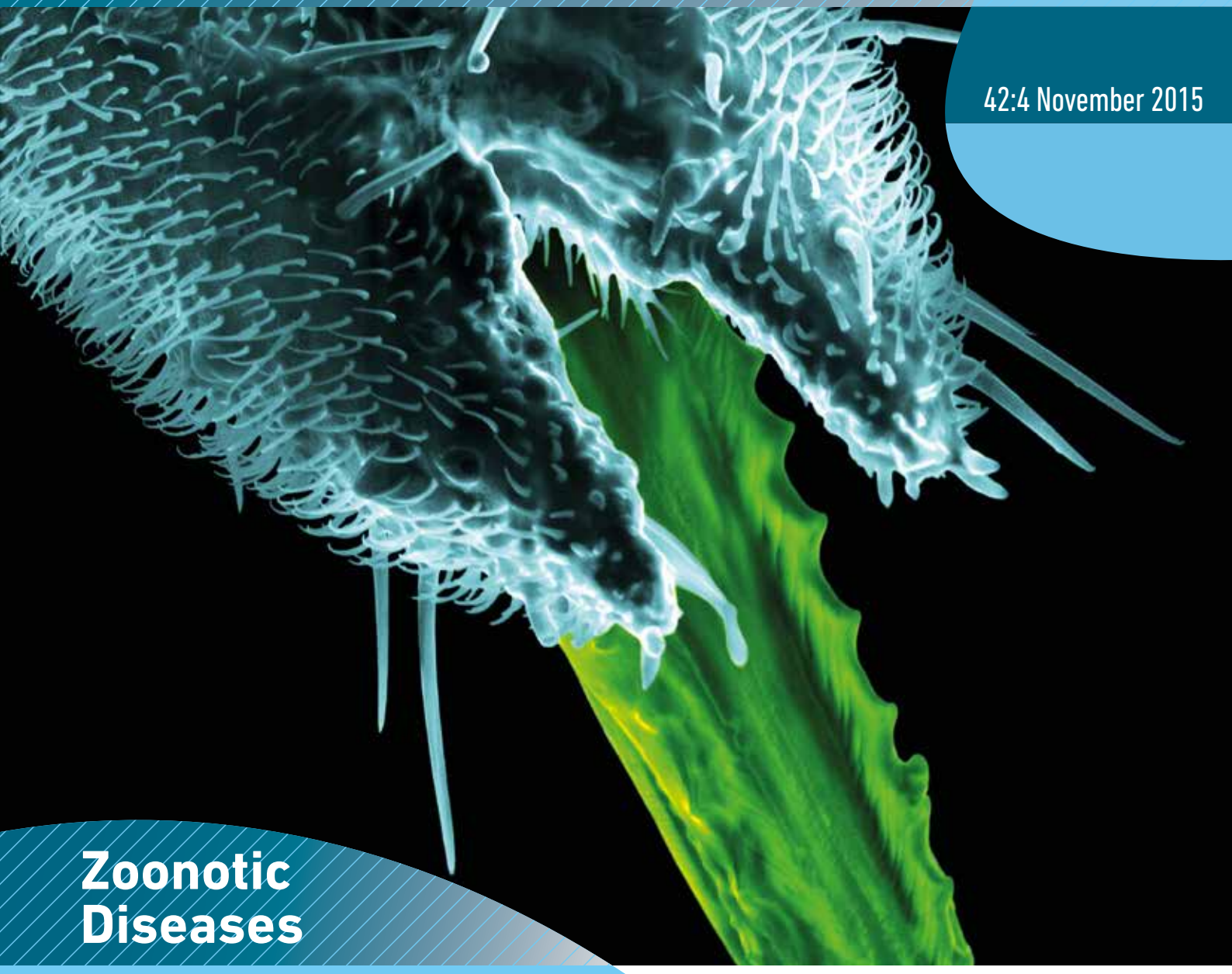
Microbiology TODAY

42:4 November 2015



Zoonotic Diseases

The problems of endemic zoonoses
Emerging zoonotic virus discovery
Hantaviruses – neglected zoonoses
Chikungunya: a decade of global pain
Campylobacter: breaking the spiral of infection

MICROBIOLOGY SOCIETY

CHLORAMPHENICOL

CAPSULES

Widely distributed throughout the body, including CSF[1]

Oral levels comparable to i.v. levels[2]

Rarely implicated with *C.difficile*[3]

Effective against serious infections including:

- *H. influenzae*[1,2]
- Typhoid[1,2]
- MRSA[4]
- VRSA[5]
- Neisseria[1,2]
- Legionella[1,2]
- Rickettsia[1,2]
- *C.difficile*[6-9]
- *E. coli*[1]

Abbreviated Prescribing Information
Chloramphenicol Capsules BP 250mg

Presentation: Hard Gelatin Capsules.
Indications: Typhoid fever and life-threatening infections, particularly those caused by *Haemophilus Influenzae*, where other antibiotics will not suffice.
Posology: For oral administration.
Adults and elderly: 50 mg/kg body weight daily in 4 divided doses. For severe infections (meningitis, septicaemia), this dose may be doubled initially, but must be reduced as soon as clinically possible. Children: Not recommended.
Contra-indications: Known hypersensitivity or toxic reaction to chloramphenicol or to any of the excipients. Should not be used for the prophylaxis or treatment of minor infections; during active immunisation; in porphyria patients; in patients taking drugs liable to depress bone marrow function; during pregnancy, labour or by breast-feeding mothers.
Special warnings and precautions for use: Use only if other treatments are ineffective. Use should be carefully monitored. Reduce dose and monitor plasma levels in hepatic or renal impairment; in the elderly; and in patients concurrently treated with interacting drugs.
Interactions: Chloramphenicol prolongs the elimination, increasing the blood levels of drugs including warfarin, phenytoin, sulphonylureas, tolbutamide. Doses of anticonvulsants and anticoagulants may need to be adjusted if given concurrently. Complex effects (increased/decreased plasma levels) requiring monitoring of chloramphenicol plasma levels have been reported with co-administration of penicillins and rifampicin. Paracetamol prolongs chloramphenicol half-life. Chloramphenicol may increase the plasma levels of calcineurin inhibitors e.g. ciclosporin and tacrolimus. Barbiturates such as phenobarbitone increase the metabolism of chloramphenicol, resulting in reduced plasma chloramphenicol concentrations. In addition, there may be a decrease in the metabolism of phenobarbitone with concomitant chloramphenicol use. There is a small risk that chloramphenicol may reduce the contraceptive effect of oestrogens. Chloramphenicol reduces the response to hydroxocobalamin. Chloramphenicol is contra-indicated in patients taking drugs liable to suppress bone marrow function e.g. carbamazepine, sulphonamides, phenylbutazone, penicillamine, cytotoxic agents, some antipsychotics including clozapine and particularly depot antipsychotics, procainamide, nucleoside reverse transcriptase inhibitors, propylthiouracil.
Pregnancy and Lactation: The use of chloramphenicol is contra-indicated as the drug crosses the placenta and is excreted in breast milk.
Effects on ability to drive and use machines: No significant effect on driving ability.
Undesirable Effects: Reversible dose related bone marrow depression, irreversible aplastic anaemia, increased bleeding time, hypersensitivity reactions including allergic skin reactions, optic neuritis leading to blindness, ototoxicity, acidotic cardiovascular collapse, nausea, vomiting, glossitis, stomatitis, diarrhoea, enterocolitis, Gray Baby Syndrome particularly in the newborn, which consists of abdominal distension, pallid cyanosis, vomiting, progressing to vasomotor collapse, irregular respiration and death within a few hours of the onset of symptoms.
Overdose: Stop chloramphenicol immediately if signs of adverse events develop. Treatment is mainly supportive. If an allergy develops, oral antihistamines may be used. In severe overdosage e.g. Gray Baby Syndrome, reduce plasma levels of chloramphenicol rapidly. Resin haemoperfusion (XAD-4) has been reported to substantially increase chloramphenicol clearance.
Pack size and Price: 60 capsules £377.00
Legal Category: POM.
Market Authorisation Number: PL17736/0075.
Market Authorisation Holder: Chemidex Pharma Limited, 7 Egham Business Village, Crabtree Road, Egham, Surrey TW20 8RB, UK.
Date of preparation: October 2014.
See Chloramphenicol Capsules Summary of Product Characteristics for full prescribing information.

Adverse events should be reported. Reporting forms and information can be found at www.mhra.gov.uk/yellowcard. Adverse events should also be reported to Essential Generics on 01784 477167.

References:
1. Martindale: The Complete Drug Reference. Chloramphenicol. [Online]. Available from: http://www.medicinescomplete.com [Accessed 18th August 2014]. **2.** Fluit, A.C., Wielders, C.L.C., Verhoef, J., and Schmitz, F.J. Epidemiology and susceptibility of 3,051 Staphylococcus aureus isolates from 25 university hospitals participating in the European SENTRY Study. Journal of Clinical Microbiology. 2001; 39(10): 3727-3732. **3.** Weigel LM *et al.* High-level vancomycin-resistant Staphylococcus aureus (VRSA) associated with a polymicrobial biofilm. Antimicrobial Agents and Chemotherapy. Published online ahead of print on 30th October 2006. http://aac.asm.org/cgi/reprint/AAC.00576-06v1.pdf. (Accessed on 22nd August 2011). **4.** Kelly, C., LaMont, T. Patient information: Antibiotic-associated diarrhea (Clostridium difficile). www.uptodate.com. 2011. **5.** Feder. H, Chloramphenicol: What we have learned in the last decade. Southern Medical Journal. 1986; (79)9: 1129-34. **6.** Ensminger, P., Counter, F., Thomas, L., Lebbehuse, P. Susceptibility, resistance development, and synergy of antimicrobial combinations against Clostridium difficile. Current Microbiology. 1982; 7: 59-62. **7.** Poilane, I., Bert, F., Cruaud, P., Nicolas-Chanoine, MH., Collignon, A. Interest of the disk diffusion method for screening Clostridium difficile isolates with decreased susceptibility to antibiotics. Pathologie Biologie (Paris). 2007; 55(8-9): 429-33. **8.** Cattoir, V., Ould-Hocine, ZF., Legrand, P. Antimicrobial susceptibility of Clostridium difficile clinical isolates collected from 2001 to 2007 in a French university hospital. Pathologie Biologie (Paris). 2008; 56(7-8): 407-11. **9.** Brazier, JS., Levett, PN., Stannard, AJ., Phillips, KD., Willis, AT. Antibiotic susceptibility of clinical isolates of clostridia. Journal of Antimicrobial Chemotherapy. 1985; 15(2): 181-5.

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Editorial

In our February edition of *Microbiology Today*, the Comment article focused on a story that has dominated our headlines: the Ebola pandemic that swept through West Africa and migrated onto European shores. It illustrates that infectious diseases are a clear and ever present danger that demand vigilance and a worldwide response, and can have catastrophic effects on societies. Middle East respiratory syndrome (MERS) has also received media attention, and it is interesting that this disease started to hit the headlines as it emerged out of the Middle East to cause cases of infection in 26 countries including parts of the European Union.

Many emerging diseases that are causing concern with the public and healthcare communities are zoonotic diseases, but they are not a 21st century phenomenon. The World Health Organization describes rabies as a vaccine-preventable, 'neglected disease of poor and vulnerable populations whose deaths are rarely reported', and it continues to cause tens of thousands of deaths every year. Rabies has circulated for centuries. Jo Halliday, Katie Hampson and Tiziana Lembo touch on this in a thought-provoking article that describes the problems of endemic zoonoses that have the greatest impacts in developing countries, but get limited recognition. Their eradication could significantly improve health and livelihoods in some countries.

Hanna Jerome, Sreenu B. Vattipally and Emma C. Thomson begin their informative article in the 19th century with Louis Pasteur, who noticed that rabies was caused by an agent that could not be visualised using a light microscope. The authors describe how the toolkit for identification of zoonotic viruses has evolved substantially over the last century, leading to the current scenario where we may be able to predict which viruses might cross the species barrier to present a significant risk to the human population.

John Fazakerley describes chikungunya, a disease that has emerged recently from the forests of Africa to start its global journey, out across the islands of the Indian Ocean to India and South-east Asia, China and the Americas. The alphavirus – a small, enveloped, positive-sense RNA virus – first emerged to prominence early in 2005, causing severe joint pain and general discomfort. The virus is transmitted by *Aedes aegypti* and *Aedes albopictus* mosquitoes, which are now widely spread around the world.

Agnieszka Szemiel discusses another virus that has recently hit the headlines, hantaviruses, which are the only bunyaviruses that are direct zoonoses. The virus is maintained in the environment by persistent infections of rodents and causes little damage to its vector. It is transmitted to humans via aerosolised rodent urine, faeces, saliva, and occasionally by bite. Infection in humans can be fatal.

Helen Brown and Arnoud van Vliet's article highlights that it is not just viruses that cause zoonotic diseases. Although *Campylobacter* was, arguably, the least known of the common bacterial food pathogens and very rarely made the headlines, the number of *Campylobacter* cases has remained relatively steady over the last few decades (an annual incidence of ~280,000 UK cases). In the past year public awareness of *Campylobacter* has been raised significantly, mainly due to the Food Standards Agency publishing data on the percentage of *Campylobacter*-positive meat samples from retail (64–79% of chicken meat was positive) putting pressure on the poultry sector and retailers to address the problem.

Zoonotic diseases are a global problem affecting both rich and poor countries. Professor Eric Fèvre has written a Comment piece describing the significant differences between a continent like Africa and one like Europe; namely the ability of national and regional systems to detect and respond to zoonotic disease threats. A common theme that has emerged from the articles is the economic burden that zoonotic diseases place on society, with the impact generally felt most keenly by the vulnerable and the poor. The articles also highlight that science offers solutions. We continue to develop vaccines, improve prevention, increase surveillance and adapt detection methodologies to sophisticate information. As mankind continues its impact on earth and its interactions, new zoonotic diseases will emerge. Maybe we are more ready now than we have ever been to meet these challenges?

Laura Bowater
Editor
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Regulars

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FSC Logo

Coloured scanning electron micrograph of the proboscis of an *Anopheles gambiae* mosquito. Science Photo Library

MICROBIOLOGY SOCIETY

Council 2015

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From the President

This is my last column in *Microbiology Today* as your President, and I have been reflecting on what the Society has achieved over the last three years. Our future is in the hands of the new President, together with the refreshed Council, Committees and Divisions, and I am delighted to hand the non-existent presidential chain-of-office to Professor Neil Gow. I know that Neil will work with the best interests of the Society at heart and will further develop our strategic aims and direction.

Ian Atherton

My first column in November 2012 identified some of the changes that the Society was proposing in publishing and in our web presence, and the changes that had just occurred in Committee structure to address the 2012–2017 Strategic Plan. I flagged the importance of ensuring equality and diversity, one of several actions continued from Hilary Lappin-Scott's presidency. I also mentioned how raising awareness of microbiological issues to policy-makers was an important role for the Society and I proposed that we engaged more with government and public bodies.

Of course, things have moved on considerably since then. In publishing we now have two new journals – *JMM Case Reports* and *Microbial Genomics* – and a new journals platform. We are happily ensconced in London with our sister societies, with many new staff working in a new internal structure under a new Chief Executive. We have just opened a second building in London which can be used for conferences and meetings and we have changed our conference structure from two full Meetings a year to an Annual Conference and a series of Focused Meetings. In addition, our engagement with other learned societies, research councils, government departments, policy-makers and opinion-formers has increased considerably since our relocation to London.

I am pleased that we now have much better and more transparent communication with the membership, and that our membership is increasing. The role of the Society's Champions has been important in developing our member engagement, and I thank them for their work. However, there is still some way to go in enlarging our membership beyond academic molecular and cell microbiologists to embrace other aspects of academic, clinical and industrial microbiology.

We also need to engage with early career microbiologists better in all our activities, not just in attendance at meetings. They are the future of microbiology and of the Society, and I know that Neil Gow sees this as a priority for the immediate future. I am pleased to be able to help in this work when I become a member of the Professional Development Committee from 1 January.

Of course, one of the biggest changes that has affected the Society in the last three years is our change of name. At our AGM in September it was agreed that we should become the Microbiology Society. I applaud the forward vision of our staff who ensured that we had captured the web and email addresses relevant to a change of name and who made sure that the logo adopted during our rebranding could be modified very simply to accommodate a name change.

Some progress is rapid and some is more hesitant. Fast or slow, the different activities of the Society are moving forward in line with our vision and mission as embodied in the Strategic Plan. For that I would like to thank the current and past members of Council, Committees and Divisions and our excellent and dedicated staff, all of whom work hard to realise the Society's objective "to advance the art and science of microbiology".

Nigel Brown
President
president@microbiologysociety.org

From the Chief Executive

At the Annual General Meeting in September, the members voted to change the name of the organisation. After 70 successful years, you agreed that a more straightforward name – Microbiology Society – would serve us better in the future and that the word 'General' had outlived its usefulness. But while the Society's title might have been updated, our long-standing commitment to serving all microbiology communities has remained constant. Over the past few months, I have very much enjoyed seeing that commitment in action at a variety of events and conferences.

Back in June, it was a great pleasure to attend the Irish Division meeting in Galway on *Microbial Interfaces*. The range of topics was impressive, but perhaps even more so was the extent to which members were engaging with microbiologists outside of their own field. Clinical work being shared with environmental microbiologists, ecologists swapping experiences with geneticists and molecular biologists interacting with epidemiologists.

In July, it was inspiring to meet the teachers at the Small World Initiative Summer School in Reading, who will encourage the next generation of microbiologists, and in September, it was great when 12 of the Society's Champions came together to share experiences of representing the Society on the ground in hospitals and universities. Linda Oyama even found the time to come while preparing for her PhD viva a few days later.

