

MICROBIOLOGY TODAY

QUARTERLY MAGAZINE OF THE SOCIETY FOR GENERAL MICROBIOLOGY

39:1 FEB 2012

THE NEONATE AND EARLY CHILDHOOD

RETROVIRAL PLACENTAL SYNCYTINS

THE GUTS OF NEONATE MICROBIOLOGY

HELICOBACTER IN CHILDHOOD

MYTHS AND LEGENDS OF *CRONOBACTER*

ERADICATING MEASLES

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Nitrofurantoin Modified Release

MacroDantin[®]
Nitrofurantoin Capsules

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Prescribing Information MacroBID[®] (Nitrofurantoin MR Capsules) MacroDANTIN[®] (Nitrofurantoin Capsules)

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pulmonary, hepatotoxic, haematological or neurological syndromes occur. Contains lactose. **Pregnancy and Lactation:** Contraindicated in pregnancy at term (including labour and delivery). Nitrofurantoin is detected in trace amounts in breast milk. Should be avoided if breast feeding infants suspected to have G6PD deficiency. **Interactions:** Concurrent use with quinolones, magnesium trisilicate, uricosuric drugs such as probenecid and sulphapyridine, carbonic anhydrase inhibitors, urine alkalising agents, oestrogen, oestrogen containing contraceptives and oral typhoid vaccine is not recommended. Increased absorption with food or agents delaying gastric emptying. **Adverse Effects:** Nausea, anorexia, emesis, abdominal pain and diarrhoea have been reported. Less common and rare are those events that affect the respiratory system. Acute pulmonary reactions occur in first week of treatment and are reversible with cessation of therapy. Sub-acute and chronic reactions (collapse, cyanosis, fever, chills, cough and dyspnoea) can occur with continuous treatment for six months or more. Hepatic (reactions including cholestatic jaundice, and chronic active hepatitis which may lead to hepatic necrosis occur rarely). Fatalities have been reported. Neurological (peripheral neuropathy and optic neuritis infrequently), Haematological (anaemia's, G6PD deficiency and other rarely reported events such as leucopenia, agranulocytosis, granulocytopenia and thrombocytopenia resolve with cessation of therapy). Allergic reactions including rashes eczematous eruptions and pruritus. Angioneurotic oedema, anaphylaxis, Lupus-like syndrome, sialadenitis, pancreatitis. Transient alopecia and benign intracranial hypertension have been reported. Superinfections by fungi or pseudomonas may occur. Please refer to Summary of Product Characteristics for detailed information. **Legal Category:** POM. **Basic NHS Price:** Macrobid: £4.89 per pack of 14 capsules, MacroDantin: £2.49 per 30-capsules pack of 50mg, £4.81 per 30-capsules pack of 100mg **Marketing Authorisation Number:** Macrobid 100mg PL 12762/0052, MacroDantin 50mg PL 12762/0048, MacroDantin 100mg PL 12762/0049 **Marketing Authorisation Holder:** Goldshield Group, NLA Tower, 12-16 Addiscombe Road, Croydon, Surrey, CR0 0XT, UK. **Date of Revision** September 2011.

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Interested in being the next Editor of *Microbiology Today*?

It's hard to believe that the current Editor of *Microbiology Today* (MT), Paul Hoskisson, is coming to the end of his very successful 3-year term of office in autumn 2012. Of course, this means it is time to appoint a new person to take over for the next 3 years, starting in October 2012.

In a break from the traditional route of in-house appointment, we are opening up the process to any Ordinary Member of the Society who is interested in being the next Editor of our prestigious and highly regarded house magazine.

As the Editor you are responsible for the scientific content and integrity of the magazine. You will be required to attend and chair four Editorial board meetings per year, read, assess and edit all submitted feature and Comment articles, liaise with the Managing Editor, Head of Communications and CEO at SGM HQ with regard to any matters arising that involve the content or development of MT, and represent and promote the magazine outside SGM.

