to receive a free copy should email **education@sgm.ac.uk**

The price to others is £10 (inc. p&p within the UK, exclusive of p&p overseas).

## Schools Basic Practical Microbiology Training Courses in 2003/4

Thanks to the SGM members who are hosting courses this year. Venues and dates are as follows:

| | |
|---|---|
| University of Surrey | 19 November 2003 |
| Yale College, Wrexham | 9 December 2003 |
| University of Leicester | 21 April 2004 |
| University of Lincoln | 29 April 2004 |
| University of Plymouth | 22 June 2004 |

Booking forms are available on **www.microbiologyonline.org.uk**

## ASE Annual Meeting

### University of Reading, 8–10 January 2004

SGM will be involved in a range of activities at the meeting. Apart from our usual stand in the exhibition (come and find us on A36, which we are sharing with MISAC), we are participating in *Biology in the Real World* – a day-long programme of talks organized by bioscience learned societies, research councils and charities on Friday 9 January. Education Officer Sue Assinder will chair the health and disease strand and new President Hugh Pennington will speak on food-borne illness. On Thursday 8 January at 1230 we are collaborating with the Thames Valley Branch of the ASE and invite you to *Science in the Pub*. Over lunch and a drink, you can debate the threats from 'Modern Plagues' such as vCJD, SARS and *E. coli* O157, with experts Hugh Pennington and Ian Jones. Tickets (£5) are available in advance from SGM (email y.taylor@sgm.ac.uk), our stand on the day, or on the door.

Full details of the Annual Meeting programme are on the ASE website at **www.ase.org.uk**

## Post-16 Summer School 2004

We are delighted to announce that the second residential microbiology summer school for post-16 biology teachers is being hosted by the University of Leeds. It will take place 12–16 July 2004 in their recently refurbished and state-of-the-art teaching laboratories. An exciting programme of lectures, hands-on, workshops and visits has been planned. Evening social events will allow the delegates to relax and network. We are very grateful to Dr David J. Adams for making all the arrangements in Leeds. Details will be available soon on the web and booking forms will be distributed to all School Members next term.

## Questionnaire

Thanks to all the School Members who took the trouble to complete the questionnaire on SGM microbiology education resources and initiatives, circulated with the last issue of the magazine. The results are being analysed and a report will be published in due course.

The winner of the prize draw was **Mrs Sue Dobson**, Polam Hall School, Darlington, Co. Durham.

# GNVQ Science – Practical Microbiology Course – July 2003 Bishop Burton College, East Yorkshire

Sue Fryer

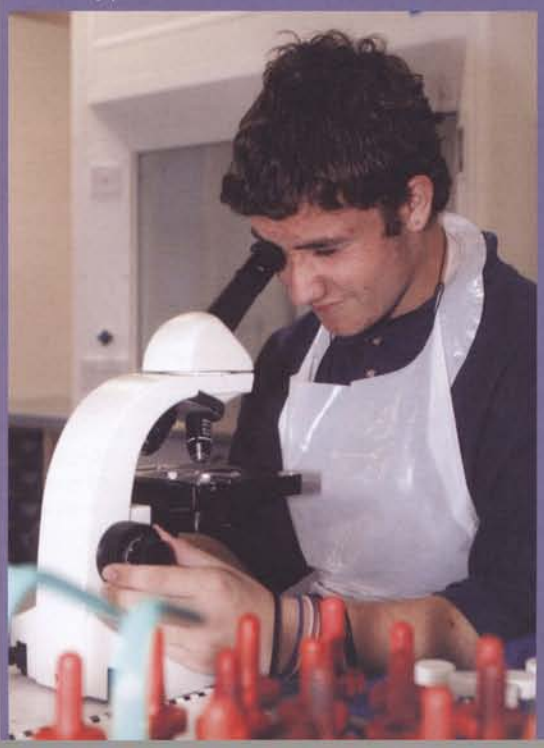
Inspired by the SGM Summer School at the University of Reading last year, and also the completion of our brand new Science Centre here at Bishop Burton College, I felt that we could offer local schools the chance to use our laboratories for some of their microbiology practical work.

Local schools were contacted, with a view to opening our Science Centre to year 12/13 'A' level Biology students studying the A2 option 'Microbes and disease'. The response was positive and this is still in the pipeline for January 2004.

However the main response was, unexpectedly, from the Head of Science at a large comprehensive school in Kingston-upon-Hull who had 160 year 10 GNVQ Science students who wanted to come to the college to complete the microbiology practical work necessary for their portfolios. This was a much bigger number of students than we had envisaged, but we decided that if we split them into four groups, each coming into the lab for 1·5 days, we could cope with the numbers.

A PUS grant from the SGM enabled us to pay for most of the consumables and to produce a booklet containing all the work they needed to complete for inclusion in their science portfolios.

On the first day we covered the health and safety implications of working with micro-organisms, aseptic technique and environmental sampling. The students then went on to make a smear slide of live yoghurt and learnt how to look at bacteria under the microscope using oil immersion technique. In the afternoon we talked about antibiotics and set up some antibiotic sensitivity plates.



The following day we looked at and evaluated their results from the previous day's work and then we moved to on to Gram staining. All the students benefited from the individual use of good quality microscopes and they all produced a successful slide.

Several teachers and technicians came from the school as well and they all thoroughly enjoyed working with the students in our labs; we are hopeful that the course will run again next year. The school science staff were extremely enthusiastic about the course and gave us a lot of positive feedback. For example, Karen Preston, Head of Science Faculty, Winifred Holtby School Technology College, Kingston upon Hull, wrote:

*'I would like to thank you and your team for the microbiology course that you prepared and delivered for us. The pupils thoroughly enjoyed their day and a half and the feedback was extremely positive. All the pupils felt that they had been given an insight into post-16 science and are more interested as a result. The portfolio work gained was very valuable and covers all the biology skills needed for Unit 1.'*

*'We were very pleased with our GNVQ results this year and expect next year to be even better. As we begin this new year we have over 170 pupils starting the GNVQ course. As the pupils found the visit so useful we would be grateful if you would consider running it again for our new Year 10.'*

At the end of the course each group filled in a short questionnaire about the course, facilities, etc. On completion of these, each student was given a pack of posters, information sheets, a badge and a pencil, provided by the SGM.

Over 80 % of the students rated the college facilities as excellent and thought it would be a good idea for students to return to the labs in the future. 98 % thought that the work they had done would help them with their course grades.

All in all we had an enjoyable, if hectic, two weeks with the students, and hopefully we have inspired a few budding microbiologists along the way!

***Sue Fryer, Laboratory Manager and Microbiology Tutor, Bishop Burton College, Beverley, East Yorkshire, UK. email fryers@bishopb-college.ac.uk***

*This project could not have been carried out without financial support from the SGM Developments in Education Public Understanding of Science Fund. Up to £1,000 is available for events such as this which promote microbiology to schools and the public. Please contact Janet Hurst if you are interested in this grant scheme (j.hurst@sgm.ac.uk) or download the rules and an application form from the web (www.sgm.ac.uk/grants).*

# Gradline

If you have any stories or news for publication in Gradline, or if you would like to see any topics featured, contact Gradline Editor Jane Westwell (**j.westwell@sgm.ac.uk**).

## GRADschools – All they're CRACked up to be!

Jane Westwell

The UK Grad programme is an initiative run by CRAC (Careers Resource Advisory Service) which aims to 'support students, supervisors, universities, and employers to realise the value of postgraduate talent'. One of UK Grad's major activities is the organization of 3-, 4- and 5-day residential courses (GRADschools) throughout the year at venues around the country. The courses offer postgrads time out of the lab and the opportunity to assess existing (transferable) skills and develop new ones. The 5- and 4-day courses place emphasis on team building and career management. The 3-day course focuses on career development. Research Council and final year Wellcome Trust funded students are eligible for free places on the courses.

The vast majority of students attending these courses are enthusiastic in their praise and for some the courses are really life-changing.

In May, in the interests of career development (mine and SGM postgrad members'), I attended the first ever careers-focused 3-day GRADschool as a course mentor. I was already familiar with GRADschools, having attended (and benefited hugely from) the traditional 5-day course as a postgrad in the dim and distant past. Mentoring proved to be a new challenge.

Mentors arrived the evening before the course and had a preliminary session before the students arrived. We had come from a variety of backgrounds (including science administration, publishing and civil service) and each of us had experience and skills that would prove useful during the week. We were all allocated to a tutor group and our role was to play an active part supporting the tutor and facilitating the students' progress during the course.

The course was very intensive and exhilarating. Without going into too much detail, I can tell you that on the first day none of us knew anything (or, in many cases, cared much) about career development strategy. However, by the end of the course all the students in my group had started to think beyond the end of their projects and the majority had a clearer idea of their career aspirations. Most importantly, they all knew how to plan their next career move. Generally, the course lifted the spirits of the researchers who were ground down by the prospect of writing up and refreshed the sufferers of mid-project blues.

The course was also enlightening for me. As a provider of careers information for the SGM it was interesting to discover another side to careers advisory work and it also helped me to plan some new activities for our members.

I asked Claire Cotterill, PhD student at the University of Edinburgh and a student in my group, her thoughts on the GRAD school. She went on the course with four other students from her lab.

**Q** *How did you find out about GRADschools?*

I found out about the GRADschool courses by word of mouth. When I was in my first year one of the final year students in my department had just returned from a course and was fervently urging everyone else to make the effort to attend during their final year.

**Q** *Why did you decide to attend the GRADschool and the careers course in particular?*

Half way through my final year it dawned on me that I had done little in the way of planning my career or for life after my PhD. It's easy to be obsessed about your progress with your research and put off making any plans until you've done the next set of experiments/ finished in the lab/finished your thesis. Going on the GRADschool careers course seemed like a minimal effort approach to a guilt free life! There were a variety of courses on offer, lasting from 3 to 5 days, and they seemed to be hosted in plush surroundings. The deciding factor for us was a hydro spa and swimming pool complex! We were frequently asked what possessed us to travel to Gatwick from Edinburgh when there were several GRADschool courses scheduled in the North of England!

We chose the careers course because it incorporated lots of elements of the other GRADschool courses, such as practice at public speaking and some teamwork, but it also focused on raising awareness of the skills you have and improving your employability.

**Q** *Did you have any expectations of the course?*

Initially I was looking forward to having a break from my research and then the emails about pre-course preparation started to come and it began to feel more and more like a hassle. I was asked to read through some of the material before arrival, prepare a CV and fill in a mock application form for CV and interview workshops. Although I remember feeling disgruntled by the prospect of more work, when I was on the course I really appreciated the need for preparation.

I also remember a moment of apprehension as I scanned through the course programme and noticed the phrase 'ice breaker'. I had visions of being forced to do embarrassing role-plays and make animal sounds. '*Hi, my name's Claire and I'm going to impersonate an aardvark!*' Fortunately, there was no such humiliation and ridiculous role-playing was reserved for when we all knew each other. After several hours of lectures and discussion groups going outside to play paper, scissors and stone as a large group of wizards, giants and dwarves was a welcome break.

BELOW:
Students enjoying a game of 'wizards and dwarves'.
PHOTO THE UK GRAD PROGRAMME

TOP RIGHT
*From left to right:* Claire Cotterill, Katie Matthews, Clive McKimmie and Clem Hindley.
COURTESY CLAIRE COTTERILL

LOWER RIGHT:
A GRADschool tutor group.
PHOTO THE UK GRAD PROGRAMME

**Q** *How did it go?*

The actual course passed in a flash. The programme was really well organized and our own preparation meant the workshops could cover everything from what should go in an application letter to how to interview and be a successful interviewee.

Frequently during the course the tutors referred to 'the process', in an attempt to persuade us that there really was method in their madness. By the end of the course I understood what they were talking about; I had an idea of what sort of career I would really enjoy and be suited to, and how to go about getting it. Having always chosen academia (or studying) as opposed to other types of career I have never had to face assessment centres or comprehend the job descriptions typical in business and commerce. I suddenly realized that I have developed a lot of desirable (and frequently requested) job skills during my PhD. At the end of the 3 days I was exhausted by the sheer *tour de force* nature of the course, but it had enabled me to discover a lot about myself and my future career direction.