There have been two Focused Meetings recently – one on arboviruses in Glasgow and one on invasive fungi in Manchester. Since they were held on the same two days as each other, it was a challenge to attend both, but it was worth it to hear about such a wide range of the microbiological questions that you are studying. Twenty different countries and five different continents were represented among the speakers and poster-presenters, who came from hospitals, universities, public sector agencies and private sector companies. It was a pleasure to meet and listen to people from all career stages, from Mariana Almeida, a PhD student at Aberdeen who gave her first presentation at a major conference, speaking about fungal hyphae, to Carol Blair, a virologist from Colorado with decades of experience and wisdom to share.

And, of course, at the AGM itself, we heard from eight inspiring finalists in the Howard Dalton Young Microbiologist of the Year competition. They conveyed their passion for their work on viruses, bacteria and parasites with applications in animal and human health and in food microbiology. I hope that their careers will be helped not merely by being finalists in the competition, but also by learning from one another and from the contacts they have made at the Society.

Perhaps most of all, I have enjoyed visiting you in your own habitats. Over the past few months, I have been to Nottingham, Glasgow, Dublin and Aberdeen to speak to members at all career stages about what matters to you and how the Society can help. I have spoken to microbiologists working on prokaryotes, eukaryotes, viruses and parasites, some working on fundamental discovery research and some on questions with clinical, environmental, and industrial relevance.

The take-home messages are simple. You value opportunities to network, both professionally and scientifically. The Society must strive to ensure that the conference programme has something for everyone, and we must offer as many chances as we can for you to develop your careers. That means not just grants – although they are crucially important – but also events and resources that allow you all to tap into the strength and depth of the knowledge and experience of the wider membership.

As we plan for the coming months and years, we are making sure that these messages are taken on board. We also need to continue listening to the views of microbiology communities, and I am always keen to accept invitations to visit members, so please let me know if you and your colleagues would like me to visit your laboratory.

Peter Cotgreave
Chief Executive
p.cotgreave@microbiologysociety.org

We are now the MICROBIOLOGY SOCIETY

At this point you've no doubt seen that the Society has made a major change: we are now the Microbiology Society. The change was made at our AGM, when 89% of votes cast were in favour of renaming the Society.

Why has this change been made?

70 years ago, when the Society was founded, the word 'General' was used to describe a learned society that encompassed all disciplines of microbiology. Over time, people's perceptions of the word have changed and to some 'General' implied a lack of focus. Also, while we will always remain a membership organisation that champions microbiology and microbiologists, we need to ensure that the public, policy-makers, school pupils, teachers and others can engage with the crucial importance of our subject. To these groups, our former name was slightly opaque.

Other than the name, will anything else about the Society be changing?

Renaming the Society is not about changing our core identity; it is about preserving it and helping us to do our job even more effectively in the future. We will continue to host world-class conferences, publish journals of international repute and engage with policy-makers, educators, journalists and the public. The Society will continue to advance its vision of "A world in which the science of microbiology provides maximum benefit to society".

Have the Society's contact details changed?

Our homepage is now: **www.microbiologysociety.org** with our journals now found at: **www.microbiologyresearch.org** and staff emails are now in the form: **initial.surname@microbiologysociety.org**, but our phone numbers remain the same. Our Twitter handle has been changed to **@MicrobioSoc**

Will this affect my membership or grant application?

Your membership will be unaffected by the name change, and we will continue to offer grants to microbiologists at all stages of their careers. You can find out more information by visiting **www.microbiologysociety.org/grants**

Will this affect my journal subscription or manuscript submission?

The peer review and publication of your papers will be unaffected, and any open access fees will be automatically updated by Rightslink. The indexing of your paper in Pubmed, or similar, will also be unaffected. To email the Editorial Office, please contact **journals@microbiologysociety.org**

Professor Neil Gow to be new Society President

The Microbiology Society is delighted to announce that our new President will be **Professor Neil Gow** from the University of Aberdeen. Professor Gow's presidency will run for three years, beginning on 1 January 2016, when our current President, Professor Nigel Brown, steps down.

Neil has a strong affiliation with the Society, having joined as a PhD student in 1980. He has been a Senior Editor of the Society's journal *Microbiology*, a member of Council, and Chair of the Eukaryotic Division.

He said of his appointment: "I am delighted and honoured to be elected President of a society that I have had a lifelong relationship with. I'm looking forward to working with old friends and new colleagues to continue to ensure that the vital science of microbiology is supported, stimulated and has a strong voice in science and medicine."

Neil is a Wellcome Trust Senior Investigator and Director of a Wellcome Trust Strategic Award in Medical Mycology and Fungal Immunology. His research group is mainly focused on fungal cell wall biosynthesis and the pathogenic species of the *Candida* genus. He gained his undergraduate degree in Microbiology from the University of Edinburgh before undertaking a PhD at the University of Aberdeen. Previously, Neil has been President of the British Mycological Society and the International Society for Human and Animal Mycology.

Dr Peter Cotgreave, Chief Executive of the Microbiology Society, added: "I am delighted that the strong leadership shown by Nigel Brown will be followed by someone of the standing and calibre of Neil Gow. He is not just an outstanding scientist, but someone who understands how to bring people together to achieve more than they could on their own. I have absolutely no doubt that his presidency will be good for microbiologists, good for microbiology, and good for the application of microbial science to everyday challenges in society."

News

Young Microbiologist of the Year honoured

Joe Kirk receives his prize from Professor Dame Anne Glover.

Joe Kirk, a PhD student from the University of Sheffield, is the Microbiology Society's Young Microbiologist of the Year 2015. The Society's Annual General Meeting heard all eight finalists present their talks; Joe's was entitled 'Scratching the Surface; Targeting the Crystal Shell of *Clostridium difficile*'.

Second place went to **Samantha Chui Sang Lee** from the National University Ireland, with **Christopher Miller** from the University of Kent taking third place.

You can read more about the Young Microbiologist of the Year finalists on the Society's website: **http://microb.io/1Jrxwlz**

Parliamentary briefings

2015 is the International Year of Soils; to mark this, the Society will publish a Parliamentary briefing on UK soil health in December. With a growing population and the threat of climate change impacting our agricultural industry, sustainable crop production is vital. Utilising the microbes present in soil to selectively enhance the health and growth of crop plants will reduce the need for fertilisers and biocides, which damage the diversity of our soil microbiome.

Our Policy intern, Rebecca Philp from The Pirbright Institute, has summarised the issues. In addition, in June 2015, the Society published a Parliamentary briefing on emerging zoonotic diseases. Both briefings can be downloaded here: **www.microbiologysociety.org/briefings**

Parliamentary events foster relations between science and policy

The Society's Policy team recently attended Science and Stormont in Belfast, and Science and the Parliament in Edinburgh. The respective themes of the events, which included networking opportunities and talks, were 'Energy and the Environment' and 'Science and the Scottish Election'. The events are organised by the Royal Society of Chemistry in cooperation with other science organisations, including the Microbiology Society.

Members interested in attending next year's events in Wales, Northern Ireland and Scotland can contact the Society's Policy Officer, Paul Richards (**p.richards@microbiologysociety.org**).

2015 Society Outreach Prize

Dr Adam Roberts from the UCL Eastman Dental Institute has been awarded the Society's 2015 Outreach Prize. Adam founded the 'Swab and Send' citizen science project aimed at increasing awareness of antimicrobial resistance with the public and is involved with the Society's own Small World Initiative. He gave a talk entitled 'Engagement by Participation' at the Society's AGM, where he shared his experiences and latest results.

Conferences

Two new Focused Meetings will be taking place in 2016:

Molecular Biology and Pathogenesis of Avian Viruses, 27–29 September

Molecular Biology of Archaea 5, 1–3 August.

Our Annual Conference 2016 will be held 21–24 March at the ACC, Liverpool.

Visit pages 168–169 for more details.

Deaths

It is with sadness that we note the deaths of the following members:

Professor George Tennant Macfarlane, Centre for Oncology & Molecular Science, University of Dundee, who joined the Society in 1983.

Dr Forbes Robert Wardrop, Biotechnology Research Institute, who joined in 1995.

Dr Elizabeth Margaret Ellis, Strathclyde Institute of Pharmacy & Biomedical Sciences, University of Strathclyde, who joined in 1988.

Please contact **mtoday@microbiologysociety.org** if you wish to notify the Society of the death of a member whose details can be included in this section.

Upcoming grant deadlines

Date	Grant	Notes
30 November 2015	Hayes–Burnet and Heatley–Payne Awards	To support postdoctoral researcher members wishing to establish/ strengthen collaborations in Australia or the USA
1 December 2015	Travel Grants	For conferences from **1 January 2016** onwards*
10 December 2015	Harry Smith Vacation Studentships	For undergraduate summer research projects in 2016
29 January 2016	Society Conference Grants	To support eligible members to attend the Annual Conference 2016, Liverpool
	Undergraduate Student Conference Grants	
	Inclusion Grants	
1 March 2016	Travel Grants	For conferences from **1 April 2016** (including the Annual Conference 2016 for Full Members)
15 March 2016	Microbiology in Schools Fund	For visits and events from **1 December** onwards

Rolling application

Local Microbiology Event Sponsorship

Eligible members can apply for funds to support microbiology-related events, e.g. sponsored talks.

**Please note, you do not need to have received confirmation of abstract acceptance to apply for these grants as conditional offers will be made. In this case, evidence of acceptance is required to claim your grant.*

Small World Initiative at Number 10 Downing Street and citizen science pop-up events

The Society visited Number 10 Downing Street to dig in the garden there, as part of the Small World Initiative project's search for antibiotic-producing bacteria. This follows the Society receiving letters of support for the project from the Prime Minister's Office, the Science Minister Jo Johnson and the Department of Health. The sample has been sent for analysis at the University of East Anglia, joining over 300 samples collected during the summer by members of the public who visited the Society's citizen science events at High Lodge, Thetford Forest and Alice Holt Forest, Surrey. The public, and the Prime Minister, will be able to follow and contribute to the analysis of their samples online. Find out more on pages 176–177.

Microbiology Society President Nigel Brown takes a soil sample with Rory Spence, aged four.

Biosciences Athena SWAN workshop

The Microbiology Society, in partnership with the Biochemical Society, British Ecological Society, Royal Society of Biology and Society for Experimental Biology, is running a workshop for university and research institute bioscience departments on best practice and the challenges in applying for an Athena SWAN award on 11 December 2015 at Charles Darwin House.

The event will be chaired by Professor Hilary Lappin-Scott. Participants will hear from current Athena SWAN award holders, panel members and the Athena SWAN team and will take part in interactive activities aimed at producing high quality applications to the Athena SWAN process.

Contributions and feedback

The Society welcomes contributions and feedback from members. Please contact **mtoday@microbiologysociety.org** with ideas.

Benjamin Thompson
Head of Communications
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Livestock form an integral part of people's livelihoods, particularly in rural areas of the developing world. Tiziana Lembo. University of Glasgow.

Invisible and ignored: lifting the lid on the problems of endemic zoonoses

Jo Halliday, Katie Hampson & Tiziana Lembo

Worldwide the lives of billions of people are affected by zoonoses – diseases transmitted from animals. These diseases have the greatest impacts in developing countries, but are poorly recognised and largely ignored as human health problems. Improved clinical management of human cases and preventive interventions targeted at animal populations have the potential to achieve major improvements for both human health and livelihoods.

Humankind has long depended on animals for their livelihoods, nutrition, trade and protection. The relationships between people and animals are multi-faceted, but on balance, these interactions are overwhelmingly positive. Unfortunately, the close association of humans and animals also provides opportunities for the transmission of zoonoses, which account for two-thirds of all human infectious diseases.

The animal sources of zoonoses are varied, and include the companion animals in our homes, the rats in our sewers, the livestock that feed us, and the wild animals that enrich our natural world. The zoonotic pathogens that attract media attention are normally those that are described as emerging and raise concern of global spread. These include, for example, Ebola virus, Middle East respiratory syndrome (MERS) and severe acute respiratory syndrome (SARS) coronaviruses, and highly pathogenic influenza viruses. In contrast, there is surprisingly little awareness of the considerable burden which endemic zoonoses impose day in, day out.

Although endemic zoonoses occur worldwide, they are particularly problematic in the developing world, where hundreds of millions of people live in close contact with animals and where animal disease control strategies are often insufficient. In contrast to the zoonoses with pandemic potential that receive the greatest global attention, many less visible endemic zoonoses, such as rabies, leptospirosis, leishmaniasis, Japanese encephalitis and human African trypanosomiasis, exert a much higher global death toll. Many others, including cysticercosis, echinococcosis and brucellosis are highly debilitating diseases that cause prolonged suffering and disability.

Small stock are an important livestock resource for women in traditional livestock-keeping systems in rural Africa. Maasai woman tending to a flock of goats in Tanzania. Tiziana Lembo. University of Glasgow.

The invisibility of zoonoses

Why are endemic zoonoses invisible? Very few zoonoses have distinctive diagnostic signs, making clinical diagnosis impossible for both human and animal infections. Non-specific symptoms and signs in humans, such as fever, fatigue, muscle pain, joint pain, malaise, lack of appetite, weight loss and generic neurological signs can be caused by a range of zoonoses. There are currently insufficient diagnostic tests available to accurately and rapidly identify the cause of these non-specific clinical presentations. Furthermore, many zoonoses have complex ecology and epidemiology, which limits our ability to clearly observe and understand their infection dynamics. As a result, zoonoses suffer ongoing neglect in terms of training, research and surveillance efforts, and raising awareness.

There are also strong associations between zoonoses and poverty and the populations most afflicted by these

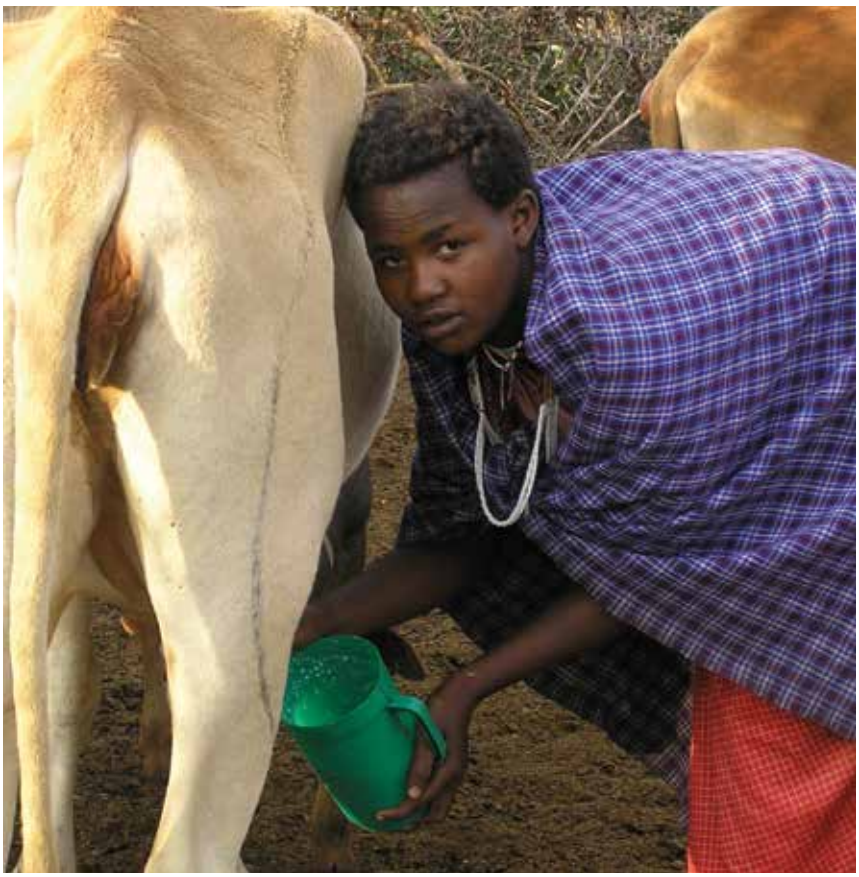
diseases are the same populations that have least voice. Poor people often lack political agency and are rarely able to mobilise resources or galvanise action to control these diseases. The diseases themselves are diagnostically challenging, particularly at more chronic stages of infections, which is when people from poor and remote communities are mostly likely to reach health facilities.

The multiple impacts of zoonoses

There is growing recognition of the impacts of zoonoses on both people and animals that extend beyond clinical disease to a wider socio-economic context. An often overlooked feature of zoonoses is their effect on livelihoods, owing to the impacts they have on the health and productivity of the animals on which humans depend. Brucellosis and leptospirosis, for example, cause reductions in growth and milk yield, and also affect the fertility of livestock.

Only a limited number of studies have robustly quantified the multiple impacts of a given zoonosis. A recent study that estimated the global burden of endemic canine rabies is one of the few studies to have addressed these knowledge gaps. Considering the direct health impacts first, rabies is the most deadly viral disease, killing almost 100% of people affected. The vast majority of human rabies deaths, around 59,000 per year, result from dog-transmitted rabies in Asia and Africa. A similar death toll has been estimated for human leptospirosis, with a particularly high incidence of cases in Oceania, South-east Asia, the Caribbean and East Africa.

> **Unveiling the magnitude of the existing problem of endemic zoonoses is a crucial first step in increasing awareness about and action towards minimising the multiple impacts of these zoonoses.**



Raw animal products may be a source of exposure to zoonotic pathogens. Maasai woman milking a cow in northern Tanzania. Tiziana Lembo. University of Glasgow.

Like many other zoonoses, rabies exerts substantial economic burdens. The estimated overall economic cost of canine rabies is ~8.6 billion US Dollars (USD) per year. Much of this cost (2.27 billion USD) arises from lost economic productivity – a result of the premature deaths caused by this disease. Following a bite by a suspect rabid animal, the administration of post-exposure prophylaxis (PEP) can save lives. However, direct global expenditure on PEP is approximately 1.7 billion USD, with a further 1.3 billion USD lost from the incomes of those travelling for multiple doses of PEP, which often has poor local availability.