In addition, you will sit on SGM Council as a co-opted member (attending four meetings per year), ensuring that MT reflects the views of Council and the Society.

Desirable qualities for the position include:

- an interest in a wide range of subjects across the whole of microbiology
- an ability to assess contributions for their suitability for publication
- excellent communication skills, including the ability to deal with sensitive issues
- enthusiasm about the communication of microbiology to key stakeholders, including other scientists, parliamentarians, policy-makers, educators and the general public

You do not need to have been involved with the Editorial Board or SGM Council previously to apply, but you do need to be a member of the Society. If you are interested, please send your CV with the names of two referees to Yvonne Taylor at y.taylor@sgm.ac.uk by **30 March 2012**. The appointment will be subject to approval by SGM Council.

We look forward to hearing from you.

IAN ATHERTON, DESIGN MANAGER AND MANAGING EDITOR
MICROBIOLOGY TODAY

BIG BUZZ AROUND HONEYBEE ISSUE

The headline may be corny, but there is nothing remotely hackneyed about the response we have had to the November 2011 issue of *Microbiology Today* on the microbiology of the honeybee.

Following a plug at my local beekeeping association in Wokingham, Berkshire, our regional bee inspector, Nigel Semmence, a former microbiologist who was giving a talk that night, took away a copy and later contacted me to ask for a further 70 copies to be distributed to all national bee inspectors and managers at the National Bee Unit (NBU).

Another 160 or so copies were distributed to the executive officers of the British (BBKA) and Scottish (SBA) Beekeepers Associations, the International Bee Research Association (IBRA), the Central Association of Beekeepers (CABK), the Federation of Irish Beekeepers' Associations (FIBA) and Apimondia. Copies have also been sent to contacts in all the British and Irish regional associations.

In addition to the 80 or so parliamentarians who take *Microbiology Today* regularly, the November issue of the magazine is always sent to all MPs, MSPs, AMs, MLAs, Members of the House of Lords, TDs and Senators, and this issue was no exception.

Judging by the very positive feedback received and the numerous requests for additional copies, the issue has aroused a great deal of interest and discussion well beyond the membership of SGM and the usual readership.

IAN ATHERTON

National Science & Engineering Week 2012

If you are a member and you are organizing or getting involved with an event for National Science & Engineering Week, we would love to hear from you. Let us know by contacting Yvonne Taylor at y.taylor@sgm.ac.uk

Help shape the future of SGM

If you are an Ordinary Member of SGM, please consider the opportunity to join Council and have a bigger say in how your society develops in the next few years.

MARK HARRIS and **GARY ROWLEY** will retire from Council in September this year. This opens up two vacancies for elected members to serve on Council from September 2012 until September 2016. SGM, under Council's guidance, is undergoing a period of review and change and we would ideally like to recruit new Council members with expertise in one or more of the following areas: (i) senior management in a similar organization to SGM, (ii) electronic records or document management systems, (iii) marketing and brand management, (iv) development of membership support and services, and (v) STEM careers awareness/promotion to school students.

Council meets four times each year and, in addition to the formal meetings, elected members also have an important role to play in several of the committees that influence different aspects of SGM business, such as journal production, finance, policy, conferences, education and outreach activities. Council members are also encouraged to participate in SGM conferences where the cost of their attendance is supported by the Society.

If you would like to contribute, and have expertise you would like to share with us, we would very much like to hear from you. Nominations should come from individuals who have been an Ordinary Member of SGM for at least 2 years and must be supported by two other Ordinary Members. Please use the nomination form at www.sgm.ac.uk/about/objectives.cfm and include information about the skills you can offer and how you would like to contribute to Council. Please send your nominations to Jane Westwell, SGM, Marlborough House, Basingstoke Road, Spencers Wood, Reading, RG7 1AG (j.westwell@sgm.ac.uk). The closing date is **20 April 2012**. Informal enquiries about the role of elected Council members can be made to Simon Festing (s.festing@sgm.ac.uk).

If the number of nominations exceeds the number of vacancies an election will be held in early summer.