**Q** *Has it made a difference to your career plans?*

I would say yes, a big difference. I came away with a more positive attitude to career planning. Talking with tutors and mentors brought suggestions of a few careers I hadn't previously thought of, reinforcing the idea that I have more than one option in terms of a career. The course also convinced me that networking and asking for help are good ways of speeding up the process of finding a job and ultimately a career I'm happy with.

**Q** *Would you recommend the GRADschool to other PhD students?*

I would recommend the course to final year students in particular, because above all else it will end up giving them respite from the pressures of generating data. They'll also get great support from the many students they'll meet in the same (and sometimes worse) position! Apart from having several days away in a nice hotel, the course is a great boost to get you thinking about, and doing something about your next career move.

■ *More information about GRADschools (including venues and dates) is available from the UK GRAD website* ***www.grad.ac.uk***

## SGM supports GRADschools

At the Education & Training Group session at the SGM meeting in Edinburgh last April, GRADschool funding came up for discussion. In response, SGM Council has decided to launch a new grant scheme to support GRADschool attendance by Postgraduate Student Members who are not able to obtain sponsorship from their funding bodies. If you are not funded by the BBSRC, MRC, EPSRC or the Wellcome Trust you will be eligible to apply for a grant of £300 towards the cost of course fees. Grants will be available for courses in 2004 and application forms are available from **www.sgm.ac.uk/grants**

## SGM Career Development Workshop in 2004

We will be running an evening workshop, *PhD and Beyond*, at the Spring 2004 meeting in Bath. A talk on presentation skills will be followed by a session on CV writing and career planning with Sara Shinton (the Career Doctor from *Science*'s Nextwave). Sara is a freelance careers consultant and worked with UK Grad to develop the careers course.

A buffet and drinks will close the workshop, giving you the opportunity to practise networking skills and chat informally to the speakers and other careers specialists.

During the afternoon preceding the workshop we will be running a CV clinic. We will review and give feedback on a first come, first served basis.

For further information, contact Jane Westwell (j.westwell@sgm.ac.uk).

## *Young Microbiologist of the Year* Competition

The UMIST meeting saw the first finals of the SGM's new science communication contest. Eight keen postgrads and postdocs, who had been selected as finalists by the Special Interest Groups and Irish Branch on the basis of their offered presentations (either oral or poster) at recent SGM meetings, gave 10-minute talks on their research. Five minutes were allowed for questions to each speaker. The standard was amazingly high and the judges, provided from the Group committees and chaired by Jo Verran, Convener of the Education & Training Group, had a very difficult job. The winners were announced at the Society dinner later that evening. The first prize of £500 went to Stephen Griffin of Leeds University for his presentation *The hepatitis C virus ion channel protein p7: characterization of a novel anti-viral drug target.* The second prize of £200 was won by Paraic Cuiv (Dublin City University) and third prize of £100 went to Heather Thompson (University of Ulster). The other finalists received £25 in recognition of their efforts and everyone will get free membership of the Society in 2004.

Further details of the competition and an entry form are available on the meetings page of the SGM website. Why not enter by submitting an offered poster or oral presentation at the spring meeting in Bath next year? The closing date for abstracts is **28 November**.

## Postgraduate Student Membership

Postgraduate Student Membership of SGM is available to postgraduate students worldwide who have no taxable income. For an annual subscription of only £20 (US$35) Student Members can take advantage of benefits such as special registration fees at Society meetings and the purchase of SGM publications at greatly discounted prices. In addition, eligible Postgraduate Student Members may apply for awards from the President's Fund and Postgraduate Conference grants (see p. 177 for details) which provide for attendance at scientific meetings.

## Undergraduate Membership

Undergraduate Membership is open to students resident and registered for a first degree in the UK or Republic of Ireland. For £10 Undergraduate Members receive *Microbiology Today* and may attend SGM meetings without payment of a registration fee. Careers advice is also freely available. However, Undergraduate Members are not eligible for travel or conference grants.

## Life Science Careers 2003

- 1 November 2003 — King's College, London
- 15 November 2003 — UMIST
- 29 November 2003 — University of Wales Cardiff

These all day conferences are for life science undergraduate (graduating in 2004 or 2005) and postgraduate students. Each conference includes a talks on career choices and further training, an exhibition and a CV clinic. Don't miss the nearest event to your institution – further information and a booking form are at **www.bsf.ac.uk/careers**

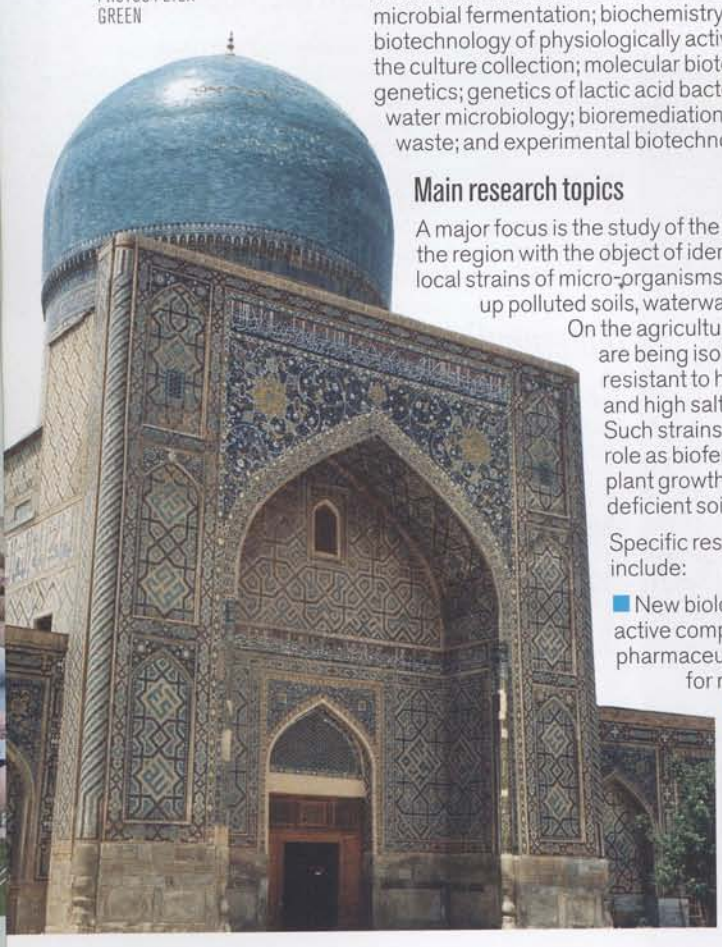
# International Development Fund report

SGM helps microbiologists in developing countries through this fund, usually by supporting training courses and other small technology transfer projects. The full rules of the fund appear on the grants page of the SGM website at **www.sgm.ac.uk** The closing date for applications is in October each year.

TOP RIGHT:
Staff from the Uzbek Institute of Microbiology, with Peter Green at the back.

BELOW:
A holy temple in the city of Samerkand, Uzbekistan.

PHOTOS PETER GREEN



## Culture Collection Management in Uzbekistan

■ Kakhramon Davranov & Dilfuza Egamberdiyeva

### Institute history

The Institute of Microbiology of the Uzbek Academy of Sciences based in Tashkent, is the only organization in Uzbekistan specializing in theoretical and applied problems of microbiology and microbial biotechnology. Microbiology was first practised as a discipline in the Academy in 1943. The first microbiological department was founded at the Institute of Zoology and Botany in 1947. It specialized in virology, water and soil microbiology.

Microbiology gained independent status as a subject in 1965, which was considered to be a major event in the development of this science in Uzbekistan. The microbiology elements then taught were microbial physiology, biochemistry, genetics and yeast taxonomy. In 1977 the department gained institutional status and became the Institute of Microbiology of Uzbekistan.

Within the Institute there are 10 laboratories: microbial fermentation; biochemistry and biotechnology of physiologically active compounds; the culture collection; molecular biotechnology and genetics; genetics of lactic acid bacteria; soil and water microbiology; bioremediation of plant waste; and experimental biotechnology.

### Main research topics

A major focus is the study of the biodiversity of the region with the object of identifying unique local strains of micro-organisms which will clean up polluted soils, waterways and effluents. On the agricultural side, strains are being isolated which are resistant to high temperatures and high salt concentrations. Such strains have an important role as biofertilizers, promoting plant growth in arid, nitrogen-deficient soils.

Specific research areas include:

- New biologically active compounds and pharmaceutical preparations for medical use.
- Valuable feed additives and microbially active compounds for the food and pharmaceutical sectors, e.g. feed proteins, vitamins, amino acids, growth regulators, veterinary preparations, etc.
- Biological control formulations to protect plants from different diseases, insects and pests.
- Ecologically clean bio-fertilizers.
- New technologies for use in food, chemical, microbiological, mining and oil industries.
- Processing and bioremediation of agricultural, household and industrial wastes, sewage and greenhouse gases for biogas production.

### The culture collection

The National Collection of Agricultural and Industrially Important Strains is key to many of the above activities and the loss and poor management of many important cultures has been a major concern. On-going research to solve fundamental problems relating to the physiology, biochemistry and genetics of bacteria, viruses, actinomycetes, yeast and microscopic fungi was being severely hampered without such a reliable genetic resource.

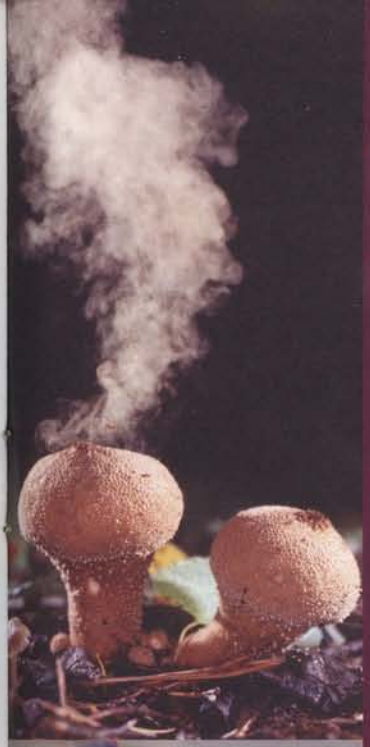
### Problems and challenges

One of the main issues facing our Institute was a lack of equipment for preserving valuable micro-organisms. Due to this, many industrially important bacterial and fungal strains were lost. However, recently we received financial support from the SGM, which at last is allowing us to safeguard these valuable cultures. Now we can maintain all industrially and agriculturally important organisms frozen at –80 °C. The grant will also contribute towards labour and consumables.

The visit of Dr Peter Green to administer the SGM grant was a very significant experience in the history of our institute. We learned new approaches to the preservation and maintenance of micro-organisms and about the management of a culture collection. We were also able to exchange ideas for future collaborative projects.

In summary this grant will help us to plan for the future on a more sound footing and to have collaborative projects with developed countries such as the UK. We wish to thank the SGM most sincerely for the grant from the International Development Fund and we are most grateful to Dr Peter Green of NCIMB for his visit to our Institute and for his lecture on culture collection management and bacterial preservation.