We have the tools to control zoonoses

The wider value to controlling zoonoses is in the multiple health and wealth

Diseases of animal origin pose increased risks to human health in areas where people and animals live in close proximity. Pastoralist farmer herding cattle in Tanzania. Tiziana Lembo. University of Glasgow.

gains for both populations at risk and governments. The maximum benefits are realised when preventive strategies tackle the disease problem in animal reservoir populations, and so address the problem at the source. The tools exist to prevent animal infection with many zoonoses – vaccination and mass treatment approaches have been used successfully to control zoonoses in higher income countries.

Preventing human African trypanosomiasis by treating cattle can provide substantial benefits in terms of human lives and treatment costs saved, as well as improved livestock productivity. For rabies, investment in the control of disease in domestic dog populations – by mass vaccinations – saves human lives and can eliminate rabies as a human health problem entirely. This is very well illustrated by the situation in Latin America and the Caribbean, where dog vaccination programmes, regularly implemented since the 1980s, have reduced human deaths by >99%.

Conclusions – seek and you shall find

The relative impacts and importance of different zoonoses vary between different countries and regions. Communities that keep a range of animal species are vulnerable to different sets of pathogens. However, it is invariably the case that when efforts are made to quantify the prevalence and impacts of one or more zoonoses in communities with close human–animal associations, substantial disease burdens are revealed.

Many tools to control endemic zoonoses exist, and have been applied successfully in higher income countries. Unveiling the magnitude of the existing problem of endemic zoonoses is a crucial first step in increasing awareness about and action towards minimising the multiple impacts of these zoonoses. By revealing and adding up the true burdens of endemic zoonoses we strengthen the case for sustained efforts to reduce the global impacts of these zoonoses, on both humanitarian and economic grounds.

Acknowledgements

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Can we identify potential viral zoonoses before they cross the species barrier?

Hanna Jerome, Sreenu B. Vattipally & Emma C. Thomson

The toolkit for the identification of zoonotic viruses has evolved substantially over the last century. In the early days of pathogen research, the presence of a viral pathogen was suspected if filtered bodily fluids could be used to infect experimental animals (filtration was used to remove bacterial pathogens). Subsequently, serological assays, *in vitro* culture in cell lines, such as Vero cells, and the polymerase chain reaction (PCR) were added to the armamentarium.

The field is now being revolutionised by next generation sequencing (NGS), an extremely powerful technique that can be used to generate millions of sequence reads in a matter of hours. As little as 1 ng of DNA can be used to identify multiple pathogens in a sample, without the need for specific primer-based amplification. Since NGS became routinely available at the start of the millennium, the number of new viral sequences deposited in the NCBI database has risen exponentially (Fig. 1).

History of the discovery of zoonotic viruses

In the 19th century, Louis Pasteur realised that rabies was caused by an agent that could not be visualised using a light microscope. Dmitry Ivanovsky and Martinus Beijerinck later showed that fluid passing through a porcelain Chamberland filter (Fig. 2) contained an agent infectious to tobacco plants. Beijerinck called this agent a virus although it was not until the 1930s that the structure of the tobacco mosaic virus was described using electron microscopy and X-ray crystallography.

During the 20th century, inoculation of experimental animals and tissues with filtered materials from infected individuals allowed scientists to

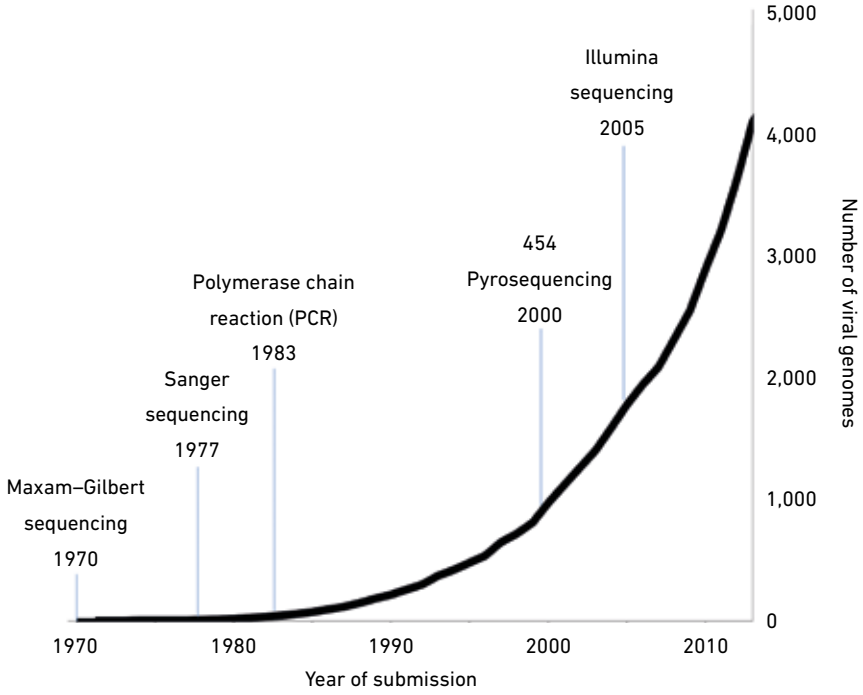

Fig. 1. Number of viral genomes submitted to NCBI 1970–2014.

postulate that viruses were responsible for several zoonotic infections. In 1927, blood samples from humans with yellow fever were used to infect *Macacus sinicus* and *Macacus rhesus* monkeys (this was the first rhesus monkey experiment). These were further passaged *in vivo* by inoculation of filtered plasma or exposure to *Aedes aegypti* mosquitoes. The likely viral aetiology of yellow fever was solved in this series of experiments, lasting only three months. Sadly, the senior author of the paper, Adrian Stokes, was accidentally infected during the study and died of yellow fever prior to publication of the report. Shortly after this, Rift Valley fever was identified in sheep in a farm in rural Kenya. Dilutions of plasma filtered through Berkefeld and Chamberland filters were found to infect sheep, cows and a goat experimentally (but not horses or pigs), even after 54 days of storage. In this study, all the investigators also subsequently became infected and, in an experiment reflecting the ethical standards of the day, a deliberate infection of a patient suffering from recurrent malaria was carried out to see if he would be cured; he was not - he developed Rift Valley fever and then relapsed shortly afterwards with severe malaria. In both of these studies, Koch's postulates were fulfilled for the first time for viral infection. However, sequencing of the viral genomes of these organisms was not carried out until 1985, using cloning and chemical sequencing.

In vitro techniques for culturing viruses were developed in the middle of the 20th century. Vero cells derived from *Chlorocebus* African green monkey kidneys became available in 1962 and Lassa fever was one of the first zoonotic viruses to be identified using Vero cell culture in 1970 by Buckley and Casals. The presence of a virus was confirmed by filtration and *in vivo* infection in experimental mice (rodents of the *Mastomys natalensis* species were later found to be the natural reservoir; Fig. 3). By this time, a number of additional techniques were available to confirm infection. Serological assays including complement fixation and neutralisation assays were carried out, and the virus was additionally visualised by electron microscopy. Two of the laboratory workers in Buckley and Casals lab at the Yale Arbovirus Research Unit (YARU) in New Haven became infected and one died of the illness.

Fig. 2. Chamberland–Pasteur filter. Courtesy of the Division of Medicine & Science, National Museum of American History, Smithsonian Institution

NGS is emerging as a powerful technique for the identification of infectious pathogens and has great potential as a surveillance tool.

Sequences of viral pathogen genomes were not commonly published until the 1980s. HIV infection was sequenced at the time of discovery by Francoise Barré-Sinoussi and colleagues although the sequence itself was not actually included in the first report. Later virus discovery publications have almost always described the viral genetic sequence. When related genomes have been available, a common approach has been to amplify viral genomic segments using random or conserved PCR primer sets from multiple virus families. Sin Nombre virus was identified in clinical samples and *Peromyscus maniculatus* deer mice after an outbreak of severe respiratory illness in patients in the USA in 1993 using conserved hantavirus PCR primers. In 1994, PCR-based sequencing using conserved primers from the *Paramyxoviridae* family allowed identification of Hendra and Nipah viruses. Hendra was identified during an outbreak of severe neurological and respiratory illness in horses and stable workers near Brisbane and an outbreak of encephalitis in humans and respiratory illness in pigs in Malaysia was found to be associated with the closely related but distinct Nipah virus. This use of conserved primers is based on a best guess of which primer sets to use to detect an unknown pathogen and requires that these bind to conserved regions of viral genetic material present in infected samples. The advent of unbiased next generation sequencing (sequencing of all the RNA or DNA in a sample including human- and pathogen-derived DNA and RNA) that is not based on the presence of specific PCR primers means that many more pathogens are likely to be discovered over the coming years.

Fig. 3. The multimammate mouse *Mastomys natalensis*. Tom McHugh/Science Photo Library

Next generation sequencing

One of the first emerging zoonotic viral pathogens to be identified using unbiased NGS was Lujo virus, following an outbreak of viral haemorrhagic fever in five individuals (an index case from Zambia, a paramedic, two nurses and a cleaner). Within 72 hours, a new arenavirus was identified. This was one of the first studies to harness the power of NGS to make a rapid diagnosis during an outbreak. Unbiased Illumina NGS during the recent outbreak of Ebola virus in West Africa also resulted in the rapid publication of multiple full genome sequences, contributing hugely to the international effort to develop a vaccine and other therapeutic interventions.

Predicting the next epidemic

NGS is emerging as a powerful technique for the identification of infectious pathogens and has great potential as a surveillance tool. It has been estimated by scientists at Columbia University that as many as 320,000 viruses in mammals remain undiscovered. In a single study, 58 new viruses from nine virus families were identified in a single species (the Indian

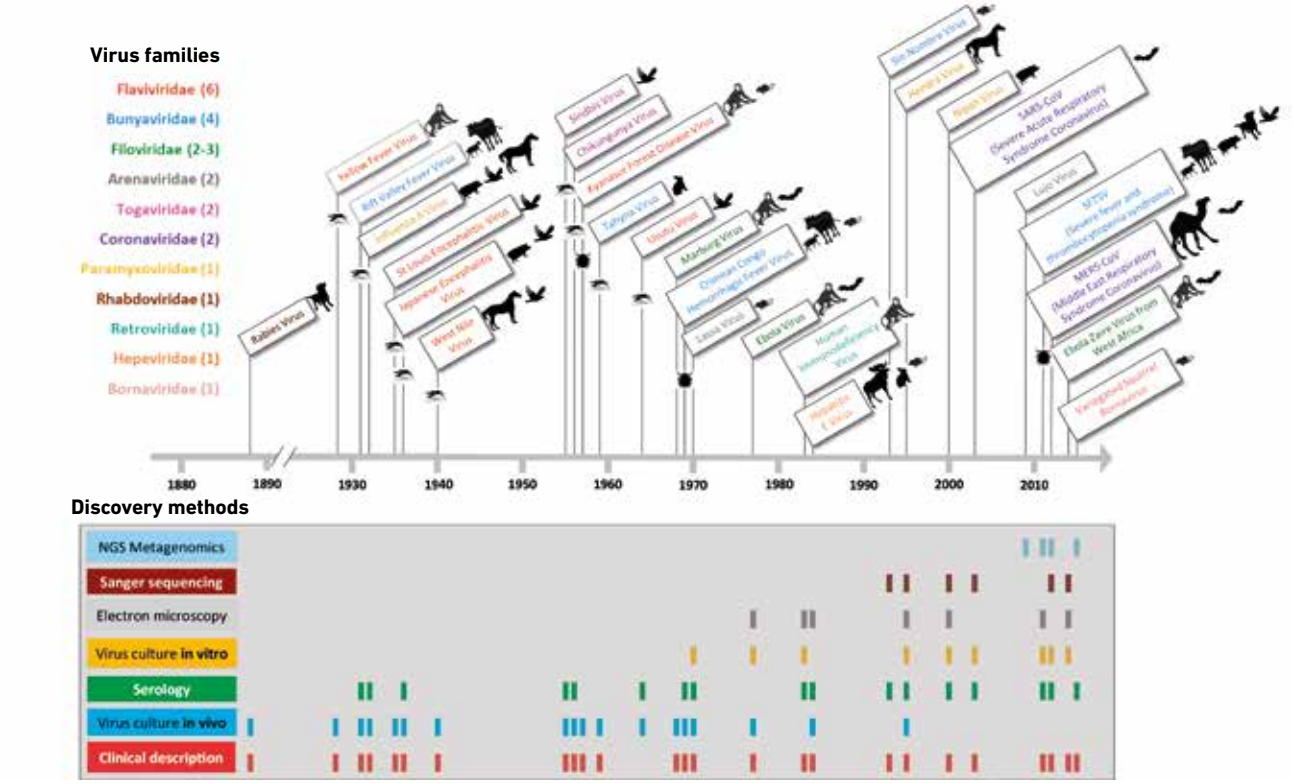

Fig. 4. Examples of zoonotic viruses and method of discovery over time. Hanna Jerome, Vattipally B. Sreenu & Emma C. Thomson

flying fox bat *Pteropus giganteus*). The majority of emerging viral pathogens are zoonotic and future research now aims to identify viruses in the zoonotic pool before they transit into human populations. This pool is deep, however, and much work remains to be carried out. While some investigators aim to target outbreaks of disease causing deaths in wildlife, zoonotic pathogens such as Ebola virus may not cause disease in their natural host, so predicting the possibility of spillover events before they happen will require surveillance of high-risk reservoir hosts such as bats. Such events are more likely to occur in association with changes in environmental equilibrium; deforestation, for example, may have contributed to the recent Ebola outbreak in West Africa, resulting in increased contact between bats and humans.

Several ongoing studies such as the Wellcome Trust VIZIONS project are now attempting to identify pathogens present in animal reservoirs that have the potential to transfer across the species barrier into humans. Combining high-throughput NGS data with functional virological assays, for example, whether or not a cognate receptor is present in humans, may help us to predict which viruses present a significant risk to the human population. Canine distemper virus, as an example, has crossed from dogs into several other species including seals, tigers, foxes and non-human primates but has not entered the human population despite the presence of the receptor in human cells. Many other potential pathogens await discovery.

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Acknowledgements

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Zoonotic diseases are defined as infections that are transmitted from vertebrate animal sources to humans, sometimes by way of an arthropod vector. Approximately 60% of infectious human pathogens are zoonotic. These diseases can be caused by viruses, bacteria, fungi and other parasitic organisms.

Hantaviruses – neglected zoonoses

Agnieszka Szemiel

The most commonly mentioned zoonotic viruses are influenza, rabies, severe acute respiratory syndrome (SARS), and more recently, Middle East respiratory syndrome (MERS) and Ebola. However, there are many other emerging viral zoonoses, some of which belong to the largest RNA virus family, the *Bunyaviridae,* comprising over 350 known isolates. The family is classified into five genera, *Orthobunyavirus*, *Nairovirus*, *Phlebovirus*, *Hantavirus* and *Tospovirus.* The first three are maintained in nature by a propagative cycle involving blood-feeding arthropods (such as mosquitoes, ticks, midges) and susceptible warm-blooded vertebrate species, and include a number of human and animal pathogens such as La Crosse virus, Oropouche virus, Schmallenberg virus, Crimean-Congo hemorrhagic fever virus and Rift Valley fever virus. The fifth genus, the tospoviruses, are important plant pathogens.

Bank vole, taken at British Wildlife Centre, Newchapel, Surrey. Peter Trimming

Hantaviruses are the only bunyaviruses that are direct zoonoses. They are maintained in the environment as persistent infections of rodents (voles, field mice, rats), which cause little damage to their rodent vector. They are transmitted to humans via aerosolised rodent urine, faeces, saliva, and occasionally by bite. Infection in humans can be fatal. Based on symptoms, hantaviruses are classified into two groups: the Eurasian Old World hantaviruses, which cause haemorrhagic fever with renal syndrome (HFRS) and are associated with approximately 0.1–15% fatality; and the New World hantaviruses with almost 40% fatality, causing Hantavirus cardiopulmonary syndrome (HCPS) in the Americas.

A brief history

Although formally described half a century ago, history suggests that hantaviruses have been around for much longer. A disease with symptoms resembling HFRS was mentioned in a Chinese medical book dating back as early as 960 AD. Although there is no direct evidence, English sweating sickness, a mysterious illness that struck England in the 15th century, is consistent with a hantavirus infection. There were five major epidemics of sweating sickness in England recorded between 1485 and 1551. The disease must have already been known earlier, as Lord Stanley, an influential Welsh Lord, used sweating sickness as an excuse to withdraw from the Battle of Bosworth Field in the Wars of the Roses in 1485. The outbreaks occurred during summer months, preceded by harsh winters and periods of prolonged rainfall, which probably resulted in spikes in the numbers of small insectivore mammals. This is consistent with current observations of hantavirus epidemics, which coincide with fluctuations in rodent populations. Looking back at the English sweat, Seoul hantavirus and its vector, the black rat (*Rattus rattus*), a common rodent in Europe, could have been the culprit.

In 1718, another similar, but less lethal, infection was recorded in France and, later on, around Europe. It was named 'Picardy sweat' after the region where it first occurred. It was also referred to as military fever. Outbreaks mainly occurred in rural areas, and were subsequently linked to fluctuations in field rodent populations. Some scientists suggest that Picardy sweat could have been caused by Puumala virus or similar, which still occurs in these regions.

Hantavirus disease was also suggested to be the causative agent of many cases of epidemic nephritis during the American Civil War and World War I. The first clinical descriptions of HFRS can be found in hospital records from 1913 in eastern Russia and in 1932 the disease was linked to exposure to rodents. During World War II outbreaks of nephritis among German troops on the Eastern front and in Finland, where hantaviruses are endemic, were classified as rodent-borne. During the Korean War in the summer of 1951, an

outbreak of haemorrhagic fever with renal syndrome struck United Nations troops. Later the pathogen was identified as a virus and named Hantaan after the river where the wild rats harbouring the virus had been captured. In the 1980s, based on the genome analyses, the group of viruses causing HFRS were assigned to the *Bunyaviridae* family as the *Hantavirus* genus. In the summer of 1993 the first cases of HCPS were recorded in the Four Corners region of the USA. The disease was caused by a new hantavirus later called Sin Nombre virus, which was spread by deer mice.

Hantaviruses in the UK

Several hantaviruses circulate in Europe: Seoul (carried by rats), Puumala (bank vole), Tula (common vole), Saaremaa and Dobrava (both in wild mice).

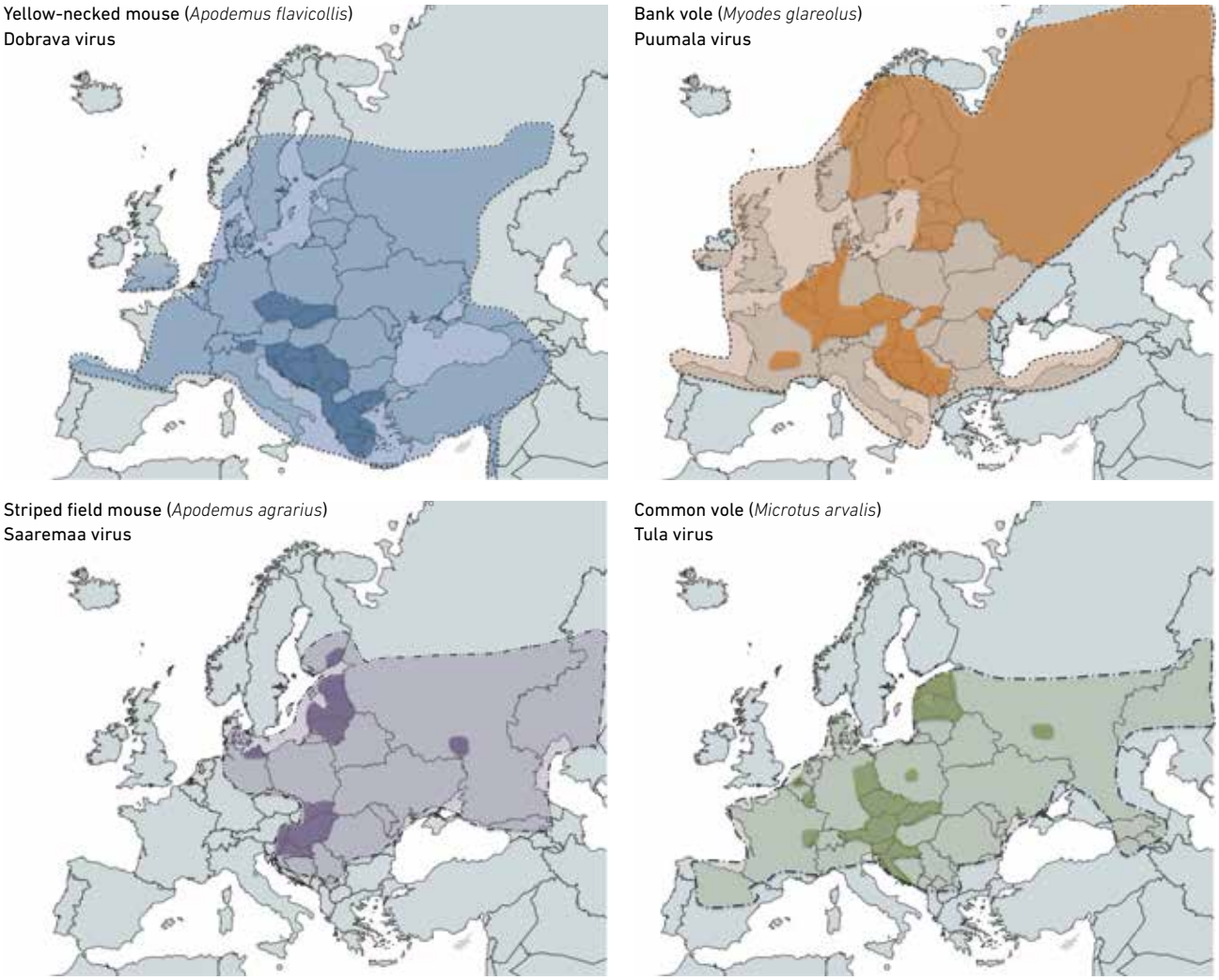
Hantaviruses very rarely appear in the news headlines in the UK. Also, when we look at distribution maps of hantaviruses in Europe, the UK is one of the few countries with little or no epidemiological data. General consensus is that HFRS is under-diagnosed in the UK, as, unless the patient develops kidney failure, the disease demonstrates as flu-like symptoms, and may easily pass unnoticed. Recent studies by Public Health England showed that, although they don't pose a major threat, hantaviruses are widely spread in the UK. Sero-surveillance studies showed that over 30% of pet rat owners had antibodies against hantaviruses, suggesting previous infection. In the case of occupationally exposed people like farmers, sewage and waste disposal workers, veterinarians, pest control, forestry and nature conservation workers, only 1–3% were sero-positive, which was similar to the numbers obtained for the general public. There are no extensive studies on the percentages of pets and wild rodents persistently infected with hantaviruses. Though whenever human cases of HFRS are diagnosed, animals in the surrounding environment test positive. In the last 4 years there were a few pet rat acquired cases of Seoul hantavirus which had to be hospitalised in North Wales, Yorkshire, Humberside and Scotland. On the other hand, studies of rodents trapped in the Tattenhall area, in Cheshire, showed the existence of a novel hantavirus, which is related to Puumala and Tula hantaviruses, which was previously not detected in the UK. Interestingly, rodents from the *Arvicolinae* family, that spread Puumala and Tula hantaviruses, are also found across mainland UK and Ireland. Looking at distribution maps of rodents that are known hantavirus vectors, it is striking that there have been so few confirmed hantavirus cases in the UK.

Future outlook

Although hantaviruses don't pose an immediate threat to the UK, rodent surveillance studies should be carried out on a regular basis. Some viruses, including hantaviruses, can spread to new areas, including the UK, and are associated with climate and environmental changes. With a growing human population and increasing urbanisation, natural habitats of indigenous rodents are disrupted, which causes changes in behaviour and animal migration. Change in land use may lead to the emergence of viruses previously not known or not common in certain environments. Such changes may also put pressure on pathogen evolution, which could result in increased

Although hantaviruses don't pose an immediate threat to the UK, rodent surveillance studies should be carried out on a regular basis.

Pet rat. Artur Malinowski



Geographical distribution of European hantaviruses and their vectors. Light-coloured areas indicate distribution of the rodent host species, and the darker coloured areas show where appropriate virus has been found. Areas not filled indicate that there is either no data available or neither host nor virus is present. Adapted from Olsson *et al.* (2010), *Vector Borne Zoonotic Dis* **10**, 549–561

pathogenicity or even switching to a different vector.

In most cases European hantavirus infections result in a mild form of the disease. However, with easy access to cheap international travel, there is a risk of importing more dangerous hantaviruses and other pathogens into the UK. For example, last year a rare case of hantavirus cardiopulmonary syndrome (HCPS), caused by Choclo virus, was diagnosed in a UK resident who returned from Panama. Similarly, pathogens could be imported with exotic pets.

Currently there are no vaccines or antivirals available to combat hantaviruses, as the molecular biology of hantaviruses in not very well studied. Advances in diagnostics can lead to the discovery of novel viruses, but it also allows for the detection of previously mis- or under-diagnosed pathogens. Hantaviruses are just one example of emerging diseases that present an increasing threat to human health. Like the hantaviruses, there are many other zoonoses lurking around that are worth studying or still await discovery. In our rapidly changing world it is evermore important to keep up surveillance.

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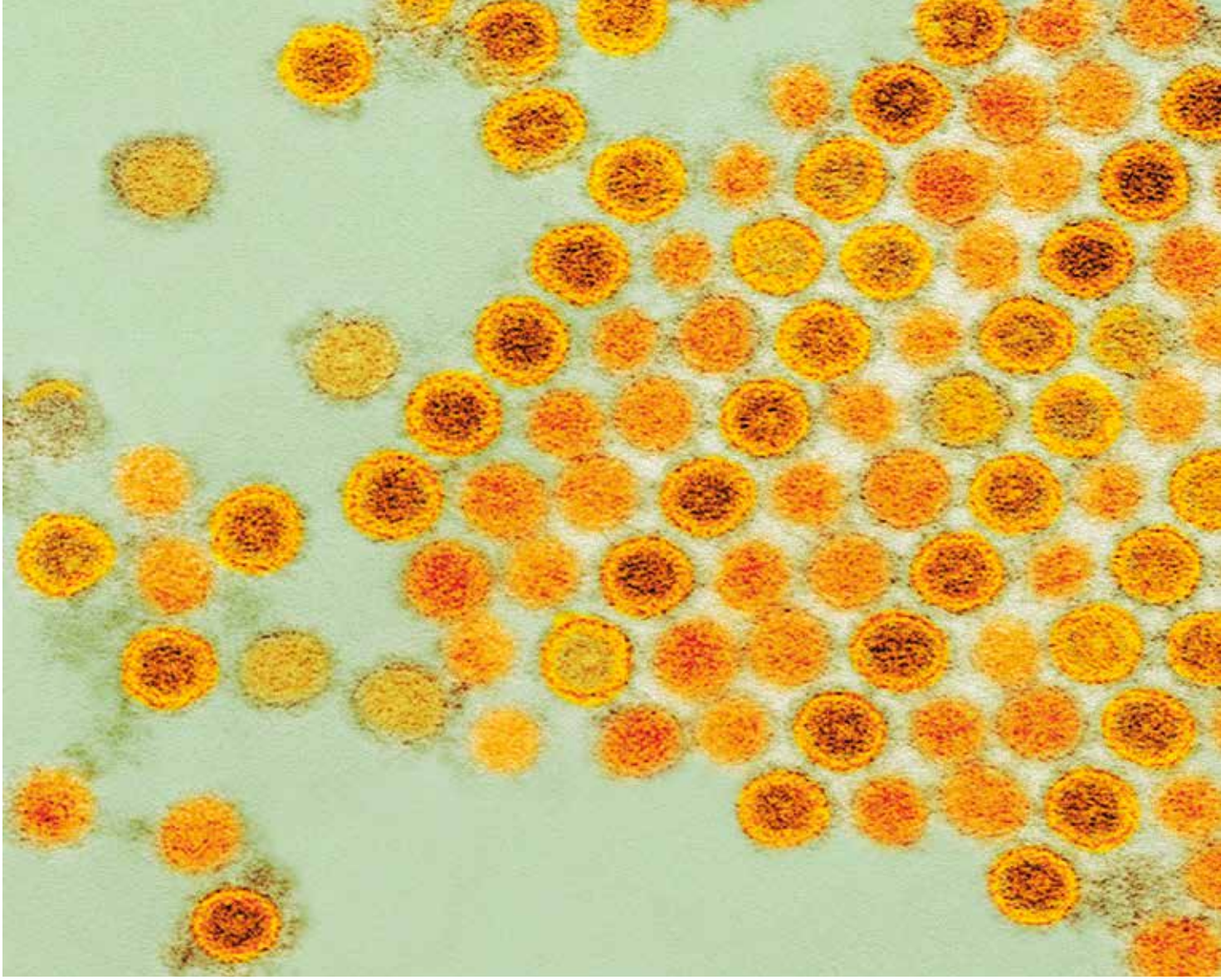
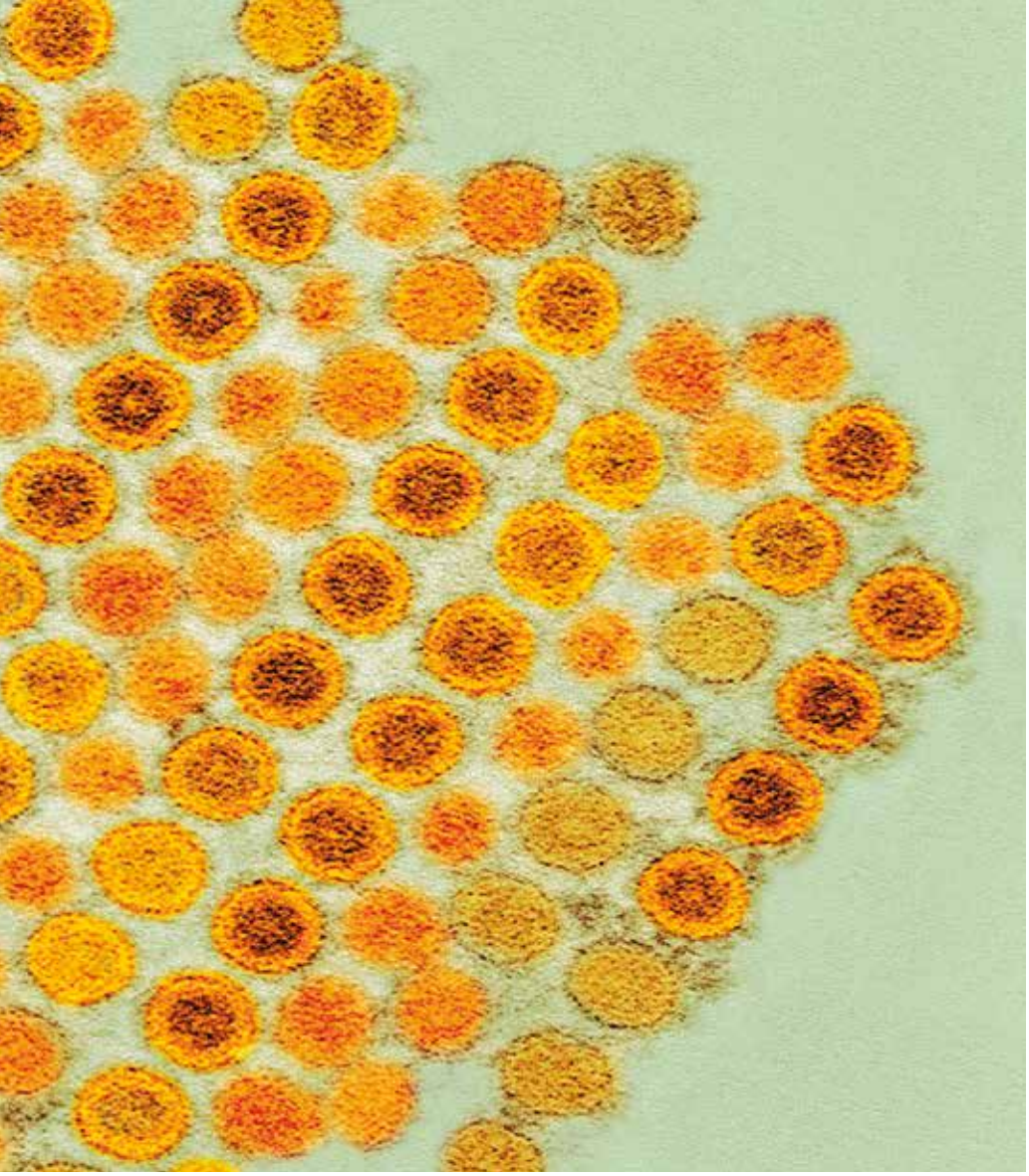


John Fazakerley

Chikungunya: a decade of global pain

It is has been 10 years since chikungunya emerged from the forests of Africa and started its global journey, from Africa out across the islands of the Indian Ocean to India and Southeast Asia, China and the Americas. The virus first emerged to prominence early in 2005 on the French island of La Réunion.

La Réunion, an island off Madagascar in the Indian Ocean, is part of France and has a population of around 750,000. In 2005, a third of the population became infected with chikungunya virus and there were 237 deaths (approximately 1 per 1,000 cases). Healthcare workers and scientists were rushed to La Réunion from France to deal with the outbreak.



Coloured transmission electron micrograph of chikungunya virus particles.
CDC/Cynthia Goldsmith, James A. Comer and Barbara Johnson/Science Photo Library

There are three major strains of the virus: East Central South African (ECSA), West African and Asian. These differ in their geographical distribution, transmission by mosquitoes and genome sequence. Chikungunya virus is classified as an alphavirus within the family *Togaviridae*. These are small, enveloped positive-sense RNA viruses. Related viruses include Semliki Forest virus, Sindbis virus, Ross River virus and Venezuelan equine encephalitis virus. Chikungunya, Sindbis and Ross River viruses are often grouped as 'Old World alphaviruses' since, until recently, they were not present in the Americas. The Old World alphaviruses generally cause arthralgia (joint pain). The 'New World alphaviruses' found in the Americas include Eastern, Western and Venezuelan equine encephalitis viruses. These are more dangerous; they cause encephalitis (brain inflammation) and can be fatal.

Transmission

Chikungunya virus is transmitted by mosquitoes of the *Aedes* genus, specifically *Aedes aegypti* and *Aedes albopictus*. These two mosquitoes are now widely spread around the world. *A. albopictus*, the Asian tiger mosquito, was disseminated from South-east Asia through international trade in houseplants and tyres in which mosquito larvae survive in pools of water. These two mosquitoes transmit both chikungunya and dengue and, as they prefer to bite humans, they are very effective disease vectors and are responsible for most cases of chikungunya. Generally, at least for the ECSA strain, *A. aegypti* is the vector in urban areas and *A. albopictus* is the vector in suburban and rural areas. In the 2005 epidemic in La Réunion, a point mutation in the E1 virus envelope glycoprotein of an ECSA virus increased the ability of this virus to replicate in *A. albopictus*. This almost certainly contributed to the massive outbreak on La Réunion. The Asian strain of the virus, currently circulating in the Americas, predominantly in the Caribbean, is transmitted preferentially by *A. aegypti*. Differences in the genome between ECSA and the Asian strain seem to be preventing the same point mutation in the E1 glycoprotein adapting the Asian strain to transmission by *A. albopictus*.