■ ***Professor Kakhramon Davranov, Director, Institute of Microbiology, Uzbekistan, and Dr Dilfuza Egamberdiyeva, Curator of the National Culture Collection of Agricultural Micro-organisms, Tashkent.***
***email dilfuza_egamberdiyeva@yahoo.com***



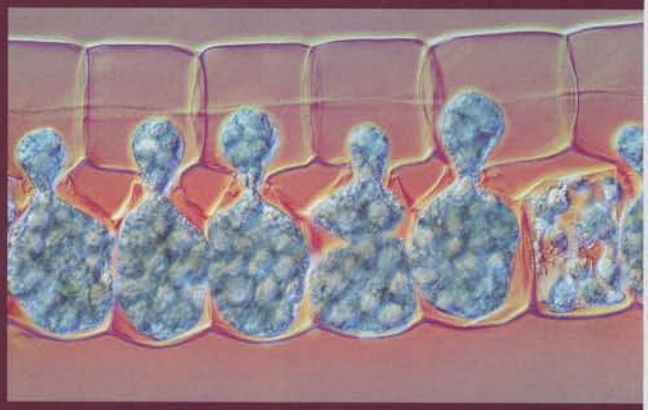
# The Novartis and *The Daily Telegraph* Visions of Science Photographic Awards 2003

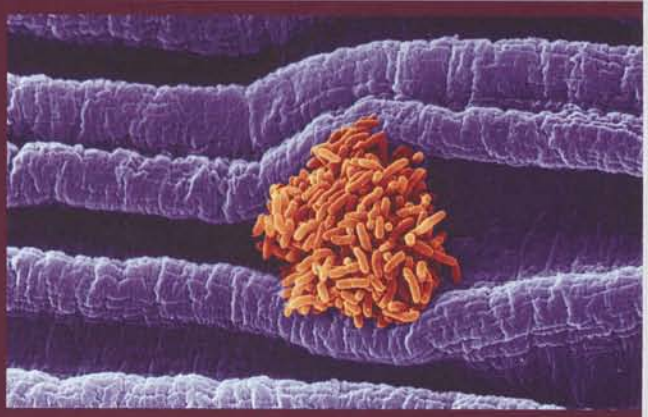
Now in its fourth year, the competition is organized as a means of encouraging discussion about science in the UK. The images are intended to showcase the very best in scientific photography. In 2003 the Awards attracted 1,400 entries from professional photographers, research scientists, civil servants, journalists and students.

The panel of judges, headed up by Adam Hart-Davis, himself a photographer as well as television presenter, had a difficult task in selecting the winners of the 8 categories. They defined a 'vision of science' as an eye-grabbing picture that gives new insight into the working of nature. Despite agreeing on the basic criteria, they argued fiercely about which of the entries were arresting, original and visionary. There were also some concerns about computer-generated pictures, but all concurred that many of the stunning images would not have been possible without the use of Photoshop and other technical aids.

The awards were announced by Adam at a ceremony at the Royal Society on 23 September and the winners received their certificates and prizes from Lord May, President of that institution. Pleasingly, many of the shortlisted and winning entries had a microbiological association. Some of the winning and shortlisted pictures are reproduced here with kind permission of the organizers of the awards.

● *Janet Hurst*





THE NOVARTIS & THE DAILY TELEGRAPH
Visions of SCIENCE
PHOTOGRAPHIC AWARDS

LEFT TOP:
Puffball fungus emitting spores.
LES CLARKE – 'ACTION' CATEGORY HIGHLY COMMENDED

LEFT MIDDLE:
Imperfections and assymetry revealed in a diatom at high magnification.
PROFESSOR DAVID G. MANN – 'ART' CATEGORY HIGHLY COMMENDED

LEFT BOTTOM:
Radial gills on the underside of a fungus.
TOMMY GA KEN WAN – YOUNG PHOTOGRAPHER AWARD (17–18 YEARS) HIGHLY COMMENDED

ABOVE TOP:
Two threads (male below; female above) of the green alga *Spirogyra* during conjugation.
PROFESSOR DAVID G. MANN – 'CLOSE-UP' CATEGORY 1ST PRIZE

ABOVE BOTTOM:
A colony of *Pseudomonas fluorescens* on nutrient agar.
PAUL GUNNING – 'CLOSE-UP' CATEGORY SHORTLISTED

CENTRE TOP:
A patient at the Mutemwa Leprosy Settlement, Zimbabwe.
SIMON ROBERTS – 'PEOPLE' CATEGORY 2ND PRIZE

CENTRE BOTTOM:
Decaying figs.
ERIKA CRADDOCK – 'ART' CATEGORY 2ND PRIZE

ALL IMAGES COURTESY THE NOVARTIS AND *THE DAILY TELEGRAPH* VISIONS OF SCIENCE AWARDS 2003

# Obituaries

## David Kelly

### 17 May 1944–18 July 2003

David Kelly's tragic death at the age of 59 has illustrated all too readily the problems that arise when a scientist, seeking to retain professional integrity, becomes caught up in establishment power politics.

David was born in Wales and took his first degree at Leeds. He decided to specialize in virology and completed the MSc course organized by Peter Wildy at Birmingham before moving to the NERC Unit of Invertebrate Virology at Oxford to undertake postgraduate research with Tom Tinsley on insect iridescent viruses.

After graduating with a DPhil in 1971 he did a post-doctoral project with Nigel Dimmock at Warwick on influenza virus before returning to the Oxford Unit as a staff member in 1974 to continue his interests in insect viruses. The next few years were very productive and he published a series of important papers on the molecular properties of a number of members of the Iridoviridae and the baculoviruses.

In 1984 he was attracted to the Chemical and Biological Defence Establishment at Porton Down, eventually becoming the Head of Microbiology. One of his first major tasks was to oversee the decontamination of Gruinard Island, the site of war-time experiments on anthrax. During this time he also utilized the category 4 containment facilities to carry out valuable research on the molecular and antigenic properties of simian herpes B virus (which is endemic in monkeys and almost always fatal in humans).

RIGHT:
Dr David Kelly.
COURTESY MRS J. KELLY & CHRIS PAYNE (UNIVERSITY OF READING)

His first venture onto the international scene occurred in October 1989 when he was called on to help in the debriefing of Vladamir Pasechnik, a Soviet defector. This revealed the massive extent of the Soviet bioweapons (B/W) programme and, as a consequence, in 1991 he co-led a UK/US delegation to inspect Soviet biowarfare institutes. It was during this visit that, largely through David's probing and virological expertise, it became evident that there was an ongoing programme of smallpox research and development at Novosibirsk in Siberia – contrary to the international agreement on the cessation of all work on smallpox.

His expertise in the detection of bioweapons was, by now, internationally established and this was recognized by the UK government with the award of the CMG. He played a leading role in the UNSCOM team which uncovered the five bioweapons facilities in Iraq in 1994 and he supervised their dismantling. In all, he made over 36 visits to Iraq and was preparing for another visit a few days before his death. His colleagues in the UNSCOM team described David as '*quiet, persistent, well-informed and scientifically indomitable*'. It was typical of David that, in spite of his travails there, he came to love Iraq and admired the Iraquis (see *Plague Wars* by Tom Mangold and Jeff Goldberg).

There is no doubt that the UK has lost a formidable scientist – one of the very few microbiologists who had real expertise in bioweapons. His professional colleagues will miss his courtesy, his quiet humour, his ability to make friends easily and his wide knowledge of the more practical aspects of virology. Among his many enthusiasms was a passion for Welsh rugby, a keen support for Leeds United and an ability to play the saxophone with some verve. We grieve with his wife, Jan and daughters Sian, Ellen and Rachel on the sudden and tragic loss of an exceptional husband and father.

● *Willie Russell, University of St. Andrews, UK.*
*The author was the external examiner for David's DPhil and was the Convener of the Virus Group Committee from 1984 to 1989 when David was a valued member of the committee. He is grateful for information from Richard Elliott who was his first research student at Oxford and Chris Payne who was a colleague of his there in the late 1960s and early 70s.*

# Sir Robert Williams

## 30 June 1916–24 May 2003

Sir Robert (R.E.O.) Williams was one of the outstanding figures in medical microbiology, public health and medical education of the last half century. He combined scientific work of the highest calibre with outstanding administrative achievements and won respect and admiration for both.

Born in London in 1916 into a medical family (his father, Gwyn, was a consultant surgeon and, for a period, Dean at University College Hospital, London), he did his medical studies and a degree in physiology at University College and University College Hospital, London, qualifying in 1940. After a house physician post at the same hospital, he joined the emergency medical service as a pathologist, in fact a microbiologist, and worked with the Medical Research Council's Infection Unit at the Birmingham Accident Hospital under Professor (later Sir) Ashley Miles, an inspirational influence on a young microbiologist. It was in Birmingham that Williams began his ground-breaking studies on the sources and spread of wound infections and hospital cross-infection, work that is as relevant today as it was in the 1940s and 50s. He concentrated initially on infections of wounds of the hands, which were common in the light engineering factories in Birmingham.

A second major influence on Williams, G.S. (later Sir Graham) Wilson, the visionary founder and first Director of the Public Health Laboratory Service, had recently devised a method for differentiating strains of *Staphylococcus aureus*, the commonest wound-infecting bacterium, based upon their susceptibilities to bacteriophages. Williams defined and elaborated this 'phage typing scheme and applied it to the study of sources and routes of transmission of this major wound pathogen. He published the 'phage typing method in 1952 and, using this technique, he showed how wounds could be 'self-colonized' by *S. aureus* from the patient's own nose or skin. He also showed that infections with group A streptococci usually originated outside the patient. He was one of the first to recognize the practical importance of high-resolution typing to identify the sources and links between different cases of infection, in his case wound infections. This remains a major function of the reference laboratories that became such an important part of his life. Our understanding of the worldwide epidemics of staphylococcal wound infection of the 1950s and 1960s was almost entirely due to the strain discrimination provided by 'phage typing.

In 1946, Williams had moved to the Central Public Health Laboratory at Colindale, North London, to the Streptococcal, Staphylococcal and Air Hygiene laboratory and he became Director of that laboratory in 1949. His work on wound contamination covered the varied contributions of direct contact, large falling droplets and air-suspended droplets, and showed many of the environmental factors that affected these routes of transmission in different hospital settings.

By now recognized as an international authority, he provided simple guidance on how to prevent wound infections and how to treat them and, in 1960, he was one of the authors of the first classical book on hospital-acquired infections, which remains as highly relevant for today's infection control teams as it was then to those of us who would follow in his footsteps.

In 1960 he was lured back into clinical academic medicine as Professor of Microbiology at St Mary's Hospital Medical School. He was the first full-time academic Dean of St Mary's from 1967 to 1973, bringing the innovative approach of his bacteriological research to the running of a medical school. He was also a member of the Medical Research Council from 1969 to 1973. 1973 marked the last major phase of his professional life when he returned to the PHLS at Colindale as Director of the Service, with its central reference laboratories and network of regional and area laboratories, based in hospitals where they combined diagnostic and epidemiological work. During the same period, he was elected President of the Royal College of Pathologists (1975–78) and in a pioneering move for a medical Royal College, he guided the extension of membership to include medical scientists who were not medically qualified. He was knighted in 1976.

Under his direction, the PHLS enlarged its responsibilities with the establishment of the Communicable Disease Surveillance Centre in 1977 and then the addition of the Centre for Applied Microbiology and Research, Porton Down, in 1979. Before he retired in 1981, he was closely involved in planning and designing the new institute at Colindale to house both CPHL and CDSC. This was his physical legacy to microbiology and public health services, but his real legacy to medicine and science has formed the basis of many of the developments of the last 20 years. He was one of the classical tradition of doctors who were also important medical scientists. Throughout, he remained an unassuming and approachable man with a good sense of humour. He was always available and his advice was generously given and always supportive.

In 1944, he married Margaret Lumsden; she died in 1990. They are survived by a son and two daughters, one of whom followed her father into microbiology and works for the World Health Organization.

● *Brian I. Duerden, Professor of Medical Microbiology, University of Wales College of Medicine; lately, Director, Public Health Laboratory Service*

ABOVE:
Sir Robert at the building of the PHLS laboratories in 1980/81.
COURTESY HEALTH PROTECTION AGENCY, COLINDALE

# Hot off the Press

Science writer Meriel Jones takes a look at some papers in current issues of the Society's journals which highlight new and exciting developments in microbiological research.

## HME – an emerging human disease

Human monocytic ehrlichiosis (HME) is an emerging disease in the USA. Although only a few hundred cases have been reported, there are probably many more, because the symptoms are similar to several other diseases. Patients may have a fever, headache, muscle aches and feel tired. About half have a rash. A course of antibiotics clears up the problem in most people, although the illness can occasionally develop life-threatening complications. The disease is caused by *Ehrlichia chaffeensis*, small Gram-negative bacteria that are unable to live outside the cells of their animal hosts. They probably live in blood cells in wild deer, transmitted between animals by the lone star tick if it picks up the bacterium in its meals of blood. The danger to humans is during walks in woodland when ticks can bite them.

Researchers at the Center for Biodefense and Emerging Infectious Diseases, and the Animal Resource Center of the University of Texas Medical Branch, have been learning more about the disease. They knew of reports that dogs have been naturally infected by *E. chaffeensis*, and wanted to discover more about this. One fear is that domestic pets could become a reservoir for the disease, bringing it into closer contact with more humans. Studying dogs that had been experimentally infected would also give a better idea of any disease symptoms and how long the infection persisted.

With the approval of their University's Institutional Care and Animal Use Committee, they designed an experiment in which two beagle dogs were infected with *E. chaffeensis*, while two others acted as uninfected controls. They collected regular blood samples over 6 months to test for bacteria. When the researchers found signs of bacteria, they decided to investigate the structure of one of the bacterial surface proteins. Several bacterial species that set up persistent infections have a mechanism to change surface features frequently, to continually evade the immune system. *E. chaffeensis* looked as if it should have this system, and the researchers wanted to check on it.

Although the infected dogs had decreased blood platelet counts, neither of them ran a fever or lost weight. The researchers started detecting bacteria in the dog's blood 23 days after infection, and continued to find evidence of *E. chaffeensis* for 2·5 to 3·5 months. Both dogs developed antibodies to the bacteria, and when the researchers looked at their results for the surface proteins, it was clear that these did not change during the course of the infection. The study indicates that dogs can harbour an infection by *E. chaffeensis* for up to 4 months without showing any symptoms of illness. As well as adding to public health information about HME, this gives the researchers an opportunity to study the infection in more detail.

**Zhang, X.-F., Zhang, J.-Z., Long, S. W., Ruble, R. P. & Yu, X.-J. (2003).** Experimental *Ehrlichia chaffeensis* infection in beagles. *J Med Microbiol* 52, 1021–1026.

## *West Nile virus* in the UK?

Researchers at the Centres for Ecology and Hydrology at Oxford and Monkswood in the UK have been checking on the immunological status of wild birds to arboviruses in the UK. *West Nile virus* (WNV) causes fever and inflammation of the brain which may be fatal, while other arboviruses, *Usutu virus* and *Sindbis virus*, rarely cause serious disease. WNV was first identified in Uganda in 1937 and is found throughout northern Africa, southern Europe, much of Asia and Australia. It recently hit the headlines through killing more than 300 people in the USA, and also many wild birds and other animals since emerging in 1999. Previously, outbreaks in Europe, the Middle East and Russia have affected birds, animals and many human fatalities have been recorded.

The natural life cycle of all three viruses involves migratory birds and the mosquitoes that feed on them. The mosquitoes transmit the virus between the birds, and can pass it on to animals and non-migratory birds. The risks of infection are greatest when large numbers of mosquitoes, people and birds are found together, especially in towns near lakes or slow moving rivers. The recent experience in North America has indicated how dangerous WNV can be when it encounters bird or animal or human populations that have never been exposed to it before. The researchers knew that there is no evidence that any of these viruses is present in the UK, although for thousands of years birds have migrated to Britain from regions of Africa where the viruses are found. In a country with so many keen bird-watchers, the sudden death of wild birds on the same scale as in the USA would be rapidly detected. There have also been no reports of illness in UK citizens that can be attributed to these mosquito-borne infections. However, the researchers decided to survey birds in Britain to see whether the virus was really absent.

They used very sensitive tests to search for both the virus and any evidence that birds had been exposed to the virus. Exposure to any pathogen normally causes the immune system of the body to try to produce a series of defences against it, including antibodies that persist for many years. The researchers tested serum from birds that had been trapped in mist nets and then released, as well as

OPPOSITE PAGE (UPPER): Transmission electron micrograph of *Borrelia* sp. from *H. aegyptium*. Five insertion points of flagella were observed in the tapered ends. COURTESY T. MASUZAWA, SHIZUOKA, JAPAN

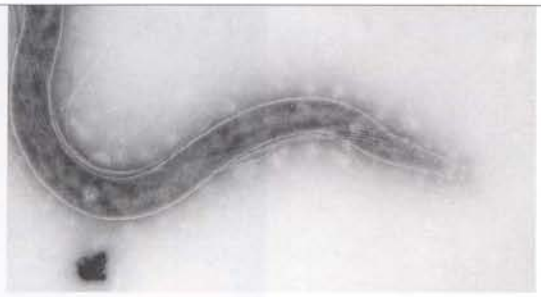
OPPOSITE PAGE (LOWER): An adult hard tick, *Hyalomma aegyptium* (adult), from an infested tortoise. The diameter of the hole in the 5 yen coin is 5 mm across. COURTESY T. MASUZAWA, SHIZUOKA, JAPAN

from free-range poultry. In total, they had over 430 samples from 30 species of birds collected in Cambridgeshire, Dorset and South Wales. These included house martins, swallows and several species of warblers that had migrated from Africa, as well as others, particularly juvenile birds and free-range poultry, that had never left the UK. In addition, they tested the brains of carrion crows and magpies that had been killed during pest control programmes, for the presence of WNV RNA.

The researchers found signs that all three viruses had been introduced into UK-resident birds from migrating birds. About a quarter of the sera from apparently healthy resident birds had antibodies to WNV. This difference from the North American experience suggests that either UK birds have managed to build up immunity over the years, or the virus is less virulent than that circulating in the USA. The authors conclude that since there are few mosquitoes in the UK compared with warmer countries like North America and Southern Europe, the risk to human health is probably low at the moment. However, they think that if the climate becomes warmer, the risk may increase. They intend to extend their study to more birds, insects and animals in the UK, to assess the situation more thoroughly.

**Buckley, A., Dawson, A., Moss, S. R., Hinsley, S. A., Bellamy, P. E. & Gould, E. A. (2003).** Serological evidence of *West Nile virus*, *Usutu virus* and *Sindbis virus* infection of birds in the UK. *J Gen Virol* **84**, 2807–2817.

PHOTO IAN ATHERTON, SMI



## A new fast-growing species of *Borrelia*

Bacteria of the genus *Borrelia* cause diseases in humans called Lyme disease (see pp. 165–166) and relapsing fever (see p. 167), as well as illnesses in animals, including epizootic bovine abortion and fevers. Ticks, and their relatives, transmit the bacteria between hosts, which include animals and humans. As people and wild animals are brought closer together by changes in climate and agriculture, as well as by tourism and cities encroaching onto previously uninhabited land, there are more opportunities for people to pick up previously rare diseases. Microbiologists are therefore keen to learn more about both the bacteria and the ticks that transmit them.

Researchers at Yeditepe University in Turkey, and Aichi Medical University and the University of Shizuoka in Japan, have collaborated to study a novel species of *Borrelia* that is carried by tortoises. They collected 150 tortoises from woodland and scrub near the coast, 80 km from Istanbul. Almost all of the tortoises had hard ticks (*Hyalomma aegyptium*) on them. As the ticks went through their life-cycle from larvae to nymphs to adults by the end of summer, the average number on each animal decreased from around 15 to 4. Around 40 % of the adult and nymphal ticks were infected with the novel bacterium, which was absent from the larvae.

*Borrelia* are spirochaete bacteria. The cells of this new species were slender, corkscrew-shaped and swam vigorously using flagella attached to the ends of the cells. The researchers sequenced part of a gene for a protein that makes up the flagella. When they compared it with the same gene from other *Borrelia* species, the bacteria isolated from the tortoises formed a separate group. However, the feature that dramatically distinguished this species from other *Borrelia* species was its rapid growth. The cells doubled in number in about 5 hours, about twice as fast as all previously known *Borrelia* species. The researchers want to investigate the reason for this rapid growth, as well as determining whether this new species poses any risks to the health of humans or domestic animals.

**Güner, E. S., Hashimoto, N., Kadosaka, T., Imai, Y. & Masuzawa, T. (2003).** A novel, fast-growing *Borrelia* sp. isolated from the hard tick *Hyalomma aegyptium* in Turkey. *Microbiology* **149**, 2539–2544.

## A new threat from bats

Scientists from the American Centers for Disease Control and Prevention have been collaborating with colleagues in the Ministry of Health in Cambodia to assess the public health risk from bats. These are the only flying mammals, and can harbour several viruses, including rabies. The scientists tested *Chaerephon plicata* bats from Kampot Cave in southern Cambodia, where local villagers had reported that the number of bats in the colony was declining. Apart from being a tourist destination, the cave is an important source of fertilizer from the bat guano, so there were considerable opportunities for contact between the bats and people. If the bats were suffering from a disease that could be transmitted to people, there could be a real problem.

The researchers obtained materials from both healthy and dead bats, and began searching for viruses. To find out whether the dead bats had died of anything that could be passed on to other mammals, the investigators injected extracts from the bats' brains into young mice. A substantial number of the mice died, supporting the idea that the bats had died from disease rather than another cause. The researchers used electron microscopy to look for virus particles, and found some that looked like members of the virus family Bunyaviridae. There was one report of Kaeng Khoi virus (KK), a member of this family, in the same species of bat from Thailand around 1970. When the researchers used molecular biological methods on their samples, they identified the presence of KK virus.

The researchers now have good evidence that KK virus is present across hundreds of kilometres of southeast Asia, but they are unsure of its effect on local bat populations, or human health. Without further observations it is impossible to know if KK virus has caused a decline in the numbers of bats in the Kampot caves. And, although in an earlier study almost a third of bat guano collectors had been shown to possess antibodies to KK virus, none of them associated serious illness with the caves. However, arthropods are often the vectors that transmit bunyaviruses between hosts, and the previous researchers had isolated KK virus from bedbugs within the caves in Thailand. The locals reported that bedbug bites could cause an influenza-like illness, which is a typical symptom of infection by several family Bunyaviridae viruses. Further studies to determine the virus effect on the bats, if this virus is associated with human disease, and potential for spread are warranted.

**Osborne, J. C., Rupprecht, C. E., Olson, J. G., Ksiazek, T. G., Rollin, P. E., Niezgoda, M., Goldsmith, C. S., An, U. S. & Nichol, S. T. (2003).** Isolation of Kaeng Khoi virus from dead *Chaerephon plicata* bats in Cambodia. *J Gen Virol* **84**, 2685–2689.

## Rabbit, rabbit!

Rabbit numbers are a very serious problem in Australia and New Zealand. After a new, highly contagious and virulent viral disease of rabbits had emerged from China in 1984 and spread to the rest of Asia and Europe, the Australian government approved trials of its use as a bio-control agent. The rabbit haemorrhagic disease virus (RHDV) can kill susceptible adult rabbits in hours from massive internal bleeding. Trials were held on an offshore island, so that the virus could be contained if problems arose. However, the virus escaped to the mainland, possibly with human help, where it spread, at least initially, as a phenomenally fast and lethal epidemic throughout the southern Australian states. The New Zealand Government rejected the idea of using RHDV, but the virus was deliberately and illegally introduced into New Zealand in 1997. Many farmers set out baits of oats and carrots contaminated with RHDV, initiating the first epidemics of the disease, although with only 10–50 % mortality. After the disease was established, the New Zealand Government changed its mind and approved importation, manufacture and sale.

However, by 2001 there were many healthy rabbits in areas of New Zealand where the virus was released. In Australia as well, the disease has become much less lethal. Researchers in the UK have been trying to work out how this has happened. Although people had initially assumed that New Zealand was free of RHDV before the illegal releases, re-analysis of stored serum samples has shown that a RHDV-like virus was already present. Since there were no records of disease symptoms, it must have been present as a subclinical infection. One implication is that rabbits with immunity to this virus may also have been able to resist RHDV, explaining the variable mortality in the first epidemics in 1997.

The researchers checked for RHDV in healthy rabbits shot near Alexandra in Central Otago. This was among the first places to report the disease following release of the virus in 1997. They used an extremely sensitive version of the polymerase chain reaction to find out the sequence of one of the most variable regions of the viral genome. They detected very small amounts of two types of the virus in about half of the rabbits. When the researchers tried to infect healthy, virus-free rabbits with extracts from these rabbits, they were completely unsuccessful, indicating that although the viral RNA was present, there was insufficient to cause a new infection at least under experimental conditions.