Chikungunya derives its name from an outbreak of the disease in Tanzania in the 1950s. The name derives from the local language Makonde and translates as 'that which bends up'. It refers to the fact that people with chikunguyna, due to pain in their joints and muscles, have a stooping, bent posture. The majority of infections are symptomatic and the incubation period is 2 to 7 days. Chikungunya causes an abrupt onset severe fever and debilitating joint and muscle pains, which are generally asymmetric and affect the ankles, wrists, fingers, knees, shoulders and elbows. Frequently there is also a rash. Headache, fatigue, nausea, conjunctivitis, and in newborns, meningitis and encephalitis, have also been described. The acute disease generally lasts for a couple of weeks. The differential diagnosis is dengue fever. Indeed, it is likely that some of the epidemics of

fever, described since colonial times throughout the tropics as dengue fever, were caused not by what we now term dengue virus but by chikungunya virus.

Structure and pathogenesis

We know relatively little about the molecular virology of chikungunya. In contrast, other alphaviruses have been much studied for many decades, particularly Semliki Forest virus, a close relative of chikungunya virus, Sindbis virus and Venezuelan equine encephalitis virus. The alphaviruses have relatively few proteins. A polyprotein is processed into four functional proteins, which together act as the virus replicase replicating the virus RNA and transcribing a subgenomic RNA. The subgenomic RNA provides an amplification mechanism for producing large numbers of viral structural proteins, a capsid protein and two or three virus envelope glycoproteins. The envelope glycoproteins mediate entry into cells and are determinants of host range and transmission. As with many, perhaps all, RNA viruses, in order to replicate in mammalian cells, the virus must suppress the interferon response. For the alphaviruses, non-structural protein 2 (nsP2) is responsible for this. The alphavirus capsid and nsP3 proteins also have important roles to play in modulating the functions of mammalian cells. For the mosquito vectors, as for all invertebrates and plants, the key suppressor of virus replication is not interferon but RNA interference (RNAi). Arboviruses, at least most of them, do not, or do not strongly, antagonise the RNAi response resulting in a low-level persistent infection with minimal fitness cost to this virus distribution system.

We also know relatively little about the pathogenesis of the human disease. There is a transient high titre viraemia (virus in the blood), which peaks 1 to 2 days post-infection and can last for a week. This drives acute inflammatory and immune responses with most commonly high levels of interferon-alpha and interleukins-1, -6, -12 and -18. The virus is observed in monocytes in the blood and in fibroblasts and macrophages in tissues. Interestingly, macrophages are infected by the uptake of apoptotic debris from virus-infected cells, not by purified virus. Whereas the acute infection, as characterised by fever, generally resolves within 2 weeks, the polyarthralgia generally lasts longer; one study estimated a mean duration of 90 days. For some people there is chronic disease with long-term arthralgia and in some cases

■, **Countries and territories where chikungunya cases have been reported (as of 10 March 2015). Does not include countries or territories where only imported cases have been documented. This map is updated weekly if there are new countries or territories that report local chikungunya virus transmission.** Taken from Centers for Disease Control and Prevention/National Center for Emerging and Zoonotic Infectious Diseases (NCEZID)/Division of Vector-Borne Diseases (DVBD)

arthritis. The immunological findings in these patients are different to those of rheumatoid arthritis with no C-reactive protein, no rheumatoid factor, a different cytokine profile in the blood and joints and no evidence of autoimmunity. Anti-chikungunya virus IGM antibodies can persist for years, indicative perhaps of a persistent infection. Consistent with this, in one case, virus RNA and proteins were detected in synovial macrophages 18 months post-infection. We know that in mouse models alphaviruses can persist in tissues for long periods of time. It would not be surprising if chikungunya virus can do so in a tissue such as the joint. Studying this is not easy for ethical reasons and most people with joint pain are sensitive to having their joints biopsied. Close cooperation between virologists and surgeons undertaking elective surgery in locations which have had many cases of chikungunya might further inform the pathogenesis of the chronic disease. The chronic joint disease in chikungunya raises the interesting question; could viruses be responsible for a subset of arthritic diseases worldwide?

As with many infectious diseases, an understanding of the pathogenesis is greatly facilitated by the study of animal model systems. Mice are not easily infected with chikungunya virus but inoculation close to a joint results in local joint swelling. Infection of mice with poor type-I interferon responses has been studied as a more disseminated model of infection. Macaque monkeys experimentally infected with chikungunya provide a better model of the human disease. As with humans, acute disease is characterised by a high-level transient viraemia with virus in monocytes, rash, severe fever and joint swelling with persistence of virus RNA and proteins in lymphoid tissues and liver.

It is estimated by the European Centre for Disease Prevention and Control that within a few decades the range of the *Aedes albopictus* vector could extend across northern Europe into southern Britain.

The outlook

At present there are no licenced vaccines for chikungunya though several candidates have been shown to be efficacious in pre-clinical testing including in macaques. There are vaccines based on attenuated chikungunya virus and other alphaviruses, virus-like particle systems, RNA and DNA constructs, and other engineered virus vector systems including vaccinia (MVA) virus and measles virus. At least three candidate vaccines have been through or are in Phase I human clinical trials. It should be relatively easy to protect against the acute disease since we know for most alphaviruses that good antibody responses correlate with protection. More difficult factors might be longevity of immunity, distribution in the tropics and cost per dose. Until the virus hit the Americas, developing a vaccine for this disease was generally not considered to be commercially viable.

In December 2013, the virus reached the Caribbean. Since then there have been an estimated 1.5 million cases in the Americas, mostly in the Caribbean but also in the countries in South America. As elsewhere, this has been an explosive epidemic in a naïve population. It will probably resolve with time and the virus will disappear back into the forests of Africa and Southern Asia, where it presumably persists in an arboreal cycle between monkeys and mosquitoes; whether the monkeys and mosquitoes of South America are also able to sustain this virus remains to be seen.

So far chikungunya cases have been recorded in 60 countries with, according to some estimates, over five million cases. Europe hasn't escaped the pain. There was an outbreak in Italy in 2007. A traveller from India introduced the virus into the Po river delta where there are plenty of mosquitoes. This resulted in local transmission and almost three hundred cases. Vector control by the local public health authorities brought the outbreak under control. In 2010, there were autochthonous cases in southern France. *Aedes albopictus* is now established around the northern shores of the Mediterranean and it is estimated by the European Centre for Disease Prevention and Control that within a few decades the range of this vector could extend across northern Europe into southern Britain.

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Campylobacter: breaking the spiral of infection

Helen Brown & Arnoud van Vliet

***Campylobacter jejuni* is an excellent example of a zoonotic pathogen, but for many years it was arguably the least known of the common bacterial food pathogens.**

Those of us who grew up in the UK in the late 1980s may remember the injustice of no longer being allowed to eat uncooked cake mix, due to the risk of *Salmonella* infection. More recently, many will recall headlines about the 2011 German *Escherichia coli* outbreak. *Campylobacter* very rarely makes the national headlines, although the number of *Campylobacter* cases have remained relatively steady over the last few decades, with an estimated annual incidence of ~280,000 UK cases.

In 2012, the BBC *Face the Facts* programme asked the public to name bacteria that cause foodborne illness. Not surprisingly, many people mentioned *Salmonella* and *E. coli*, some people even named *Listeria*, but *Campylobacter* was the great unknown. However, that picture is changing rapidly and in the past year the public profile of *Campylobacter* has been raised significantly, mainly due to the Food Standards Agency (FSA) publishing data on the percentage of *Campylobacter*-positive meat samples from retail sources. While insiders were not surprised by the levels of *Campylobacter* reported (64–79% of chicken meat was positive), the numbers were widely publicised, putting pressure on the poultry sector and retailers to address the problem.

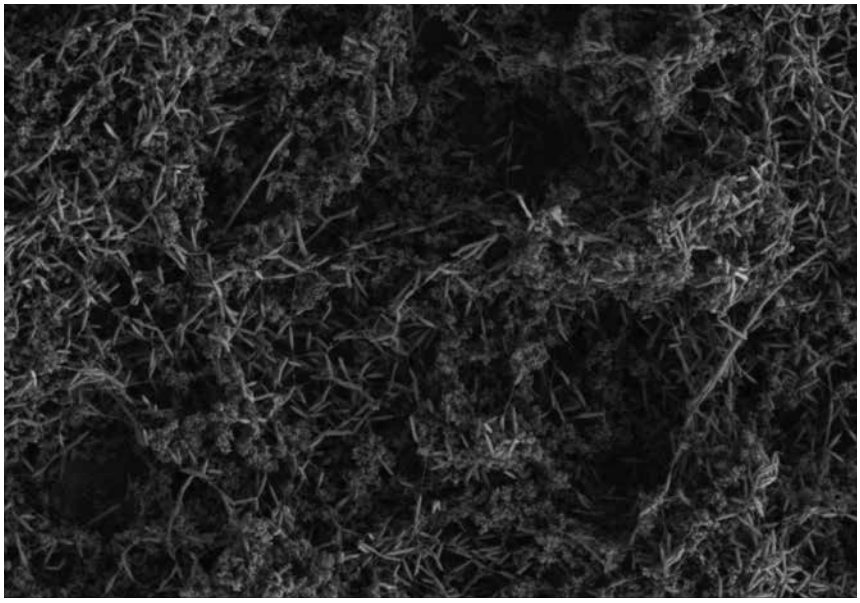
Why is tackling *Campylobacter* so important?

For such a common pathogen, it may seem surprising that there is so little public awareness. There may be several reasons for this: firstly, *Campylobacter* infection is typically a self-limiting illness. Symptoms usually last between 3 and 10 days and include fever, stomach cramps and diarrhoea, but rarely vomiting. Although infection can lead to potentially severe autoimmune

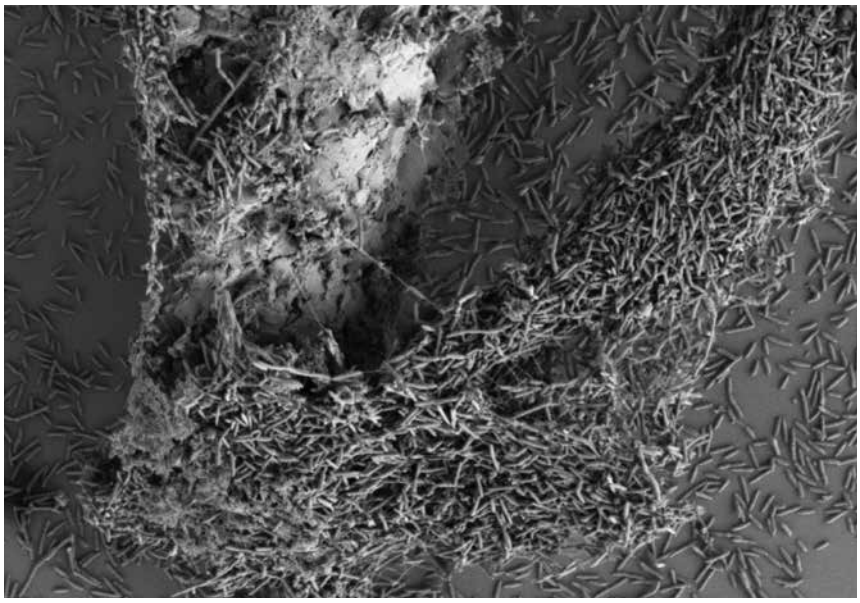
complications, these are rare and illness is typically self-resolving, requiring little professional attention. This means that the infection burden is economic rather than medical, costing an estimated £900 million each year in the UK, an amount the recovering economy could put to much better use! Secondly, *Campylobacter* is ever-present and hard to eradicate. Food safety measures implemented to reduce *Salmonella*, another zoonotic pathogen linked to poultry, have had little effect on *Campylobacter* levels. Birds show few symptoms when infected, so poultry producers have no outward signs of infection. Additionally, as a result of the rapid slaughter and processing methods, uninfected birds may become contaminated during processing.

The *Campylobacter* conundrum

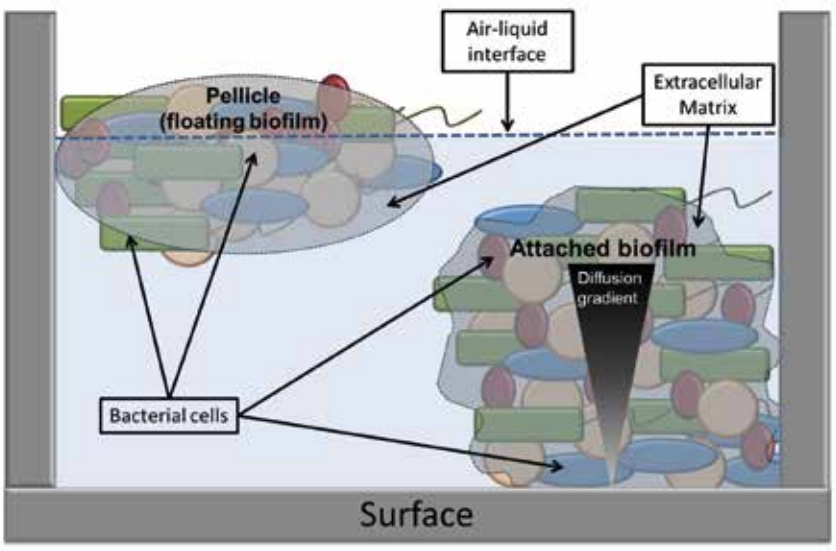
In addition to the *Campylobacter* retail survey, the FSA have been active with several public awareness campaigns, focusing on how people should handle raw chicken. The food industry has argued against the policy of 'naming and shaming', and to an extent we sympathise. *Campylobacter* has had many millions of years to learn its trade, and they are now expected to produce immediate results. *Campylobacter* is a fastidious organism, requiring a rich growth medium, a narrow range of temperatures and modified atmospheric conditions for growth. Researchers working with the bacterium have many anecdotes of how *Campylobacter* cultures left on the bench for a few hours die and how opening an incubator door one too many times led to the loss of a weeks' worth of experiments. Yet, despite this fastidious nature, *Campylobacter* is able to survive for extended periods within the food chain (a contradiction,



Scanning electron microscope (SEM) image of a *Campylobacter jejuni* biofilm forming on a soiled surface. The *Campylobacter* is able to attach to the soiling particulates more easily that it can to a clean surface, and so the biofilm can grow much quicker. Dr Louise Salt, IFR



In this SEM image, a *C. jejuni* biofilm has formed around a small wooden splinter. This splinter could not been seen by eye. This highlights that any surface imperfections or debris (however small) may help *Campylobacter* form a biofilm and improve its chances of survival both in the abattoir or on surfaces in the home. This is why thorough cleaning of hands and equipment after handling raw poultry is so important. Dr Louise Salt, IFR



Schematic representation of two different types of biofilm and key components. Biofilms can either be free floating (pellicles) or attached to a surface, typically near the interface between air and liquid. Biofilms are often comprised of several different types of bacteria and surrounded by a complex matrix which they produce themselves. Mature biofilms are usually highly structured, with a level of cooperation and metabolite sharing between the inhabitants. As a biofilm becomes more dense a diffusion gradient occurs, where the outer levels of the biofilm are more rich in nutrients and have atmospheric conditions similar to those found in the external environment. In deeper layers of the biofilm, the atmospheric conditions can be very different to those in the surrounding environment, potentially allowing species such as *Campylobacter* to survive in conditions that would quickly kill an individual cell. Equally, antimicrobials are less able to penetrate the deeper layers of the biofilm, meaning that at least some of the biofilm's population will likely still be viable after cleaning and disinfection routines are carried out.

which, within the field is nicknamed the '*Campylobacter* conundrum'). We still don't fully understand how this survival is achieved, or how to interfere with these mechanisms. This is not good news for poultry producers who serve the consumer demand for low-cost chicken, but want to avoid possible future government sanctions for non-compliance. The ACT (Acting on Campylobacter Together, #actonfarm on Twitter) campaign shows the drive of regulators and industry to solve this problem.

Where can we stop *Campylobacter*?

Campylobacter is commonly found in the intestine of birds and animals, and also survives in agricultural environments. Flocks commonly become *Campylobacter*-positive in the latter stage of their growth, and infection spreads rapidly within flocks. In the kitchen the bacteria either cross-contaminate other foodstuffs and equipment (one reason why you shouldn't wash your chicken!), or survive when the meat is improperly cooked. There are several points in the process where interventions may be successful. Firstly, preventing birds from becoming infected. This is currently attempted by strict biosecurity measures, but the FSA results show these measure are unsuccessful in their current form. Other interventions being trialled include feed additives and improved disinfection procedures. Secondly, contamination of meat may be prevented at the abattoir stage, and we discuss some ideas on how *Campylobacter* survives in that environment in the diagram above. Promising interventions include surface chilling, disinfectant rinses and combinations of steam and ultrasound. Finally, consumers can protect themselves with good kitchen hygiene and food preparation. Retailers are doing their bit, for instance with the 'double-bagged whole chicken', where consumers don't touch the chicken until it's fully cooked.