Most of the infected rabbits contained a version of the virus that was very closely related to the Czech V351 strain that was deliberately introduced in 1997. All but one of the remaining positive rabbits had a very similar, but distinctly different version, corresponding to the virus introduced illegally by the farmers. The biggest surprise was a third version in one rabbit, most closely related to a type of RHDV isolated in Spain. After carrying out experiments to check this result, the researchers were convinced that this was real, but were left without an explanation of how it had got to New Zealand. The presence, in healthy rabbits, of similar viral sequences to the strain responsible for the first epidemics indicated that the virus has become less able to cause a lethal infection. The big question, which the researchers are now attempting to answer, is how has RHDV become able to cause these persistent, non-lethal infections, and how can it be awakened back to a virulent infection.

**Forrester, N. L., Boag, B., Moss, S. R., Turner, S. L., Trout, R. C., White, P. J., Hudson, P. J. & Gould, E. A. (2003).** Long-term survival of New Zealand rabbit haemorrhagic disease virus RNA in wild rabbits, revealed by RT-PCR and phylogenetic analysis. *J Gen Virol* **84**, 3079–3086.

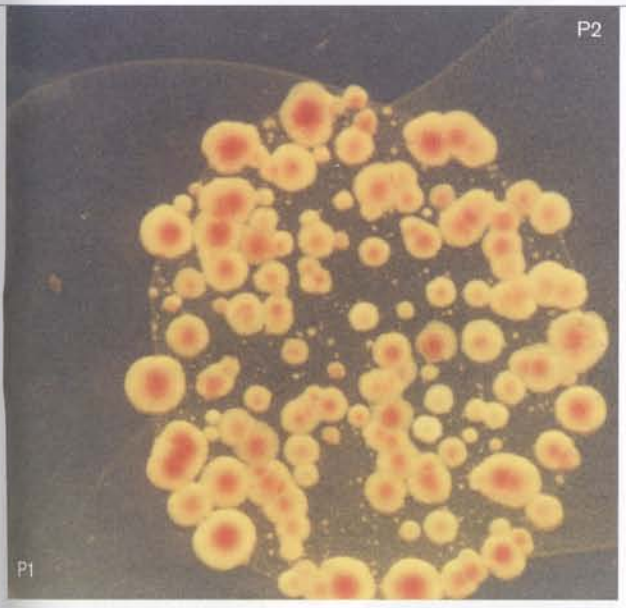


## New member of the CF lung flora

Cystic fibrosis causes many life-threatening problems, including repeated infections of the lungs. The genetic change that causes the disease makes the respiratory tract a much more attractive ecological niche for a wide variety of bacteria. Bacterial species such as *Pseudomonas aeruginosa* and *Burkholderia cepacia* frequently set up persistent infections. Researchers have found that a number of unusual species of bacteria can also take advantage of the situation. Scientists from the University of Michigan have collaborated with Peter Vandamme of the University of Gent in Belgium to study some of the less well-known species.

They focused on five strains that appeared to be members of the genus *Ralstonia*, isolated from patients receiving treatment in three US states. After carrying out a thorough investigation of the media on which the bacteria could grow, their cellular fatty acids, the sequence of one of their genes and a profile of their proteins, the researchers concluded that the strains originated from two previously unknown species. They have named one *Ralstonia respiraculi* (after the Latin noun *respiraculum* respiration). However, they are waiting until they discover more strains of the other one before giving it an official name.

**Coenye, T., Vandamme, P. & LiPuma, J. J. (2003).** *Ralstonia respiraculi* sp. nov., isolated from the respiratory tract of cystic fibrosis patients. *Int J Syst Evol Microbiol* **53** 1339–1342.




LEFT:
Mating between strains marked by complementary auxotrophic mutations. Colonies are formed on minimal agar in the zone where the dropped cultures P1 and P2 overlap.
COURTESY J.-P. GRATIA, PASTEUR INSTITUTE OF BRUSSELS, BELGIUM

## Real sex in bacteria!

Among the first things that students are taught about bacteria is that they do not indulge in real sex. Bacterial cells are haploid, with only one copy of every gene. They have several ways to pick up bits of DNA from the environment, and to transfer it into other bacterial cells, but the result is never a diploid cell with two copies of every gene like the average animal cell. Up until the 1950s bacteriologists speculated that sexual reproduction might be waiting to be discovered in bacteria. Since then, apart from a few scattered reports, most people quietly dropped the idea.

However, in 1994 Jean-Pierre Gratia from the Pasteur Institute in Brussels found something that appears to be closer to true sexuality in bacteria than has ever been seen before. He isolated a strain of *Escherichia coli* with very surprising characteristics. At the time, the best explanation was that each cell contained two entire sets of *E. coli* genes, only one of which was in use at any time. Now he and his colleague Marc Thiry from the University of Liege in Belgium have been following up this discovery, applying new techniques in molecular biology and cell imaging to gain a more detailed understanding of this phenomenon. They call it 'spontaneous zygogenesis', or Z-mating for short.

The researchers performed crosses between strains carrying genetic markers for antibiotic resistance or requirements for particular amino acids in the growth medium. The results were much more like those from diploid cells, where the progeny contain all the genetic markers. However, one other possibility that would explain the results would be that the *E. coli* cells had become particularly sticky. To check on this, the researchers used several sorts of microscopy to look at both the arrangement of the cells, and also the DNA within them. They could use minute gold particles to label the DNA from each of the parental strains in their crosses, and then examine the labelling of the progeny.

Putting together all their results, the researchers feel that they have convincingly demonstrated the existence of a transmissible Z-factor in *E. coli* that gives the cells an ability to fuse and exchange genetic information in a way that has never been observed before. They are sure that this new process opens up exciting prospects for both fundamental research and biotechnology.

**Gratia, J.-P. & Thiry, M. (2003).** Spontaneous zygogenesis in *Escherichia coli*, a form of true sexuality in prokaryotes. *Microbiology* 149, 2571–2584.

## Cleaning up the environment

Chlorinated hydrocarbons are very useful man-made chemicals. Their major advantages are that they do not dissolve in water and they are resistant to degradation by microbes. They are essential industrial solvents and can be found in brake fluid in cars and in dry-cleaning liquid. However, their chemical nature means that they are toxic and must be handled with care. Nevertheless, their usefulness means that large volumes are manufactured.

These advantages turn into a problem when the chemicals are spilt or reach the end of their useful life and need to be disposed of. Although many bacteria attempt to use chlorinated hydrocarbons as a source of food, degradation is slow. The chemicals can remain in the soil for decades and seep into underground rock to contaminate water supplies. A substantial portion remains resolutely inaccessible, perhaps adsorbed onto soil particles or sequestered within pores in the soil minerals. The only way to encourage microbes to get at this so-called 'aged' pollutant is to spray the ground with expensive detergents. Digging up all the soil from a contaminated site and burning it in a furnace at a very high temperature is sometimes another costly solution. It is no surprise, therefore, that biotechnologists continue to look for more environmentally friendly ways to clean up 'aged' chlorinated hydrocarbons.

Although many Gram-negative bacteria can digest chlorinated hydrocarbons, this ability is rare among Gram-positives. Many Gram-positives, however, can degrade non-chlorinated hydrocarbons, and some produce a detergent during the process. A bacterial species that combines both abilities could be exactly right for bioremediation. In the late 1990s Peter Rapp and Lotte Gabriel-Jürgens found a contaminant in a culture of Gram-negative bacteria that looked like it might have these properties. They have now described the remarkable abilities of *Rhodococcus* sp. strain MS11 in a recent issue of *Microbiology*.

Not only could this aerobic, Gram-positive, non-motile bacterial strain degrade an unexpectedly wide range of chlorinated and non-chlorinated hydrocarbons, but it made its own detergent as well. It could grow on 1,2,4,5-tetrachlorobenzene, as well as versions of this chemical containing only two or three chlorine atoms. This is a rare ability, even among Gram-negative bacteria, so the researchers checked out the genes that provided this characteristic. They turned out to be surprisingly similar to those in the Gram-negative bacterial culture from which the *Rhodococcus* strain had been isolated as a contaminant. The researchers suspect that when the contaminant had been in the life-or-death situation of needing to use a novel chemical as a source of food and energy in order to live, it had acquired exactly the right genes from the culture. In combination with the innate ability of *Rhodococcus* sp. strain MS11 to synthesize a detergent, the researchers have found an organism that might be well suited for bioremediation of sites polluted with industrial chemicals.

**Rapp, P. & Gabriel-Jürgens, L. H. E. (2003).** Degradation of alkanes and highly chlorinated benzenes, and production of biosurfactants, by a psychrophilic *Rhodococcus* sp. and genetic characterization of its chlorobenzene dioxygenase. *Microbiology* 149, 2879–2890.

# Reviews

If you would like your name to be added to our database of book reviewers, please complete the book reviewer interests form now available on the SGM website.

A classified compendium of book reviews from 1996 to the present is also available on the website.

A list of publishers' website addresses is given on p. 195.

## Pioneers of Microbiology and the Nobel Prize

By U. Lagerkvist
Published by World Scientific (2003)
HB US$48.00/£33.00;
PB US$24.00/£16.00, pp. 182
ISBN: 981-238-233-X (HB);
981-238-234-8 (PB)

Just over 100 years ago the first Nobel Prize in Physiology or Medicine was awarded. This book is an authoritative and entertaining account of four of the early winners. The life and work of Robert Koch, Emil von Behring, Paul Ehrlich and Elie Metchnikoff are closely examined in the light of contemporary scientific knowledge and ignorance. Politics appear to have had as much to do with their final acceptance as Nobel Prize winners as did their outstanding scientific achievements. Many full-page portraits add to the charm of this book. Members of the public wanting to learn more about the birth of microbiology will find this book invaluable. Trainee as well as experienced microbiologists and immunologists will also find much of interest. There should be a copy in the often neglected history section of all good microbiology libraries.

■ ***Paul Wright***
***Conquest Hospital, St Leonard's-on-Sea***

## Vaccines for the 21st Century: A Tool for Decisionmaking

Edited by K.R. Stratton, J.S. Durch & R.S. Lawrence
Published by National Academy Press (2001)
£30.95, pp. 460
ISBN: 0-309-05646-2

Vaccines, both prophylactic (for infections) and therapeutic (for diseases such as diabetes, rheumatoid arthritis, multiple sclerosis and melanoma) are reviewed in terms of economic priority for development and implementation. The introductory chapters cover some of the background to economic evaluations, which is then demonstrated using a hypothetical vaccine. Each disease is evaluated in terms of the economic burden, morbidity and mortality, target populations for vaccination and costs of delivery and they are considered against the likely benefits of disease prevention using 'disease scenarios'. The priority list produced in 1985 is virtually unchanged now. It would have been interesting to have an analysis of the economics issues surrounding the development and strategies for the implementation of an HIV vaccine, but the only reference to HIV was as a confounding factor in many other infections. The subtitle is the key – a tool for decision making – it should be compulsory reading for all who are involved in decision making both at the vaccine development and implementation stages. The individual reader will, however, find it interesting to browse particularly the workshops on the mechanism of actions of some vaccines.

■ ***Liz Boxall***
***Public Health Laboratory, Birmingham Heartlands Hospital and National Blood Service, Birmingham***

## Tumor-Suppressing Viruses, Genes and Drugs. Innovative Cancer Therapy Approaches

Edited by H. Maruta
Published by Academic Press (2001)
£66.95, pp. 400
ISBN: 0-12-476249-2

The title is wide-ranging. So is the book. This volume reviews the gamut of novel anti-cancer therapies that are currently in clinical trial, under development or merely being considered. At least that is the stated intention, but whilst some contributors admirably discuss the therapeutic aspects, others ignore it and choose to describe detailed molecular mechanisms with absolutely no reference to their potential as therapeutic targets. The result is a somewhat schizophrenic book. This is a pity and detracts from the overall appeal. Notwithstanding the diversity, there are substantial areas of overlap between several chapters and the overall cohesion of the book would have benefited from more aggressive and comprehensive editorial cross-referencing. Similarly, the organization of chapters appears somewhat erratic – for example there are two chapters about p. 53, yet they are 300 pages apart. Overall, a brave attempt to encompass a broad, exciting and developing field. Inevitably it will soon be out of date so probably best as an institutional purchase.