> **It should be stressed though that the consumer plays a central role in breaking the infection cycle and is firmly in control of their health.**

A potential *Campylobacter* survival strategy

Biofilms, formed of bacterial communities enveloped by an extracellular matrix, are an important mechanism for bacterial survival of suboptimal conditions. Typically, biofilms contain multiple bacterial species and can be dense enough to see with the naked eye. Reduced oxygen levels can be found in the deeper levels of biofilms, perfect for survival of microaerobic bacteria such as *Campylobacter*. Similarly, multispecies biofilms produce a wide range of metabolites, allowing many different bacteria to thrive by co-operatively producing the nutrients required by the community. There is now a large body of evidence indicating that *Campylobacter* is able to not only integrate into existing multi-species biofilms, but can form *de novo* biofilms on food chain relevant surfaces such as stainless steel. Surface soiling with meat juices or other organic matter increases *Campylobacter's* ability to attach to surfaces, increasing biofilm formation. We, and others, have shown that DNA

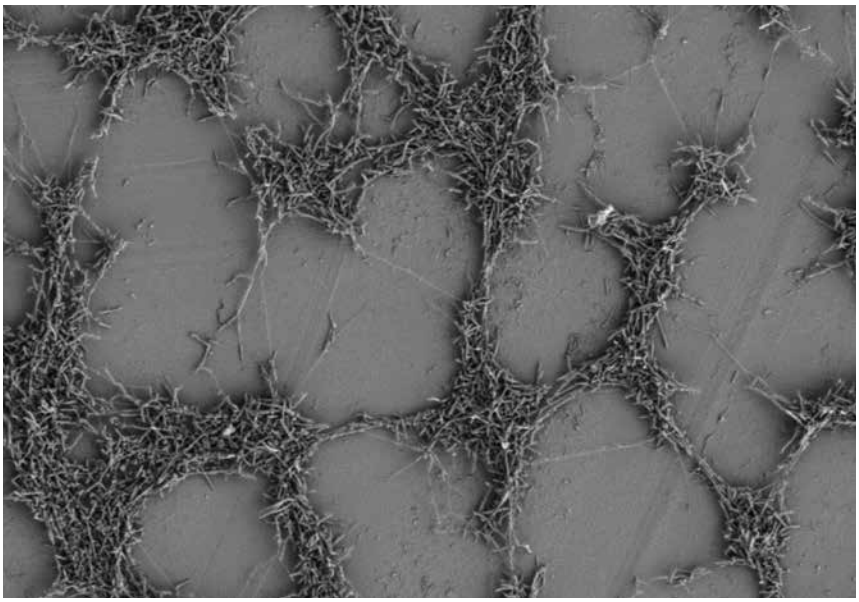
is an important component of the extracellular matrix, and its enzymic degradation allows rapid dispersal of attached *Campylobacter* populations. Motility also appears to be an important factor in biofilm formation, allowing bacterial cells to migrate towards, and away from, surfaces and helping to form surface attachments in the initial stages of biofilm formation.

The future of biofilm investigation

The majority of bacteria exist in mixed species biofilms, and we expect *Campylobacter* to behave no differently. Multi-species biofilms are, by their very nature, extremely complex, and this complexity has, until recently, hindered their study. Advances in 'omics technologies, *in silico* modelling and the development of techniques which allow single cell interactions to be studied are allowing us to better understand how multi-species biofilm communities are able to thrive. These new technologies should increase our understanding of *Campylobacter* and how it is transiting the food chain, and may lead to the development of knowledge-led *Campylobacter* eradication strategies throughout the food chain.

What should we do until then?

It should be stressed though that the consumer plays a central role in breaking the infection cycle and is firmly in control of their health. They must be aware of the risk posed by zoonotic pathogens such as *Campylobacter*. Consumers should be cautious when handling raw chicken and wash their hands and utensils immediately afterwards in order to ensure that cross contamination does not occur. *Campylobacter* does not tolerate freezing very well and so freezing chicken before consumption will reduce the number of viable bacteria on the meat. Washing raw chicken should be avoided, as contaminated water could be splashed onto surfaces or other foods. Fortunately, *Campylobacter* is also relatively easy to destroy by cooking, so ensuring that chicken is cooked adequately before consumption should be enough to stop infection. Following these simple guidelines should be adequate to ensure that you stay *Campylobacter*-free!



SEM image of a *C. jejuni* biofilm forming on a plastic surface. This picture highlights how structured and organised the cells within a biofilm can be. The long threads that link the microcolonies are the extracellular matrix. They look like threads because of the method of processing used to prepare the biofilms for microscopy. Dr Louise Salt, IFR

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Further reading

Acting on Campylobacter Together. Food Standards Agency. **http://microb.io/1JVwg0U**. *Last accessed 25 August 2015.*

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Publishing

A great start to the #MGeneration

The Society's new open access journal, *Microbial Genomics*, launched earlier this year. This article highlights some of the latest papers and gives an update on the journal's pioneering Open Data policy.

MICROBIAL GENOMICS

Bases to Biology

OPEN MICROBIOLOGY

OPEN DATA

Microbial Genomics (MGen) launched this summer with a selection of innovative published papers. These included an article on chromosome remodelling during a protracted MRSA infection, which Dr William Hanage, an Editor for MGen, summarised as "showing us a trick a drug-resistant pathogen used to make itself at home during a serious infection". He added that "these are the type of results we need genomic methods to find, to study, and ultimately to combat. *Microbial Genomics* is going to be the place to read and publish such research". The journal has since published a number of articles including a paper on the origin and dispersal of Shiga-toxin-producing *Escherichia coli* O157:H7, using phylogenetic methods to present a comprehensive population structure on this zoonotic pathogen for the first time. More recently, Cornick *et al.* presented the largest reported sequencing collection of a single *Streptococcus pneumoniae* serotype to date to show how country-specific selective pressures have driven the evolution and diversification of this important pathogen within Africa. MGen aims to reach a first decision on submitted papers within 4 weeks, with Accepted Papers published as the author version within 3 working days.

Microbial Genomics' Open Data policy leads the way in microbiological science

MGen provides the Society with its first open data publication, recognising the benefits to be gained from authors sharing their research data. In keeping with the journal's mission to support the discoverability and accessibility of research data, the Editorial Board – led by Professors Nicholas Thomson and Stephen Bentley, both at the Wellcome Trust Sanger Institute – decided that a mandatory open data policy would be necessary to achieve these goals. The journal's Open Data policy has been recognised as a leader in the field by the publishers of other microbiology journals. This includes the American Society for Microbiology, who are looking to follow MGen's lead and have taken guidance from the journal in developing their own policy for the publication of supporting data.

> **The ability to generate microbial genomic data at scale has not only facilitated rapid development in established fields, but has given rise to whole new branches of science.**
>
> Dr Kathryn Hall, Senior Editor, *Microbial Genomics*

Examples of data presented in MGen articles, such as large datasets, sequences and videos, that have been published through our data partner figshare.

> **Open data is what will help us to prevent the 21st century pandemics...**
>
> Jennifer Gardy, Senior Editor, *Microbial Genomics*

Many of the journal's authors have now taken advantage of MGen's agreement with the online repository figshare, to publish and link their data to the final article. Mark Hahnel, founder of figshare, said of the partnership: "We are delighted that the Society has chosen figshare to offer an enhanced data service to their authors and readers. Society publishers play a key role in the dissemination of academic content and MGen's Open Data policy puts them at the forefront of one of the most important movements in academia." The journal also provides seamless linking with a number of other repositories including GenBank, Microreact and GitHub. For more information go to **http://mgen.microbiologyresearch.org/opendata**

Parita Patel
Product Manager
p.patel@microbiologysociety.org

Microbial Genomics: Standing on the Shoulders of Giants


Lonely/Shutterstock.com

Introducing 'Standing on the Shoulders of Giants', which recognises the influential work of pioneering researchers in the field. The first published articles in this new feature include:


S. Falkow

Professor Stanley Falkow
Stanford University, USA
Described by many in the field as the 'father' of molecular pathogenesis.


G. Dougan

Professor Gordon Dougan
Wellcome Trust Sanger Institute UK
on Professor Stanley Falkow
A pioneer of enteric diseases and vaccine usage in developing countries.


J. Thornton

Dame Janet Thornton
Director of EMBL European Bioinformatics Institute, UK
One of the world's leading researchers in structural bioinformatics.

Go to **http://mgen.microbiologyresearch.org/sotsog** to read the full articles.

- *Microbial Genomics* is a gold open access journal, with all article processing charges waived during the launch period.
- Submit your article now at **http://mgen.microbiologyresearch.org**
- For more information, please email **mgen@microbiologysociety.org**
- Join the discussion on Twitter using **#MGen**

Conferences

Focused Meetings 2015

International Meeting on Arboviruses and their Vectors (IMAV)
University of Glasgow
7–8 September 2015

September was an exciting month for the Society as two incredibly successful Focused Meetings took place. The International Meeting on Arboviruses and their Vectors (IMAV) saw a high attendance with over 20 offered oral presentations and 30 posters. The prestige of the meeting was heightened by the calibre of invited speakers, such as Carol Blair (Colorado State University, USA) and Simon Carpenter (The Pirbright Institute, UK). Delegates came from 16 different countries to attend and network with influential microbiologists.

The topics covered included:

- arbovirus–vector interactions and immune responses
- preventing arbovirus transmission: novel strategies
- arbovirus–vertebrate host interactions
- vertebrate immune responses to arbovirus infection
- arbovirus replication and evolution

International Meeting on The Invasive Fungus
Mercure Hotel, Manchester
7–9 September 2015

The International Meeting on The Invasive Fungus was another success in the Focused Meeting series. Delegates enjoyed the opportunity to listen to and network with big names in their field such as Robin May (University of Birmingham, UK) and Dilip Shah (Donald Danforth Plant Science Center, USA). Over the two-and-a-half days in Manchester, delegates covered the following topics:

- hyphal tip growth
- tropisms
- invasion of animal plant tissues
- host defence responses
- invasion of ecological environments

The Focused Meeting on Industrial Applications of Metal–Microbe Interactions takes place at Charles Darwin House, London, UK, 9–10 November. Topics include biomining; biorecovery and bioprocessing; bioremediation; and biofabrication of higher value products.

2016 dates for your diary

Molecular Biology of Archaea 5
London School of Hygiene & Tropical Medicine, London
1–3 August 2016

Molecular Biology and Pathogenesis of Avian Viruses
Charles Darwin House, London
27–29 September 2016

To register your interest in these meetings, email: **conferences@microbiologysociety.org**

FIS 2015 update

Only a couple of weeks now until FIS 2015 takes place! The theme is Action on Infection and the event provides a fantastic programme, with world renowned speakers and many networking opportunities. The event is at Glasgow SECC between 21 and 23 November 2015 and it's not too late to register! Visit **http://actiononinfection.com/registration**

Grants opportunities

The Society offers grants to support eligible members presenting research at our Annual Conference, Focused Meetings and Irish Division meetings.

Society Conference Grants

Open to technicians, postdoctoral researchers or PhD student members and those attending their first Society meeting, whether presenting or not.

Undergraduate Student Conference Grants

These grants enable undergraduate student members to attend our meetings, and to cover travel and accommodation costs.

Inclusion Grants

Grants for members who wish to attend a Society meeting and cover interim caring arrangement costs.

Travel Grants

To support members who are not eligible for the above grants to present at any Society conference. Minimum membership period applies

For full eligibility criteria, please visit: **www.microbiologysociety.org/eventsfunds**

Shape our events programme

Our programme of events is developed and driven from proposals submitted by our members. There are three ways our members can help shape our programme to share research and build networks and communities.

Option 1 – Annual Conference Session

Our Annual Conference brings together a collective audience of over 1,200 delegates over a four-day event, providing an opportunity to share and develop research in all areas of microbiology. Proposals are welcomed from session organisers who wish to deliver a session at conference.

Option 2 – Focused Meetings

Each year the Society's Conference team oversees the running of three to four Focused Meetings, providing a forum for researchers and specialists to come together, share knowledge and build connections. These meetings attract over 100 delegates and often run over a two-day period. We welcome proposals from session organisers who have identified an area of research that will draw in delegates and provide a forum for networking.

Option 3 – Society Supported Conference Grants

To ensure that our members and the wider microbiology community have continuous access to a varied programme of events we regularly work in collaboration with other organisations and session organisers to sponsor UK and international speakers. If you have an event taking place and are looking for sponsorship you can apply for a Society Supported Conference Grant.

Decision-making

All proposals and applications are submitted to the Scientific Conferences Committee (SCC), which is made up of representatives from the Virology, Eukaryotic, Prokaryotic and Irish Divisions. This process ensures our conferences programme covers a broad spectrum of microbiology. Annual Conference sessions and Focused Meeting proposals are considered a year in advance.

Key dates

The SCC meets twice a year. Upcoming deadlines and notification dates for proposals are as follows:

Deadline:	14 December 2015	17 June 2016
Notification date:	15 February 2016	19 August 2016

For further information and application forms visit:
www.microbiologysociety.org/conferences

Annual Conference 2016

Our 2016 Annual Conference takes place between 21 and 24 March at the ACC Liverpool and once again we are providing a full programme covering a broad spectrum of microbiology, including:

- climate change and disease transmission
- Clinical Virology Network (CVN)
- DNA viruses
- environmental and applied microbiology
- evasion of host defences
- gene and genome manipulation
- host–pathogen interactions – nutrition, immunity, metals
- intrinsic antiviral immunity
- membrane transportation
- microbial interactions with insects
- mining microbial diversity
- mycobacteria
- negative-strand RNA viruses
- plant viruses
- positive-strand RNA viruses
- prokaryote cell biology / genetics / infection
- protein post-translation modification
- restriction factors
- retroviruses
- Small World Initiative
- the model microbe
- viral haemorrhagic fever

In addition to these sessions, we will have our five Prize Lectures at the start and end of each day, including the Prize Medal, Fleming Prize, Peter Wildy Prize, Marjory Stephenson Prize and Colworth Prize.

Plus, in 2016 all of this...

- an audience with the Prize Medal Winner
- Meet the Speaker sessions
- lively debates
- evening entertainment
- Live at Lunch sessions
- poster presentations
- networking opportunities
- trade exhibition

Registration and abstract submission are both open. Visit **www.microbiologysociety.org/conferences**

Outreach

The tale of the 13-week-old cheese and pickled egg sandwich

The 13-week-old sandwich. J. Verran

Manchester Children's Book Festival (MCBF) is an annual event run by Manchester Metropolitan University (MMU) championed by MMU Professor and Poet Laureate, Carol Anne Duffy. Every year, we try to shoehorn in some science – particularly microbiology – and we have used children's or teen novels involving vampires (*Twilight* by Stephenie Meyer), zombies (*Warm Bodies* by Isaac Marion) and smallpox (*Code Orange* by Caroline Cooney) to deliver messages around hygiene, disease transmission and vaccination.

This year, the guest author was Matt Brown, writer of *Compton Valance – The Most Powerful Boy in the Universe*. Imagine our pleasure when we found out that the book series was about a boy who nurtured a cheese and pickled egg sandwich in a shoebox for 13(!) weeks. This was an unmissable opportunity to repeat the experiment. However, after Compton and his friend sampled it(!!), the sandwich turned out to be magic, conferring time-travelling talents on the brave consumers. This we could not replicate – nor did we try to.

Jo Verran with writer Matt Brown. James Draper

It was an interesting experiment. I had no idea what would happen to a sandwich left for 13 weeks. I was worried that the pickled egg might prove particularly offensive, so I also made a cheese sandwich control. Interestingly, the vinegar pickling the egg actually delayed the rot for a couple of weeks. After about 8 weeks, there was a distinctly earthy odour, and by 13 weeks both sandwiches were considerably shrunken, crumbly and brown.

After appropriate risk assessments, we took the 13-week-old sandwiches (sealed in their boxes) to the MCBF Family Fun Day. Our message (apart from not eating the sandwich) was about fungi, their importance in deterioration and degradation, and their morphological diversity. We had some prepared slides and sealed plates for examination, alongside some information to take away about fungi, and about hygienic sandwich preparation and storage.

We were very busy! The target audience for the book would, I imagine, be around nine-year-olds. Families examined the sandwich, and sniffed the sealed box – from which odour still permeated. There were discussions around whether or not consumption of mouldy food would be undesirable or harmful. Children were keen to use the microscopes and parents took away information. Finally, as well as joining in with our teaser Twitter campaign, Matt Brown came to see the sandwiches and meet the scientists. That was exciting too!

The science continues: we are identifying the 'sandwichome' and screening sandwich isolates for antibiotic production to tie in with and complement the Small World Initiative (SWI) work to find antibiotics in soil. We have also partnered a school as part of the SWI, so the work with the sandwich was an interesting and relevant introduction.

Who would have guessed, without a glimpse into the future, that so much could be garnered from a time-travelling sandwich?

Joanna Verran

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Further reading

Bad Bugs Book Club. **www.hsri.mmu.ac.uk/badbugsbookclub**. Manchester Children's Book Festival. **www.mcbf.org.uk**.

Matt Brown, Writer. **http://mattbrownwriter.com/science-mirroring-art** *All last accessed 11 September 2015.*

Member careers: Vivian Moses

V. Moses

Looking back on a career in science, we asked long-standing member Professor Vivian Moses to give an overview of his career and work.

Towards the end of World War II, I was studying for my Higher School Certificate, the equivalent of A-Levels. I was keen on chemistry but that had caused a bit of a mess at home so I turned to biology, instead bringing home frogs and earthworms for dissection. With lots of enthusiasm for both subjects, I decided to combine the two when it came to thinking about a career.

Before starting university, and having passed the necessary exams, I continued at school 2 days a week and worked for three as a lab assistant at the Express Dairies on London's Euston Road. There I spent 9 months as assistant to a lady who sampled foods and food-processing machines, checking for microbial contamination. That gave me a good grounding of the main microbiological techniques before I went up to Cambridge in 1946 to read Biochemistry.

Cambridge was the only university at that time that offered an undergraduate Biochemistry course. The first 2 years focused on chemistry and physiology. The third year was devoted entirely either to animal or to microbial biochemistry: I was offered a place on the microbial course and so became a microbial biochemist.

It was not possible for me to begin a PhD immediately. National Service was still in force at the time: like other science graduates, my obligation would be fulfilled if I found a job 'of national importance that used my science degree' and held it for 2 years. So, with official approval, I became a lab scientist with J. Lyons (now Allied-Lyons PLC). The lab building was in Hammersmith Road near Olympia; I was in microbiology while upstairs in chemistry worked someone called Margaret Roberts (now better known by her married name of Margaret Thatcher) but I can't say I knew her.

Cream for ice cream was not then readily available so the product was based on partially liquefied flour. To prepare that, Lyons were buying in amylase but wanted to generate their own supply; my first project was to find how best to do so. Later I developed a replacement procedure for a disposal unit to remove residual fermentables from the waste water coming from Lyons' food products factory at Greenford before being discharged to the sewers; the system then in use was running at low pH and the equipment had become badly corroded. My aim was to develop a higher pH procedure for use in a rebuilt tank; I completed the development just before I left to begin my PhD at University College London (UCL) and heard later that it had indeed worked.

I was awarded my PhD in 1953 and stayed on at UCL for 3 years as a Junior Lecturer. Then I was very fortunate to secure a postdoc with Melvin Calvin at the University of California in Berkeley, joining his group on photosynthesis research (for which he was awarded the Nobel Prize in 1961) and other metabolic problems. After 2 years there my visa expired; I returned to the UK to another two-year postdoc on aspirin and rheumatism with Mervin Smith at King's College Medical School.

Tenured academic science jobs were difficult to come by in Britain in 1960: the first round of new universities were founded only in the early 1960s. So, when Calvin invited me to return to Berkeley on a permanent basis as a research director, I did so, concentrating first mainly on metabolic compartmentation and later on microbial enzyme synthesis, particularly catabolite repression of the lac operon system in *Escherichia coli*.

Including a sabbatical year (1967–1968) in Oxford spent with Joel Mandetstam and Michael Yudkin, I stayed in California until 1971 but eventually felt I would prefer to be in the UK and was fortunate to be appointed Professor of Microbiology at Queen Mary College. I held that position for 22 years (retiring in 1993), gradually moving towards biotechnology which has, indeed, kept me very busy in the years following retirement.

Highlight of my career

Perhaps the most exciting point in my career was my postdoc in California in 1956–1958. UCL was fine but it was nowhere near as lively as the Berkeley lab. Calvin was a very vigorous director who was an inspiration to us all; he always wanted to talk to everyone and all the members of that large multidisciplinary group of at least 30 people were interested in what everyone else was doing. Some possible research line might come up and that very day we might start to explore with colleagues who wanted to join in. There was enough funding so we could just ask for access to a chemical or piece of equipment. That was a most exciting environment for someone coming out of the rather darker England of the late 1950s.

Schoolzone

tfoxfoto/iStock

Teaching zoonoses in schools

Zoonoses are a fascinating topic for school students; the spread of diseases that can pass between animals and humans holds a gruesome appeal. But how can we teach about zoonoses in schools in an engaging way, when, for obvious reasons, we cannot carry out wet laboratory experiments using any of these pathogens? In this article we explore student activities covering various aspects of the subject, including emerging zoonotic diseases, biosecurity and the spread of infection.

Investigating emerging zoonotic diseases

An emerging zoonotic disease is one that is newly recognised or evolved, or that has already been seen in humans previously but has shown an increase in incidence or geographical area. Examples include Ebola, West Nile virus and avian influenza. They can be caused by any type of infectious agent (bacteria, virus, fungi, prion or protozoa) and can be spread by pets, farm animals or wild animals.

Student research project

This topic provides a good opportunity to use a multi-disciplinary approach, with students able to research several aspects of an emerging zoonotic disease. Important microbiological themes to consider include:

- What micro-organism causes the disease?
- What is known about the biology of this micro-organism?
- Does this microbe have a history of causing disease in other geographical locations, or in different species?
- How does this microbe transmit the disease? This could be through direct or indirect transmission.

As well as the microbiology behind each disease, the students can also investigate the epidemiology of the disease, anthropological, social and economic impacts of the disease, and what can be done from a public health perspective to minimise the spread of the disease. It can cover other topics in biology and chemistry, as well as history, geography and health studies. The results of this desk-based research could be presented as a poster, a presentation, or a policy briefing.

Mint Images/Science Photo Library

Biosecurity: preventing the spread of zoonotic diseases

It is a vital skill for developing scientific researchers to be able to write good quality risk assessments and consider appropriate codes of practice. However, this can sometimes be a tedious task and not particularly engaging for students. Using a subject, such as zoonotic diseases, that has serious consequences if correct procedures are not followed, adds a good level of interest to the work and helps students think outside of their usual laboratory situations.

Student workshop

Pairs of students are given a specific environment to investigate. This could be different types of farming environments, a veterinary surgery, a home with domestic or exotic pets, or a remote field station. Students then write a full risk assessment and a code of good working practice for the people working in or visiting that environment.

There are many things to consider when completing this exercise, such as:

- How could the potential movement of people and/or animals affect the spread?
- What necessary hygiene practices are needed?
- What diseases could be an issue at these locations?
- What are the symptoms to watch out for?
- Do you catch these diseases from direct or indirect transmission? And how does this affect the risk assessment and code of practice?

How easily are infections spread?

The spread of disease through a population can be quite an abstract idea for students to understand. This is a fun game that students can play, to identify who started the outbreak of a disease and how far the disease spread through their class 'population'.

Student activity

You will need:

- a test tube and dropper for each participant
- distilled water
- 0.1 M NaOH
- phenolphthalein solution, dissolved in alcohol and diluted in water (pH indicator)

CAUTION: Sodium hydroxide (NaOH) and phenolphthalein can irritate the eyes and skin.

How to play the game:

1. Each student gets a test tube. All but one are half filled with distilled water. One is filled with the NaOH solution. With a large group of students (more than 30), two of the test tubes should contain NaOH solution.
2. Students move around the room and, when instructed, they put a drop of their fluid into the test tube of the person nearest to them.
3. Repeat until at least three exchanges have occurred.
4. When completed, put a drop of phenolphthalein in each test tube. If the fluid turns pink, the tube is 'infected' with NaOH.

Questions to consider:

- How many people in the population (the classroom) have the infection?
- Who do you think gave you the infection?
- Look at the class data and determine who started the infection.
- How would this game change depending on the method of transmission?
- Why is determining how a disease spreads important?

The Barrier Game

The Barrier Game, available as part of the downloadable sexually transmitted infection resources on our website (**www.microbiologysociety.org/STIplay**) can also be adapted to compare the spread of infection if certain control measures are in place.

Theresa Hudson

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Further reading

Emerging Zoonotic Diseases Policy Briefing. **http://microb.io/1SCnPIx**. *Last accessed 11 September 2015.*

Zoonotic diseases (zoonoses): guidance, data and analysis. **http://microb.io/1VRIYQF**. *Last accessed 11 September 2015.*

Zoonoses and the Human-Animal-Ecosystems Interface. **http://microb.io/1K11v5B**. *Last accessed 11 September 2015.*

Policy

Disease outbreaks: preparedness through policy

The global health security threat posed by emerging infectious diseases has been pushed up the policy agenda in the wake of the unprecedented Ebola epidemic in West Africa. Initial failure to launch a rapid and coordinated international response to the epidemic starkly highlighted serious weaknesses in global surveillance and disease emergency response capabilities.

Scientists testing hens for the avian influenza virus in Hong Kong, China.
Dung Vo Trung/Look At Sciences/Science Photo Library

Learning lessons

In the immediate-term, at least, the Ebola epidemic has engaged policy-makers with the need to improve global preparedness for future disease outbreaks. Numerous inquiries have been set up to 'learn lessons' from the epidemic, including the World Health Organization's (WHO) Ebola Interim Assessment Panel and the United Nations High-Level Panel on the Global Response to Health Crises. Global health security has also been a priority under Germany's presidency of the G7, with proposals from an expert group taken to the G7 summit back in June. In the UK, two House of Commons Select Committee inquiries are scrutinising what the Government has learned from the UK Ebola response.

Strengthening surveillance

One area of consensus is the need to strengthen surveillance and healthcare systems, particularly better supporting low- and middle-income countries to do so. This is important to ensure collective international health security in a globalised world where diseases can easily cross borders, but also to provide health security to individuals regardless of where they live.

The WHO and 196 countries already have an agreement under the International Health Regulations (IHR) to report public health risks and build disease surveillance and response capacities. However, many countries have been unable to meet their IHR obligations, and the long delay in the WHO reporting Ebola as an international public health emergency showed weaknesses in its own systems. Many countries also went against guidance and imposed travel and trade restrictions, the economic and political implications of which might deter swift reporting of potential outbreaks. The WHO is now reviewing how to strengthen the effectiveness and implementation of the IHR.

Finding sufficient funding might be a challenge, but the expert panel that formed proposals presented to the G7 estimated that surveillance systems could be significantly boosted in many countries with as little as $12 million to $15 million annually. The G7 did announce that they would be supporting at least 60 countries over the next 5 years to prevent epidemics, in addition to supporting other global initiatives, although precise details were limited.

Rapid responses

The need to better structure and support the WHO to coordinate rapid disease emergencies responses has also been made clear. The WHO Ebola Interim Panel stated that the WHO lacked the culture, financing and capacity to currently do this. Consequently, at the World Health Assembly in May it was agreed to establish a specific WHO emergency programme and workforce, and to pilot a $100 million contingency fund for financing initial field operations during outbreaks. Securing necessary longer-term funding increases from Member States to support this work still poses a challenge however.

The expert panel that informed the G7 summit made strong proposals for an independent multilateral organisation to be setup within the WHO to be responsible for global outbreak response. They also called for $150

million to $200 million per year to maintain a reserve of thousands of scientists and public health workers ready for deployment in the event of a serious outbreak.

Research and development

The Ebola crisis also highlighted the need for more proactive development of vaccines, drugs and diagnostics, and the need for effective and transparent frameworks to roll out trials during epidemics, and to ensure the timely sharing of research data. This is something the WHO has been developing a framework to facilitate. The UK Government has said it will champion this including ensuring open access to UK publically funded research and data. Plans to invest £20 million towards a new UK Vaccines Research and Development Network to focus on the most serious disease threats have also been announced.

Microbiology Society policy activities

In June, the Society distributed its briefing *Emerging Zoonotic Diseases* to parliamentarians and policy-makers across the UK and Ireland. The briefing, and the importance of microbiology in tackling epidemics, was cited in a House of Lords debate on the Ebola outbreak. The briefing also prompted a Parliamentary Question to be asked about Middle East respiratory syndrome (MERS) to Ireland's Minister for Health.

The Society, in collaboration with the Society for Applied Microbiology, also submitted evidence to the House of Commons Science and Technology Select Committee's inquiry *Science in Emergencies: the UK Lessons from Ebola*.

Further reading

Heymann, D. L. *et al.* (2015). Global health security: the wider lessons from the West African Ebola virus disease epidemic. *Lancet* **385**, 1884–1901.

***Health Plan* (2015).** Editorial. *Nature* **522**, 5.

Microbiology Society (2015). Briefing: *Emerging Zoonotic Diseases*. **http://microb.io/1SCnPlx**

Ebola Interim Assessment Panel. World Health Organization. **http://microb.io/1JxxoBr**. *Last accessed 18 August 2015.*

Outreach

Small World Initiative: pop-up events

It has been a year since the Society launched the Small World Initiative in the UK and Ireland on European Antibiotic Awareness Day 2014 and a lot has happened since then!

Society champion Ben Johns taking another sample into the soil bank.

The Small World Initiative team at High Lodge Forest Centre.

The Small World Initiative will give the general public, students and educators in the UK and Ireland the opportunity to work with scientists as part of a global initiative to discover new antibiotics from soil bacteria.

We have run a successful pre-pilot school partnership with the University of East Anglia and the Sir Isaac Newton Sixth Form College in Norwich, with participating students coming to our Annual Conference and presenting their work at the poster session. We have run a summer school for six undergraduate programmes and seven school partnerships, which have started their programmes in this new academic year. We have also chosen the University of East Anglia to host our match-funded PhD student, Ethan Drury, who will be working on a citizen science project as part of the Small World Initiative.

Ahead of the start of the PhD project, the Society ran two pop-up science events in August to launch the citizen science project, in collaboration with the Forestry Commission and Forest Research. The events took place at High Lodge Forest Centre and Alice Holt Forest. People visiting the event could take a sampling kit around the forest with them, collect a soil sample and prepare a spread plate of their sample when they returned to see the bacteria present in the soil, before depositing the sample in our soil bank, which was then sent to Dr Matt Hutchings' laboratory at the University of East Anglia for analysis.

Visitors to the stand were welcomed by a team of expert volunteers, who talked with people about hunting for new antibiotics in the soil, how we

find these medicines and the threat of antibiotic resistance. They could look at agar plates down the microscope that contained pre-prepared soil samples and also plates containing *Streptomyces* strains, which are the source of numerous antibiotics in common use. Interested visitors could also look at micrograph pictures of various microbes and guess whether antibiotics were effective against them or not. There was also a physical challenge, where children could throw giant microbes at an antibiotic pill, which got more ineffective the more microbes were thrown at it. But by far the most popular part of the stand was the science itself.

The project gained interest in the local media, with appearances on BBC Radio Norfolk and BBC Radio Surrey, and television slots on BBC Look East and BBC South Today. Lots of visitors to the stand had seen the project on local television and were keen to find out more and be a part of the project.

People of all ages enjoyed collecting their sample, and prepping it ready for further analysis. We were really pleased to see the enthusiasm from people who are following the analysis online. Results of the experiments have been shared on the Society's website at **www.microbiologysociety.org/smallworld** and **www.facebook.com/smallworldscience**. Even if you didn't manage to join us in the forests, please check out the website and Facebook page and see if you can spot any potential antibiotics on our samples!

The Society would like to thank Forest Research and the Forestry Commission for hosting the events. We would also like to thank Dr Matt Hutchings and his team at the University of East Anglia for providing agar plates for the stand and for providing laboratory support for the sample analysis. We also thank Dr Paul Hoskisson for providing plates of *Streptomyces* cultures for the stand, Dr Laura Bowater for joining us on the stand at the High Lodge event, our President Professor Nigel Brown and Forest Research staff Sue Benham and Peter Crow for volunteering with us at Alice Holt and all the other volunteers and Society staff who joined us for the two events. Their hard work and enthusiasm made it a success.

Theresa Hudson
Education and Outreach Manager
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Top left. **Theresa Hudson on BBC Look East news.**

Bottom left. **The soil bank.**

Right. **Demonstrating antibiotic resistance through attacking Superman with giant microbes!**

Membership

Photos Ian Atherton

Are you a postgraduate student (or know one)?

Here's one very good reason to become a member of the Society...

Postgraduate student attending Annual Conference for three days (two nights)

	Non-member	Member with grant
Conference registration – 3 days @ £160/£70 day	£480	£210
Accommodation (est.) – 2 nights @ £100/night	£200	£200
Travel and subsistence (est.)	£70	£70
Society grant		–£290[1]
Total	**£750**	**£190**

[1]This was the average Conference Grant amount awarded in 2015 – exact amounts will vary according to individual applicants' circumstances.

The Society's largest event of the year is just around the corner. Our Annual Conference brings together over 1,200 Society members and colleagues from across the world to share research and build career-enhancing networks. This event is a must for anyone with an interest in the subject, but particularly for postgraduate students, who can present their work, build their understanding and make great connections in a supportive and friendly environment.

The 2016 Conference will be held in Liverpool from 21 to 24 March. If you are interested in attending, or know someone who might be, registration and abstract submission are open now.

The Society offers generous grant support to postgraduate members, to make attendance an even more attractive proposition. Around 300 Annual Conference grants are awarded every year to eligible applicants. The discounted attendance rates and the grant, together, make a very strong financial case for membership.

This example compares a postgraduate student attending the Conference as either a non-member or a member. The figures are based on our 2015 Conference in Birmingham.

In this instance, a £33 investment in membership could have saved our non-member £560, or 16 times its value! If you have postgraduate students, colleagues or friends who would like to go to the Conference but currently don't – or do as non-members – then please let them know about what they are missing out on.

For the full list of Society grants available, plus details of our 2016 Annual Conference and how to join the Society, please visit our website **www.microbiologysociety.org/join**

Paul Easton
Head of Membership Services
p.easton@microbiologysociety.org

> **The conference content, networking and learning opportunities have all been excellent.**
>
> Society member Lidia Lasecka, 2015 Birmingham event

Obituary

Professor Richard Elliott

It is with great sadness that we note the passing of Richard M. Elliott of the Medical Research Council-University of Glasgow Centre for Virus Research.

Richard Elliott.
MRC-University of Glasgow Centre for Virus Research

Richard had long been a member and supporter of the Society. He was one of the country's leading virologists and always found the time to interact with wider communities. He served on the Society's Council twice, and was the Editor-in-Chief of the *Journal of General Virology* from 2008 to 2012. In the latter role, he saw the journal cement its position as one of the leaders in the field, at a time when competition from new online and open access journals began to increase.

In his research, Richard Elliott brought bunyaviruses, which today are recognised as a clear threat to public health and agriculture, to the attention of a broader scientific audience. Richard's contributions to the understanding of bunyavirus molecular biology were world leading. He was a pioneer during a time when an exponential increase in travel and commercial exchanges, as well as ecological and climate change, have led to a geographical expansion of many of the viruses that are transmitted by arthropods, including bunyaviruses.

His group established reverse genetics systems for newly emerging viruses such as Schmallenberg virus, which infects cattle, sheep and goats, and which causes a variety of problems from reduced milk yields to severe deformity. For this work, Richard returned to the bench and took the lead in carrying out experiments in the laboratory. He contributed to the Society's briefing note on Schmallenberg virus in 2012, which received attention in the UK and Irish Parliaments, the Northern Ireland Assembly and within government. He spoke about the Schmallenberg work at the Annual Conference in 2014. His contributions in this area helped to ensure that it was a subject to which the Society would return, producing a further Parliamentary briefing paper on emerging endemic diseases of livestock in 2014.

Richard was a member of the International Committee on the Taxonomy of Viruses bunyavirus study group, an important service to the community. He was also strongly involved in the organisation of the European Meetings on Viral Zoonoses since 2001, which have played a major role in bringing researchers in this field together. This enthusiasm for bringing virologists together was a theme throughout Richard's career. His passion and enthusiasm for virology influenced more than just those individuals that were fortunate enough to work with him; Richard has had an important influence in the careers of many virologists both in the UK and further afield.