■ ***John R. Arrand***
***Institute for Cancer Studies, University of Birmingham***

## Atlas of Sexually Transmitted Diseases and AIDS, Third Edition

Edited by S.A. Morse, R.C. Ballard, K.K. Holmes & A.A. Moreland
Published by Mosby/Elsevier Science (2003)
£125.00, pp. 408
ISBN: 0-7234-3227-9

This is a comprehensive, well-illustrated text. It is more than an atlas. Each chapter has good balance of pathogenesis, clinical features and laboratory tests. There is a very satisfying emphasis on clinical features, with excellent pictures (too graphic to be a 'coffee table' book) and illustrated laboratory diagnosis. Effort is devoted to description of laboratory investigations, but not necessarily prioritizing their clinical significance.
The content is well-balanced. Although the title is 'STD and AIDS', only 3 of the 17 chapters are devoted to AIDS alone.
The laboratory tests describe the most currently available tests, molecular and non-molecular. This should make the text useful in resource-limited, as well as resource-rich countries.
This book will be a useful reference text for GUM/Infectious Diseases trainees, who need basic laboratory information and vice versa and those microbiology/virology trainees, who need the wide clinical experience.

■ ***Sheila Burns***
***Royal Infirmary of Edinburgh***

## Microorganisms in Foods 7: Microbiological Testing in Food Safety Management

By the International Commission on Microbiological Specifications for Foods (ICMSF)
Published by Kluwer Academic/Plenum Publishers (2002)
Euro144.50/US$125.00/£88.50, pp. 388
ISBN: 0-306-47262-7

*Microbiological Testing in Food Safety Management* by the ICMSF is an excellent text for researchers working in the field of food safety. It is well written and the ideas flow in a sensible and practical manner. It should be a guide to people working in both regulatory agencies and food production on the pragmatic use of a systems approach to managing food safety. This systems approach is apparent from the 'food safety objective' principle promoted by the book. This principle takes into account the complexity of the current production, distribution and consumption of food by combining information from various disparate sources. This includes data from epidemiological studies, public health surveillance statistics, microbial growth, food production processes, retail and consumption practices. Inclusion of statistical process control techniques in chapter 13 will be an added bonus for food producers.

■ ***Pradeep Malakar***
***Institute of Food Research, Norwich***

## Principles of Genome Analysis and Genomics, Third Edition

By S.B. Primrose & R.M. Twyman
Published by Blackwell Publishing (2002)
£29.50; pp. 263
ISBN: 1-4051-0120-2

The speed of the genome sequencing revolution is staggering. This welcome book explains why and how genomes are being sequenced, and the methods we are using to make sense of the mass of data. Aimed at advanced undergraduates, it is an excellent distillation of current knowledge. The authors discuss genome structure and organization. They then describe the cloning, mapping and sequencing of genomes, a reminder in these days when genome sequences appear apparently by magic, that this is a complex and intellectually challenging process. However, the meat of the book lies in the next six chapters looking at genome annotation and bioinformatics, comparative genomics, structural genomics, global expression profiling, comprehensive mutant libraries and the mapping of protein interactions. The final chapter skips through a few vignettes showing how these techniques can reveal important biological information. The book is clearly written, well presented, and feels good. Recommended.

■ ***Neil Stoker***
***Royal Veterinary College, London***

## Emerging Infections, Vol. 5

Edited by W.M. Scheld, W.A. Craig & J.M. Hughes
Published by American Society for Microbiology (2001)
US$79.95, pp. 266
ISBN: 1-5581-216-3

This latest volume brings the total number of individual reviews in this series on *Emerging Infections* to 77. As always these are succinct, timely and highly readable contributions on a mélange of bacterial, viral, fungal, and protozoal infections. Two chapters are particularly prescient, namely, (13) 'Bioterrorism: a real modern threat' and (14) 'Bioterrorist threats: what the infectious disease community should know about anthrax and plague'. I found much of interest outside of my area. While the chapters on small colony variants of *Staphylococcus aureus* and on *Mycoplasma pneumoniae* infections were instantly attractive to me, I became readily engrossed with those on West Nile Fever in New York City, amoebiasis, enterovirus 71, and water-transmissible diseases and haemodialysis. Those who have read the first four volumes in this series will not be disappointed with this latest. The Editors' message that '*it is time to see emerging infections as true surrogates for an alien invasion*' poignantly summarizes the important contribution of this series to public health.

■ ***Cyril J. Smyth***
***Trinity College Dublin***

## Probiotics and Prebiotics: Where Are We Going?

Edited by G.W.Tannock
Published by Caister Academic Press (2002)
£90.00/US$180.00, pp. 333
ISBN: 0-9542464-1-1

This book provides an excellent summary of the current state of play in the field of pro- and prebiotics, and addresses topics ranging from the composition and functionality of the intestinal microflora, to utilizing genomic technology to improve strains from industrial and health promoting perspectives. The format of the chapters is such that researchers new to the field can quickly become familiar with the latest studies and techniques used in each area, e.g. the role of probiotics in calcium bioavailability or IBD. The contributors are highly regarded in their field and, as well as giving a basic overview, cover each topic in sufficient detail to provide the reader with a sound basis from which to begin research using the techniques and experimental designs discussed within. This book will not only be valuable to workers in the field of pro- and prebiotics, but also for those in other areas of microbial ecology.

■ ***Marie-Louise Baillon***
***Waltham Centre for Pet Nutrition, Waltham-on-the-Wolds***

## Advances in Microbial Toxin Research and its Biotechnological Exploitation

Edited by R.K. Upadhyay
Published by Kluwer Academic/Plenum Publishers (2002)
US$115.00/£80.50/Euro132.00, pp. 288
ISBN: 0-306-47255-4

This book contains a number of relatively specialized chapters concerned with microbial toxins. I did find the title of the book slightly confusing as to whether it was about toxins made by microbes or toxins acting on microbes. The toxins described and discussed in this book are mainly the low-molecular-weight secondary metabolites produced by various organisms. However, as an overview of the current research into, and applications of low-molecular-weight toxins, this book is a very useful source of information. The book will be of particular use to courses in applied aspects of the subject as there is a wealth of information on the use of low-molecular-weight toxins in biological control processes. Although some of the chapters are relatively specialized in the interactions they describe, the book as a whole provides a good overview of the current status of action and use of these agents.

■ ***Tim Mitchell***
***University of Glasgow***

## Fungal Populations and Species

By John Burnett
Published by Oxford University Press (2003)
£37.50, pp. 348
ISBN: 0-19-851553-7

It is John Burnett's hope that fungi take their rightful place in the mainstream of biological thinking. Burnett brings together in one volume the approaches and attractions of working with fungi in the areas of population biology and speciation. To quote Pringle & Taylor (2002, *Trends Microbiol*, **10**, 474–481), '*fungi are complicated, but tractable*' and it is the tenor of Burnett's book that fungi are open to investigation by a range of techniques, some of which are described in detail, for the study of populations, fitness, variation and speciation. Topics as diverse as molecular genetics and ecology are covered. In contrast, the book is weak on genomics with no mention, for example, of genome evolution and speciation in *Saccharomyces*. Also, lichen fungi receive scant attention. Overall however, this book reflects the knowledge and commitment of its author and deserves to sell well as an aid to both teaching and research.

■ ***David Archer and Paul Dyer***
***University of Nottingham***

## Viral Vectors for Gene Therapy: Methods and Protocols. Methods in Molecular Medicine Series, Vol. 76

Edited by C.A. Machida
Published by Humana Press (2002)
US$135.00, pp. 589
ISBN: 1-58829-019-0

This book covers the major viruses that have been utilized to transfer and express genetic material. At first glance it appears invaluable to all those aiming to construct viral vectors, however the chapters are often very specialized and therefore are only likely to be of use to researchers making specific modifications to vector systems. The chapters are, however, comprehensive and easy to follow and, considering the number of chapters dealing with the same virus, there is little overlap in text between them. The book would benefit from an introductory chapter describing the different viral systems and the features lending them to particular applications. Despite 'gene therapy' appearing in the title and the large number of clinical trials using such vectors, there is little mention of the administration of viruses to patients. The price of the book is likely to restrict its purchase to organizations.

■ ***Christopher Ring***
***Glaxo SmithKline R&D, Stevenage***

## Plant Viruses as Molecular Pathogens

Edited by J.A. Khan & J. Dijkstra
Published by The Haworth Press, Inc. (2002)
HB US$129.95 (HB);
PB US$59.95, pp. 537
ISBN: 1-56022-894-6 (HB);
1-56022-895-4 (PB)

Thanks to sequencing and genetic manipulations of various kinds, great strides have been made in understanding how plant viruses function as pathogens at the molecular level. This book covers aspects such as virus replication and gene expression, vector transmission, natural and engineered resistance, and molecular techniques, with two bonus chapters on the principles of virus taxonomy and virus names. It has to be said the coverage – by 37 authors in 21 chapters – is patchy. Some give good broad overviews of their subject area, but a few are restricted to work from their own laboratories. There is little or nothing on virus particle assembly and structure, symptom formation or effects on plant metabolism. In the paperback edition, many of the illustrations can only be described as 'faint and fuzzy'. The book covers some of the territory of the 2002 fourth edition of Matthews' *Plant Virology*, by Roger Hull, but that has a broader coverage.

■ ***Ron Fraser***
***SGM Marlborough House***

## Genomics of GC-Rich Gram-Positive Bacteria. Functional Genomics Series, Vol. 2

Edited by A. Danchin
Published by Caister Academic Press (2002)
£75.00/US$150.00, pp. 178
ISBN: 0-9542464-3-8

The GC-rich group of Gram-positive bacteria include diverse bacterial types with very different reputations: while *Rhodococcus* is an agent of bioremediation and *Streptomyces* species are noted for antibiotic production, both *Mycobacterium tuberculosis* and *M. leprae* are well known pathogens causing tuberculosis and leprosy, respectively. This book focuses on the impact that complete genome sequence information has and will have on our knowledge of these bacteria. Although the complete genome sequence of a *Rhodobacter* species is not yet publicly available, the chapter on the current state of *Rhodobacter* genetics shows that the tools for genetic manipulation are available to exploit knowledge of the genome sequence. The chapter on global analysis of *Streptomyces* gives an overview of its genome organization and both morphological and metabolic differentiation. The power of genomic analysis is exemplified by *S. coelicolor*, which was previously known to produce three antibiotics, but the complete genome sequence has revealed 20 gene clusters potentially capable of synthesizing secondary metabolites. The three chapters on *Mycobacterium tuberculosis* focus on the contribution of genomics to identification of new virulence factors, on our understanding of secretion and its contribution to virulence and on the *TubercuList* database, based on the *GenoList* model that has served the *Escherichia coli* (*Colibri*) and *Bacillus subtilis* (*SubtiList*) communities so well. Finally, there is a very good chapter on *Mycobacterium leprae* and its highly truncated genome making a strong case that genomic analysis will greatly expedite and facilitate research on this experimentally intractable organism. Overall this book is a welcome addition, although I felt that the Editor in his chapter on 'Genomes and evolution' could have addressed whether the pre-genomics era classification of 'GC-richness' is supported by any distinctive feature of genome architecture or stability now that we know the complete genome sequences of some bacteria in this grouping.

■ ***Kevin Devine***
***Trinity College Dublin***

## Host Response Mechanisms in Infectious Diseases. Contributions to Microbiology, Vol. 10

Edited by H. Herwald
Published by Karger (2003)
CHF240.00/Euro171.50/US$208.75, pp. 260
ISBN: 3-8055-7486-X

This is an expensive but authoritative collection of 12 highly detailed chapters by an international group of specialists in the field of host response mechanisms. Each chapter takes care to introduce the subject area before launching into detail. I was impressed by the overall clarity of writing and editing.
A recurring problem for such texts is the lag time between chapter writing and publication. Thus, the book contains virtually no 2002 references.
I particularly enjoyed Bevin's comprehensive chapter on mammalian antimicrobial peptides – there are only 100+ of them, and the text is supported by 381 references!
There are few diagrams in general, but the use of bold colours in the schematic illustrations in Würzner's chapter on Complement is to be applauded.
The book should earn a place as a reference source on library shelves of microbiology, molecular biology, immunology and infectious disease departments.

■ ***Mark Wilcox***
***University of Leeds***

## Peptide Antibiotics: Discovery, Modes of Action, and Applications.

Edited by C.J. Dutton, M.A. Haxell, H.A.I. McArthur & R.G. Wax
Published by Marcel Dekker, Inc (2001)
US$150.00, pp. 296
ISBN: 0-8247-0245-X

This excellent book reviews the current state of knowledge of peptide antibiotics in almost 300 pages. Its international range of expert contributions have been well edited to produce a cohesive text. It is comprehensive covering peptides from many animal, plant and microbial sources.
I particularly liked the chapter on amphibian peptides. As well as informing, and clearly describing what is known (and not known) it brought me back to childhood pondside activities. Clearly, the frogs we caught were protected by a range of polypeptides secreted from neuroendocrine-like cells in response to the injuries we inflicted!
This book takes us from genes through production, regulation, structure, function and practical issues, regarding exploitation and potential use of these agents. It is comprehensive and informative with a readily accessible style. I happily recommend it to anyone interested in antimicrobial agents and consider it a must for those working in the field.

■ ***Kathy Bamford***
***Imperial College London***

## Hurdle Technologies: Combination Treatments for Food Stability, Safety and Quality. Food Engineering Series

By L. Leistner & G.W. Gould
Published by Kluwer Academic/Plenum Publishers (2002)
Euro144.50/US$125.00/£88.50, pp. 208
ISBN: 0-306-47263-5

*Hurdle Technology* is the deliberate use of a combination of preservation procedures, designed to extend the shelf-life of food in a synergistic manner that not only enhances its safety, but also gives improved flavour and quality. This authoritative book on the topic, written by two very experienced food microbiologists, focuses very much on applications: it covers in great detail examples ranging from highly processed products to traditional foods. The chapter on 'Basic aspects' reviews preservative mechanisms and bacterial responses in a readable way that avoids unnecessary detail. Microbiologists wanting insight into latest research will have to look elsewhere – this is a book aimed at the multidisciplinary teams (from engineers to microbiologists) who are involved in the practical applications of hurdle technology. Academics responsible for teaching food science subjects will also find this a very useful book packed with clear explanations and interesting examples of food microbiology from everyday life.

■ ***Nick Russell***
***Imperial College London***

## MicrobeCards

By M.S. Peppler
Published by American Society for Microbiology (2002)
US$24.95, 103 cards per deck
ISBN: 1-55581-217-1

Would you like a pack of cards with a microbiological difference? If so, then *MicrobeCards* could be just for you. They cover 103 micro-organisms, including bacteria, viruses, fungi and parasites. Each card has several images on one side with a concise description of key features of an organism on the reverse. Overall the latter are accurate, clear and relevant. The choice of organisms is also very reasonable, but I do think that *Shigella dysenteriae* warrants inclusion. The vast majority of photographs used are of extremely high quality, especially the electron micrographs. These cards will appeal as a revision aid to students of medical microbiology and laboratory technology staff, as well perhaps to students of medicine and of nursing. The price is quite reasonable for individual purchase. My only real reservation is the size of the cards: at 6.5×9 cm, they are really quite small which means the print is very small and there is no room for highlighting material or for making extra notes.

■ ***Adrian Eley***
***University of Sheffield***

## Antibiotic and Chemotherapy, 8th Edition: Anti-infective Agents and their Use in Therapy

Edited by R.G. Finch, D. Greenwood, S.R. Norrby & R.J. Whitley
Published by Churchill Livingstone/Elsevier Science Ltd. (2003)
£115.00, pp. 964
ISBN: 0-443-07129-2

It is 40 years since the first edition of this book was published. In that time, it has accumulated many devotees and has become firmly established as one of the leading texts in the field of antimicrobial chemotherapy. For this 8th edition, the Editors have again collated contributions from an extensive group of internationally recognized experts. The resulting text remains authoritative and has been updated to include several new topics, such as oxazolidinones, drug discovery and regulatory considerations. The visual impact and clarity of the 8th edition have been enhanced by its slightly larger format, and by the use of more iconized and boxed sections. Anyone who owns an earlier version, and everyone with a real interest in the topic should seriously consider buying this book; a copy should certainly be placed in all microbiology libraries. Although it is not cheap, it represents excellent value for money.

■ ***Neil Woodford***
***Antibiotic Resistance Monitoring and Reference Laboratory, Specialist Reference Microbiology Division, Health Protection Agency, London***

## Publisher's website addresses

| | |
|---|---|
| Academic Press | www.academicpress.com |
| American Society for Microbiology | www.asmpress.org |
| Blackwell | www.blackwellpublishing.com |
| Churchill Livingstone/Elsevier Science | elsevier.com |
| CRC Press | www.crcpress.com |
| Haworth Press Inc | www.haworthpressinc.com |
| Horizon Scientific Press | www.horizonpress.com |
| Humana Press | www.humanapress.com |
| Karger | www.karger.com |
| Kluwer Academic/Plenum Publishers | www.wkap.nl |
| Marcel Dekker Inc | www.dekker.com |
| Nelson Thornes | www.nelsonthornes.com |
| Oxford University Press | www.oup.co.uk |
| World Scientific | www.wspc.com.sg |

### Introduction to Toxicology and Food

By T. Altug
Published by CRC Press (2002)
US$59.95/£29.99, pp. 168
ISBN: 0-8493-1456-9

Though the microbiological content of this book is limited, it may well be of some interest to food microbiologists. It provides a short introduction and overview of food toxicology which of course includes a number of toxins of microbial origin. The first few chapters serve as a general introduction to the field and are largely concerned with describing the various branches of toxicology, the classification of toxins and defining the meaning of various toxicological terms. These are followed by chapters on the mechanism of action of different classes of toxin and the various *in vivo* and *in vitro* toxicity tests used. The four main chapters describe natural toxins, contaminants (which include the bacterial and fungal toxins), food additives and chemopreventers in the diet. The book employs a rather abbreviated note-like style which makes it relatively easy to skim, although the English is a little clumsy in places.

■ ***Martin Adams***
***University of Surrey***

### Introducing Genetics: From Mendel to Molecule

By Alison Thomas
Published by Nelson Thornes (2003)
£17.00, pp. 216
ISBN: 0-7487-6440-2

It's nice to see a book that does exactly what it says on the cover. It introduces genetics for students without a strong genetics background and it does this by starting with 'whole organism' Mendelian genetics and working its way ultimately to the DNA level. This is a good approach for many students being introduced to genetics, since it starts with a phenomenon that is already familiar; variation between individuals. Through Thomas' lucid and concise prose the causal basis for phenotype variation is well explained. This is a slim, handy introductory guide that would help provide good foundations on which students could build using more complex and advanced texts, perhaps ones with more space to discuss areas not covered here, such as genomics. One reservation is that the text lacks colour, which is not ideal when representing complex genetic processes, but augmentation with appropriate websites would circumvent this problem.

■ ***Mike Speed***
***University of Liverpool***

### The Vaccine Book

Edited by B.R. Bloom & P.-H. Lambert
Published by Academic Press (2002)
£37.50, pp. 320
ISBN: 0-12-107258-4

Issues surrounding vaccination are topical and so publication of this book is timely. Although it purports not to be comprehensive, and indeed some vaccines such as those for hepatitis A and E are hardly mentioned, it is very detailed and is not quite the easy read suggested by the cover design. However, it should be considered as a useful reference book. The content covers every aspect of vaccination, including the global burden of disease, cost effectiveness, safety, ethical issues and the introduction of new vaccines. Therefore, it should appeal to a wide audience, including health economists and strategists, microbiologists and epidemiologists. Since it is not restricted to vaccines aimed at preventing infections but discusses other possible targets such as therapeutic, anti-cancer vaccines and vaccines against Alzheimer's, autoimmune and allergic disease, it should also be of interest to several other medical specialities.

■ ***Sue Skidmore***
***Princess Royal Hospital, Telford***

### Cytokines and Chemokines in Infectious Diseases Handbook. Infectious Diseases Series

Edited by M. Kotb & T. Calandra
Published by Humana Press (2003)
US$145.00, pp. 456
ISBN: 0-89603-908-0

This is a timely publication. Understanding of the role of cytokines in infectious diseases has increased dramatically in recent years. We now have a much better understanding of the role that cytokines play in host defence and the way that they may also contribute to pathology when produced in excess, or when there is inappropriate cytokine production during infection. This is leading to the development of therapies for some infectious diseases (e.g. sepsis) based on anti-cytokine strategies.
'Handbook', however, is the key word in the title. This is not a text that makes easy bedtime reading! An understanding of immunology, cytokine nomenclature and cytokine biology and associated jargon is a pre-requisite for tackling the very detailed text presented. This handbook will be of most use as a reference for academics teaching aspects of microbial immunology/ pathogenesis at an advanced level and for those actively pursuing research in the field.

■ ***Eileen Ingham***
***University of Leeds***

### Bioremediation: A Critical Review

Edited by I.M. Head, I. Singleton & M.G. Milner
Published by Horizon Scientific Press (2003)
US$180.00/£90.00, pp. 301
ISBN: 1-898486-36-0

This book is an eclectic collection of ten chapters on microbial aspects of bioremediation. Readers with slightly different backgrounds will accordingly differ in what they perceive as useful, interesting or appropriate. I particularly enjoyed the chapters on balancing the needs of researchers, practitioners, regulators and policymakers; a general treatment of microbial studies in bioremediation; ecotoxicology; anaerobic bioremediation and the application(s) of microbial inoculants. Also useful is a detailed chapter on geochemistry and hydrology of bioremediation by natural attenuation. A chapter on ecological theory, although of general interest, is too long and its relevance to bioremediation not always made clear. There is some overlap between the description of understanding bioavailability with the chapter on ecotoxicology and it might have been better to combine these two areas. Other topics covered are the applications of stable isotopes and developments in permeable reactive technology. Overall, this is a useful book that is well referenced up to 2001. However, it is too expensive for all but the committed expert.

■ ***Clive Edwards***
***University of Liverpool***

### Multiple Drug Resistant Bacteria

Edited by C.F. Amábile-Cuevas
Published by Horizon Scientific Press (2003)
£80.00/US$160.00, pp. 182
ISBN: 1-898486-45-X

This multi-authored volume focuses on the evolution of bacterial resistance to multiple antimicrobial agents. It includes chapters on mechanisms for the acquisition of resistance in both Gram-positive and Gram-negative bacteria, *mar* loci, MRSA and vancomycin-resistant enterococci, and also deals with multiple resistance in biofilms. The text is well illustrated and contains very helpful high-quality diagrams. The chapters are up to date and cited references extend to 2002. Perhaps the highlight of the volume is a chapter by Heineman & Silby on 'Horizontal gene transfer and the selection of antibiotic resistance'. This presents a critical evaluation of the behaviour of resistance plasmids and post-segregational killing mechanisms in relation to various aspects of evolutionary thinking, such as the selfish DNA theory.
For a text of 182 pages, this volume is not cheap. However, it would be a good purchase for individuals specializing in the evolution of resistance and a valuable addition to research libraries.

■ ***Jon Saunders***
***University of Liverpool***

# Diary

**Biotechnology for the Environment: Soil Remediation. Focus on Biotechnology, Vol. 3B**
Edited by S.N. Agathos & W. Reineke
Published by Kluwer Academic/ Plenum Publishers (2002)
Euro75.00/ US$68.00/£47.00, pp. 140
ISBN: 1-4020-1051-6

This book is a compilation of multidisciplinary research works on soil remediation. It successfully integrates the depth of the scientific principles with the breadth of application of biotechnology in treating contaminated soil. This book can be approached on two levels: as a useful reference and a research treatise on bioremediation. The introduction chapter gives a clear overview of the current trends in bioremediation, the chapters on humification of nitroaromatics and phytoremediation are recommended reading for any practitioner interested in bioremediating polluted sites, including those contaminated by munitions. At a more advanced level, this book describes the state of the art research in clean-up technologies such as slurry-based methods and life-cycle assessment, which will be pertinent to researchers and academics in the field of bioremediation. It is a valuable source book for soil professionals who are interested in the environmental application of biotechnology.