Richard Elliott's contributions to the Society were many and varied. He contributed to many of the Society's conferences, from the Virus Group meeting on viruses and their hosts at Southampton in 1997 to this year's Annual Conference in Birmingham, where he was an author on no fewer than five presentations and posters.

It was characteristic of Richard's passion and determination that he worked on manuscripts and was in touch with his laboratory and colleagues until the very last days. His final contribution to the Society was posthumous. He had been a member of the organising group for one of the Focused Meetings held this September - the International Meeting on Arboviruses and their Vectors. There could be no greater testament to his commitment to virology than to witness speakers and poster presenters at all stages of their careers, drawn from 18 different countries on five different continents, coming together to share their knowledge and enthusiasm for the study of arthropod-borne viruses. The first day of the meeting closed with a commemoration of Richard Elliott's life and his many outstanding contributions to microbiology.

Membership Q&A

In this issue, we're pleased to introduce Angharad Ellen Green.

A. Green

Where are you currently based?

I am a second year PhD student in the Mahenthiralingam laboratory at the School of Biosciences, Cardiff University. My BBSRC CASE project is industrially funded by Unilever's 'Safety and Environmental Assurance Centre' (SEAC).

What is your area of specialism?

Industrial antimicrobial resistance.

And more specifically?

My PhD focuses on harnessing the power of bacterial bioluminescence to understand the genetic basis of industrial preservative resistance. I have constructed bioluminescence reporters using an industrially isolated strain of *Pseudomonas*. Light emission alterations are used to identify differentially regulated genes in response to preservatives. To characterise the genetic basis for biosensors of interest, the DNA flanking the transposon insertion is being sequenced using PCR-based techniques. A panel of useful *Pseudomonas* biosensors will be developed to optimise preservative formulations. I will also use transcriptomic approaches, e.g. RNA sequencing, to validate the results and identify further genes of interest.

Tell us about your education to date

I graduated from Oxford Brookes University in 2010 with a BSc (Hons) in Equine Science, which was followed by an MSc in Medical Microbiology at Cardiff Metropolitan University. My MSc allowed me to publish my first paper on the effect of the flavonol morin on adhesion and aggregation of *Streptococcus pyogenes*, which developed my passion for research and led on to a PhD at Cardiff University.

Where did your interest in microbiology come from?

At my comprehensive school, by my very enthusiastic and inspiring biology teacher, Mrs Phillips. I loved both horses and biology and discovered the Equine Science degree at Oxford Brookes. My dissertation focused on equine distal limb wound healing and I became fascinated with the wound microbiome. This led on to an MSc in delayed wound healing, biofilms and novel antimicrobial treatments. The experience of publishing my MSc research, in *FEMS Microbiology Letters*, confirmed my choice of a microbiology PhD.

What are the professional challenges that present themselves and how do you try to overcome them?

When I first started the PhD I found it challenging to successfully manage lab work, while writing up reports and demonstrating in practicals. I overcame this by taking advice from experienced lab members and participating in courses run by Cardiff University's Graduate Centre. I now feel I can coordinate my time in work effectively, while also having an active lifestyle outside of the laboratory, achieving a good work-life balance.

What is the best part about 'doing science'?

When an experiment is proving difficult, but you persevere and it works with interesting outcomes. I also find communicating science at conferences and public engagement events rewarding.

Who is your role model?

My PhD supervisor, Professor Eshwar Mahenthiralingam, and everyone within my lab group. There is a collaborative and fun approach to science; I am appreciative to have such great support while starting out in my research career. Everyone is encouraged to discuss their experiments in meetings and there is always someone willing to give advice.

My MSc project supervisor, Dr Sarah Maddocks, is also a role model as she helped develop my research skills during the early stages of my career and encouraged me to publish my findings.

I also want to mention my mum who successfully completed a BSc (Hons) in Mental Health Nursing, while working full-time when my brother and I were young; she's my inspiration to not give up when faced with difficult challenges.

What do you do to relax?

I like to keep active and enjoy a challenge so I am training for this year's Cardiff half-marathon. I also own three Welsh Cobs with my mum and so the weekends usually consist of a gallop along the Welsh coast or competing at an event.

What one record and luxury item would you take to a desert island?

My record would be 'Tragic Kingdom' by No Doubt, and a Kindle loaded with good books.

Tell us one thing that your work colleagues won't know about you!

I rode a police horse while I was on work experience at the South Wales Police headquarters, during Year 10 at school.

If you weren't a scientist, what would you be?

An Egyptologist. I found the 'Mummy' film franchise inspiring as a teenager.

If you would like to be featured in this section or know someone who may, contact Paul Easton, Head of Membership Services, at **p.easton@microbiologysociety.org**

Baby Asian elephants in an elephant conservation centre in Laos.
Fabien Bastide

Best of the blog

It's time for our last roundup of 2015, and we've definitely saved some of the year's best content 'til last. In one of my favourite posts of 2015, Anand Jagatia went to the University of Bath to talk to some researchers there about their development of 'self-healing' concrete, which uses bacteria that secrete calcium carbonate to fill any gaps in the material (**http://microb.io/1d3SNsU**).

Anand also spoke to an international team who have discovered a rather interesting biofilm phenomenon: their growth oscillates between fast and slow. This new finding – a classic nutrient feedback loop – may help us treat persistent infections, by feeding the outside of a biofilm in order to starve the centre... (**http://microb.io/1Mfwb79**).

Earlier in the year, the Wellcome Trust's online magazine, *Mosaic*, published a fascinating article on the microbiology associated with death and decomposition. In part one of our forensic-focused podcast, we spoke to Mo Costandi, the author of the piece, about his travels to 'body farms' in the US, and learnt how microbiology is helping to identify where and when somebody died (**http://microb.io/1CPFhWA**).

When treating an infection, two drugs are better than one, right? Not necessarily. We spoke with Dr Pleuni Pennings, an evolutionary biologist from San Francisco State University, who has modelled how therapeutics that target different parts of the body may leave drug-free 'sanctuaries', from which resistance can emerge (**http://microb.io/1MtoOHk**).

Back in June, we spoke to Society Member Laura Bennett to learn about her research into elephant endotheliotropic herpesvirus, or EEHV, for short. As its name suggests, EEHV is a disease of elephants, disproportionally affecting juvenile Asian elephants under 5 years old, with mortality rates estimated to be as high as 85 per cent. (**http://microb.io/1f4Ssl4**).

Each year, the Society attends Parliamentary Links Day, an event organised by the Royal Society of Biology that brings together researchers, learned societies and parliamentarians. One of our members in attendance was Society Champion Arikana Massiah, who wrote about her experience of the event, which saw senior politicians and researchers lay out their thoughts for the future of UK science (**http://microb.io/1MXWukg**).

So that's it for another year. Thanks to you all for taking the time to read, watch or listen to our posts – we'll be back again in 2016 with more great stories, see you all then.

Benjamin Thompson
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Reviews

One Health: People, Animals, and the Environment

Edited by R. M. Atlas & S. Maloy

ASM Press (2014)

US$90.00 ISBN 978-1555818425

The One Health concept is defined as a global, interdisciplinary and cross-sectoral collaborative effort to reach optimal health for people, animals and the environment at a local, national and international level. As the world is getting riskier and pathogens keep emerging and re-emerging, this book stresses the importance of the One Health concept as a means of predicting and preventing infectious disease epidemics before they happen – an alternative to current emergency outbreak response practices.

The book is divided into 20 individual but interlinked chapters, written by leading experts that cover the following topics:

- Importance and key concepts of the One Health approach.
- Origins of infectious diseases and risk factors associated with their emergence.
- Case histories of recent zoonotic viral, bacterial and fungal infections.
- Antibiotic resistance.
- Disease surveillance systems and global information networks.
- Obstacles to implementing One Health, early successes and future directions.

I found this book well written, easy to follow and interesting. The chapters are referenced throughout and offer links to websites and online tools for further studies. A number of chapters include illustrations, tables or flow charts. Some information is slightly redundant given the independence of the chapters, but overall each chapter looks at the One Health approach from a different angle.

One Health certainly is a very relevant topic in the light of current and future infectious disease outbreaks. Thus, this book would be a useful resource for postgraduate students, veterinarians, physicians, microbiologists, ecologists, epidemiologists or politicians with prior knowledge of infectious diseases and an interest in understanding the links between human and animal health and environmental factors.

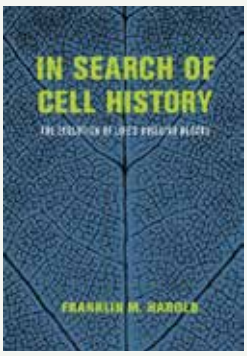
Isabelle Dietrich
MRC-University of Glasgow Centre for Virus Research

In Search of Cell History: The Evolution of Life's Building Blocks

Written by F. M. Harold

The University of Chicago Press (2014)

£28.00 ISBN 978-0226174280



In Search of Cell History: The Evolution of Life's Building Blocks is an important new book by the American microbial physiologist Franklin M. Harold, which really aims to remind the gene/genome-centric generation of (micro) biologists about the centrality of the cell itself in our thinking about the evolution of life. This is not a return to any vitalistic theory of life, but a clear and modern restatement of the importance of the membrane-bound bag of biochemicals that constitutes a cell as being equally important in the emergence and transmission of life as the genetic material it houses. No biologist would doubt Verchow's statement of 1858 of "Omnis cellula e cellula", or "every cell from a previous cell", and although Craig Venter can synthesise a complete bacterial genome, it is lifeless until it is transplanted into an existing living cell. The book aims to describe how cells came about and Harold takes us on a journey back through time, bringing together the latest microbiological and evolutionary research to try and address important and still mainly unanswered questions about the origins of cellular systems. I found it a fascinating book that one can dip in and out of, as Harold brings together many different lines of evidence from, for example, geology, symbiosis and bioenergetics, to his story. It is certainly aimed at working (micro) biologists and is not a regular popular science book with some quite technical sections, but it is well suited to readers of *Microbiology Today* and I would highly recommend it.

Gavin H. Thomas
University of York

Comment

Zoonoses in Africa

Eric Fèvre

Zoonotic diseases are those infections transmissible between animals and humans. Most diseases that infect humans are, in fact, zoonotic, highlighting that multi-host pathogens have a competitive edge by increasing the range of host environments in which they can survive. A recent briefing note by the Microbiology Society highlights that many zoonotic diseases are also emerging diseases – that is, either an altogether new disease, or a known disease that is increasing in incidence or increasing in geographical range.

Disease emergence happens because the world we (and our microbes) live in is changing (often because of the actions of humans, but also due to natural processes), giving pathogens new opportunities to infect new hosts, and creating new opportunities for hosts to interact with each other and share their infections. The intensification of farming, for example, leads to closer relationships between individual animals, generating opportunities for more rapid mutations as organisms move from host to host, while also providing a structured way for those pathogens to enter highly ordered food chains that branch out and reach very large numbers of people.

Zoonotic diseases are a global problem, with rich and poor countries both at risk, and practices in both highly developed and less developed economies predisposing to transmission. A significant difference, however, between a continent like Africa and one like Europe, is the ability of national and regional systems to detect and respond to zoonotic disease threats. Indeed, in work investigating where hotspots of disease emergence are located, developed countries stand out as being at higher risk, mainly only because more such events are detected in such places as a result of robust systems for monitoring and detection. Over the past couple of years, the struggle – now largely successful (but still not completed) – to contain the West African Ebola outbreak highlights this problem.

Other than the headline-hitting, large global outbreaks of zoonotic infections, human populations face a multitude of challenges from less dramatic, less fatal, diseases that cause long-term morbidity in large groups of people (morbidity refers to illness, as opposed to death, termed mortality). In Africa, several such zoonotic neglected diseases of neglected populations conspire to hinder the health of people and the animals they depend on for their livelihoods. This includes bacterial, protozoan and viral disease agents, transmitted in many ways.

The World Health Organization (together with its partners) has been keeping a focus on these diseases, with a series of reports and policy recommendations published since 2006 (with the latest having been released earlier this year (**http://microb.io/10iiBkB**). We'll look briefly at three of them.

Cysticercosis is a disease caused by the tapeworm *Taenia solium*. It has a

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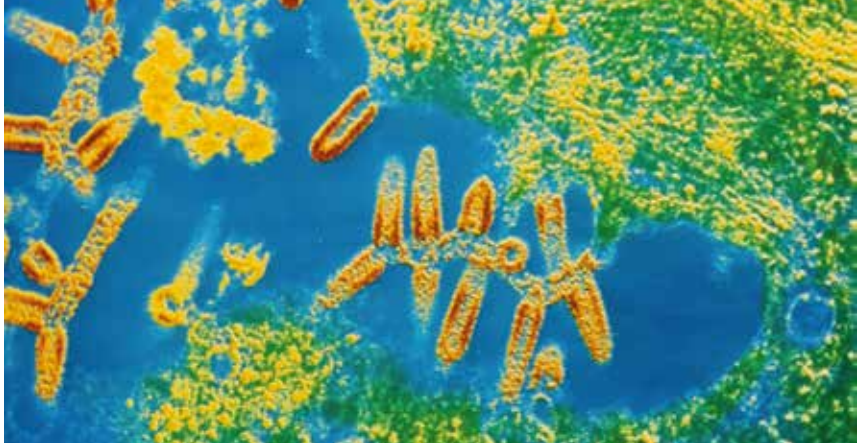

False-colour transmission electron micrograph of rabies virions (red, elongated virus particles) budding away from host cell cytoplasm. CNRI/Science Photo Library

relatively complex lifecycle, involving pigs eating *Taenia* egg-carrying human faeces that contaminates the environment, humans eating undercooked pork meat, and environmental contamination with eggs that can encyst in humans. The greatest problem with *T. solium* is that it may cause a neurological disease called neurocysticercosis in people who are infected with tapeworm eggs; in many developing countries, it is the single largest cause of acquired epilepsy in humans.

Brucellosis is a bacterial disease caused by one of several organisms in the genus *Brucella*. Cattle, sheep and goats harbour this bacterium, which they transmit to each other and humans through milk or through contaminated abortion materials (the bacterium is abortogenic). It causes a chronic debilitating disease in humans with joint pain, fatigue and recurrent fever; brucellosis is often misdiagnosed as malaria and wrongly treated.

Rabies is a well-known but none-the-less neglected zoonotic infection, caused by the rabies virus. It is transmitted and maintained mostly in domestic dog populations (though in areas with a wildlife interface, the epidemiology may get more complex), which transmit the infection to humans through bites. It is best controlled by vaccinating the dog reservoir to prevent disease from developing if infection occurs, but a human vaccine is available both as a pre- and post-exposure course, and effective and timely delivery of the vaccine will minimise mortality.

For these diseases, and others like them, many aspects of their basic biology are well understood, and the transmission of the pathogens has been controlled in many countries. The outstanding issues, which require more research for the effective deployment of intervention efforts, are i) knowing how to integrate surveillance for the diseases into national systems (because having data on occurrence, distribution and disease burden is essential to prioritisation), ii) deploying, and in some cases developing, new and better tools to diagnose the infections in humans and animals (because accurate and efficient detection is key to both delivering cure and also to gathering good surveillance data), and iii) scaling up intervention strategies. The World Health Organization has stated that "we have existing knowledge and evidence that can be transformed into strategies and applied on a large-scale; we need to be able to capitalise on experience and the growing political commitment to involve other sectors, with community participation."

Effectively tackling zoonoses requires a focus on transmission control, prevention and burden reduction in humans, but also control and transmission prevention in animals. This, in turn, requires a One Health approach, involving joint surveillance, joint control and joint policy management by veterinary, medical and other sectors. With respect to African countries, the weaker institutions that exist relative to many developing countries actually present a real opportunity in this regard, with greater possibility to strengthen those institutions with, in mind from the outset, a unified approach to disease management across sectors. Many efforts are under way to prime this process, and it is well under way in a number of places. A prime example of a One Health approach in action is the Zoonotic Disease Unit (ZDU) of the Government of Kenya, a joint initiative between the ministries responsible for human and veterinary health. It has, over the past few years, developed a highly regarded National Rabies Control Strategy, implemented large-scale studies to study brucellosis epidemiology, responded to many zoonotic disease outbreaks, and developed preparedness plans for epidemic zoonoses such as Rift Valley fever (for which 2015 is a high risk year due to unusual rainfall patterns). The ZDU is also very open to collaboration with research teams working in Kenya, including in the implementation of zoonotic disease surveillance activities.

Tackling zoonoses is a difficult, fascinating task; it requires a good understanding of biology, of the way environment influences disease, of human–animal interactions, of policy and of politics.

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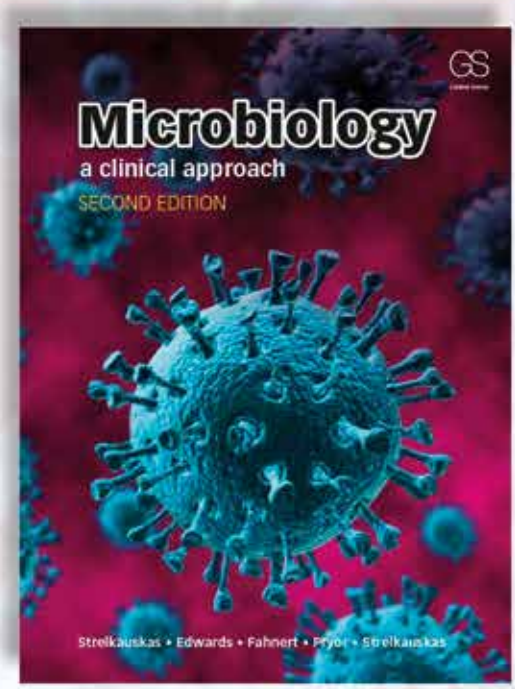

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