■ ***Diane Purchase***
***Middlesex University***

***E. coli*: Shiga Toxin Methods and Protocols. Methods in Molecular Medicine, Vol. 73.**
Edited by D. Philpott & F. Ebel
Published by Humana Press (2002)
US$125.00, pp. 375
ISBN: 0-89603-939-0

This book is aimed at both clinical (chapters 2–7) and cellular microbiologists (chapters 8–25). For the former, comprehensive protocols are provided for serological and PCR detection from humans, animals and foods and the molecular typing of STEC. These usefully indicate limitations, laboratory hazards, necessary sampling levels and controls. For cellular microbiologists six chapters cover aspects of pathogenicity in good detail, e.g. pathogenicity island analysis, generation of deletion mutants and monoclonal antibodies. However, confocal microscopy was surprisingly not mentioned. The following nine chapters provide detailed protocols for Shiga toxin biology studies, through purification, effects on host-cell functions and using the latter as tools for elucidating cellular mechanisms. The final chapters on animal models pull together otherwise widely dispersed information. Although this is certainly not a book for the uninitiated, who would require fuller indexing, the Editors have collated a very useful text with key points highlighted in the critical notes.

■ ***Martin Collins***
***The Queen's University of Belfast***

**The Way of the Cell. Molecules, Organisms and the Order of Life**
By F.M. Harold
Published by Oxford University Press (2003)
New in paperback
£12.95, pp. 305
ISBN: 0-19-516338-9

(A review of the hardback version can be found in the *Microbiology Today* Book Review section on the SGM website.)

## december 03

13TH INTERNATIONAL SYMPOSIUM ON THE BIOLOGY OF THE ACTINOMYCETES

**Melbourne, Australia**
**1–5 December 2003**

CONTACT: Symposium Secretariat, c/o Conference Strategy Pty. Ltd, PO Box 1127, Sandringham, Victoria 3191, Australia (www.conferencestrategy.com.au)

CORDIA EUROPABIO CONVENTION 2003

**Vienna, Austria**
**2–4 December 2003**

CONTACT: www.cordiaconvention.com/info

## january 04

DIAGNOSIS AND TREATMENT OF VIRAL DISEASES. EUROPEAN SOCIETY FOR CLINICAL VIROLOGY (ESCV) WINTER MEETING 2004

**Copenhagen, Denmark**
**15–17 January 2004**

CONTACT: Secretariat, Symposion International, Tranekaer Slot, Slotsgade 86, DK-5953 Tranekaer (Tel. +45 98332155; Fax +45 98332828; email kh@symposion-int.dk; www.escv-2004.dk)

HYGIENIC COATINGS & SURFACES. SECOND GLOBAL CONGRESS

**Orlando, Florida, USA**
**26–28 January 2004**

CONTACT: Janet Saraty, PRA, 8 Waldegrave Road, Teddington TW11 8LD (Tel. 020 8614 4811; Fax 020 8614 4812; email j.saraty@pra.org.uk; www.hygienic-coatings.com)

## february 04

GLOBAL CONFERENCE ON ANIMAL WELFARE

**OIE HQ, Paris, France**
**23–25 February 2004**

CONTACT: Dr A. Petrini, OIE, 12 rue de Prony, 75017 Paris, France [Fax +33 (0)1 42 67 09 87; email animalwelfare-conference@oie.int; www.oie.int]

## march 04

BSAC SPRING MEETING 2004. PARTNERSHIPS IN FIGHTING INFECTION – TIME TO ACT

**International Convention Centre, Birmingham**
**3–4 March 2004**

CONTACT: Phillippa McCoy, BSAC, 11 The Wharf, 16 Bridge Street, Birmingham B1 2JS (Tel. 0121 633 0410; Fax 0121 643 9497; email pjmccoy@bsac.org.uk)

## april 04

INTERNATIONAL CONFERENCE ON THE CONTROL OF INFECTIOUS ANIMAL DISEASES BY VACCINATION

**Buenos Aires, Argentina**
**13–16 April 2004**

CONTACT: Dr A.A. Schudel, Head, Scientific & Technical Dept. OIE, 12 rue de Prony, 75017 Paris, France [Tel. +33 (0)1 44 15 18 88; Fax +33 (0)1 42 67 09 87; email oie@oie.int; www.oie.int]

## june 04

MANAGEMENT OF PLANT DISEASES AND ARTHROPOD PESTS BY BCAS AND THEIR INTEGRATION IN AGRICULTURAL SYSTEMS (JOINT IOB/WPRS MEETING)

**St Michele all'Adige, Trentino, Italy**
**9–13 June 2004**

CONTACT: Yigal Elad, Convener (current email during sabbatical: y.elad@sbc.bbk.ac.uk; www.agri.gov.il/Depts/IOBCPP/JGroup/IOBCWPRSintegration1st.html)

## july 04

23RD ANNUAL SCIENTIFIC MEETING OF THE AMERICAN SOCIETY FOR VIROLOGY (SPONSOR MCGILL UNIVERSITY, MONTREAL)

**Montréal, Québec, Canada**
**10–14 July 2004**

CONTACT: Sidney E. Grossberg, Secretary–Treasurer, American Society for Virology, Dept of Microbiology and Molecular Genetics, Medical College of Wisconsin, 8701 Watertown Plank Road, Milwaukee, WI 53226-0509, USA (Tel. +1 414 456 8104; Fax +1 414 456 6566; email ASV@mcw.edu; www.mcw.edu/asv)

BIOSCIENCE2004: FROM MOLECULES TO ORGANISMS

**SECC, Glasgow**
**18–22 July 2004**

CONTACT: Meetings Office, Biochemical Society, 59 Portland Place, London W1B 1QW (Tel. 020 7580 3481; Fax 020 7637 7626; email info@BioScience2004.org; www.BioScience2004.org)

12TH INTERNATIONAL CONGRESS OF IMMUNOLOGY/4TH ANNUAL CONFERENCE OF THE FEDERATION OF CLINICAL IMMUNOLOGY SOCIETIES (IMMUNOLOGY/FOCIS 2004)

**Montréal, Québec, Canada**
**18–23 July 2004**

CONTACT: Immunology/FOCIS 2004 Secretariat, National Research Council Canada, Building M-19, 1200 Montreal Road, Ottawa, ON K1A 0R6 Canada (Tel. +1 613 993 7271; Fax +1 613 993 7250; email immuno2004@nrc.ca; www.immuno2004.org)

## august 04

10TH INTERNATIONAL SYMPOSIUM ON MICROBIAL ECOLOGY (ISME). MICROBIAL PLANET: SUB-SURFACE TO SPACE

**Cancun, Mexico**
**22–27 August 2004**

CONTACT: Prof. H.M. Lappin-Scott (email h.m.lappin-scott@ex.ac.uk; www.kenes.com/isme)

## october 04

XIII BOTRYTIS SYMPOSIUM

**Antalya, Turkey**
**25–31 October 2004**

CONTACT: Dr Yigal Elad (email elady@volcani.agri.gov.net.il)

## july 05

24TH ANNUAL SCIENTIFIC MEETING OF THE AMERICAN SOCIETY FOR VIROLOGY (SPONSOR PENN STATE UNIVERSITY)

**University Park, Pennsylvania, USA**
**18–22 July 2005**

CONTACT: Sidney E. Grossberg, Secretary–Treasurer, American Society for Virology, Dept of Microbiology and Molecular Genetics, Medical College of Wisconsin, 8701 Watertown Plank Road, Milwaukee, WI 53226-0509, USA (Tel. +1 414 456 8104; Fax +1 414 456 6566; email ASV@mcw.edu; www.mcw.edu/asv)

# Comment

## The importance of veterinary microbiology: the role of the SGM

Many current infectious disease scares involve animal hosts as in SARS/vCJD or the animals themselves, as in BSE or foot-and-mouth disease. Expertise is required to deal with these outbreaks, which not only cause suffering but can have severe social and economic consequences. Veterinary microbiology has never been more important, but does it receive enough support?

Animal diseases have been recognized since antiquity as significant threats to human health. Since the time of Pasteur's pioneering work on anthrax and rabies, there can be little doubt that the study of infectious agents of animals has been used to great effect in informing our understanding of human transmissible disease. Yet there is considerable disquiet as to the present state of veterinary microbiology as manifested by the difficulty in persuading veterinarians and science graduates to adopt a career in animal infectious disease, coupled with a perception that fundamental questions of pathogenesis can be best answered by studying human infections. Recently, The House of Lords Select Committee on Science and Technology* has served to focus attention on the acute need for enhancing capacity in veterinary microbiology, emphasizing that we neglect the study of animal sources of infection at our peril.

As part of this strengthening process, it is clearly necessary both to gather information about infections in humans and to set this in the context of the increasing relevance of zoonoses and food-borne disease to human health, as well as to the animal populations with which we share this planet. We must also rekindle in the wider microbiology community a fascination and enthusiasm for agents which are often more accessible for study, offer fresh insights into the disease process, and may have a serious economic impact. It is thus timely that this issue of *Microbiology Today* carries several very interesting articles on new agents of wild animals and zoonotic infections. Since 1970, previously unrecognized agents have emerged as significant threats to human and animal health at an average rate of nearly one a year, most of which are proven, or likely to be proven, as zoonoses. The list is now extensive, and includes BSE, vCJD, Ebola, HIV, Lyme Disease, Legionnaire's Disease, Nipah and Hendra viruses. Others have adopted new mantles or new habitats, such as *E.coli* O157 and West Nile virus.

The concern over zoonoses is not confined to wild animals. Companion animals increasingly are implicated in the transmission of diseases to humans, such as cat scratch fever, monkey pox, salmonelloses, psittacoses and *Campylobacter*. West Nile virus has now become a major disease of equines in the USA. The keeping of exotic pets has increased sharply over the last decade, offering further opportunities for zoonotic spread of pathogens that are, as yet, unrecognized.

Effective surveillance and control of animal disease requires the mobilization of a wide spectrum of microbiological knowledge. Engaging expertise in the wider academic, research and commercial communities not only increases the knowledge base but adds value to individual contributions through communications and collaboration. This relationship is sorely lacking in the UK and in many other countries. For example, the technology used for serodiagnosis of foot and mouth disease virus during the 2002 UK outbreak compares poorly with the sophistication and sensitivity of tests used for the screening of human blood for infectious agents. Comparative study of microbes across species adds substantially to the knowledge base by providing clues as to the drivers of genetic diversity, pathogenesis and how more effective vaccines may be developed. A notable example is the work on influenza viruses of humans, birds, animals and fish.

SGM Council is currently considering how best it may serve veterinary microbiologists, in the belief that it is ideally placed to foster close interactions between the wider community and those with a specific interest in animal infectious diseases. We believe that international collaboration is vital, particularly by forging links with the developing world where epidemics amongst farmed animals and wildlife, if left uncontrolled, can have a devastating impact. Aquaculture should not be forgotten as this is an increasingly important sector of food production. Training and continuing professional development needs for veterinarians and microbiologists employed by Government, universities, institutes and industry must be developed and tailored to meet current requirements. The SGM can and should be playing a pivotal role and we would be pleased to receive suggestions and comments as to the way forward. Please send these to Janet Hurst at Marlborough House (email j.hurst@sgm.ac.uk; Tel. 0118 988 1809).

● ***Professor Colin Howard, The Royal Veterinary College***

● ***Dr Geoffrey Schild, SGM Professional Affairs Officer***

**'Fighting Infection' published by the House of Lords Select Committee on Science and Technology (2003) is available at www.parliament.uk/hlscience*

● Please note that views expressed in *Comment* do not necessarily reflect official policy of the SGM Council.