REPORT ON COUNCIL ACTIVITIES FROM THE CHIEF EXECUTIVE

It is a little over 6 months since our previous Chief Executive, Ron Fraser, retired and I took over the reins at SGM. Since then there have been a few changes at the Society, including the publication of approved Council minutes on our website (www.sgm.ac.uk/whos_who/). This is intended as an exercise in openness and to encourage interest in the affairs of the Society. As a result, the report back from Council in *Microbiology Today* can be less detailed than it was previously.

Along with the publication of the minutes, Council is reviewing the governance arrangements of the Society to make sure our rules are transparent and everyone knows how we operate.

Since I started, I have been working closely with Council to determine the future direction of the Society. Perhaps the most significant initiative we have undertaken is to review our strategy. It is, of course, quite customary for a learned society to refresh its strategy on a regular basis and it was timely that this was done for SGM. Council is currently reassessing and revising our strategy with a view to publishing it on our website this spring.

In the meantime, here are some of the areas of SGM activity that are currently under review.

Improving membership services is always important for a membership organization. We are pressing ahead as fast as we can to update our database structure and our website in order to deliver better online services (such as subscription renewals and electronic voting), and these should be in place by the end of the year.

It is vital that we keep up the good work that the Society has built up over many years in areas such as education and public engagement. We are one of the most active societies when it comes to engaging with school children, media and the public. We also wish to raise the profile of microbiology in policy debates more widely, for example through the publication of our recent position statement on *Food Safety and Security* (www.sgm.ac.uk/NEWS/PositionStatements.cfm).

Our Council and committees are also working to review and improve two major areas of activity. First, we

are examining our journals publication business to ensure that we can best adapt to the changing external environment. This includes financial austerity, increasing competition and a continued drive towards open-access in some quarters.

Second, we will continue to consider the future shape of our scientific conferences, seeking to get the right balance between the content of the meetings, good attendance and a reasonable cost. These can be difficult areas in which to get the balance right.

Finally, we are most grateful to those members of the Society who have responded to our electronic surveys. It is important that members get the opportunity to feed in their views to the development of the Society's activities. I likewise give my thanks to the great many volunteers who give their time to serve on our Council, committees and journals. We depend enormously on your efforts and goodwill to keep the Society going and to help keep microbiology thriving into the future.

DR SIMON FESTING, Chief Executive

SOCIETYBUSINESS

Get **LinkedIn** with **SGM**

Join the SGM members group on LinkedIn and stay up to date with opportunities for members.



Be part of SGM's online community.



News of Members

The Society offers its congratulations to **PROF. JOHN SISSONS**, University of Cambridge on his award of Knight Bachelor for services to Research and Education in Clinical Medicine (in the Queen's New Year Honours); **PROF. ANNE GLOVER** on her appointment as Chief Scientific Adviser to the European Union President (see p. 50); **PROF. DUNCAN MASKELL** who has been reappointed as an expert member of the General Advisory Committee on Science; **PROF. POLLY ROY** who has been awarded the General President's Gold Medal by India's Prime Minister in recognition of her research; and former Fleming Lecturer **DR NICOLA STANLEY-WALL** who has been announced as the 2012 winner of an RSE Beltane Innovator's Award for Public Engagement.

The Society notes with regret the death of **PROF. HARRY SMITH** (member since 1955; see obituary on p. 65). The Society also sadly notes the passing of **DR GAIL FERGUSON** (1969–2011, and member since 1991). Gail was the SGM departmental rep at the University of Aberdeen and a contributor to careers workshops that we organized for graduates and postgraduates. Her colleague Ian Booth writes: 'Gail was an immensely talented molecular microbiologist who focussed on bacterial stress response mechanisms. Gail's legacy is founded on the training she provided and her ability to draw together diverse insights. She was intellectually and experimentally rigorous and possessed a rare gift for collaboration. Her work encompassed the effects of electrophile toxicity, cell-wall development, high-pressure physiology, metagenomics and models of skin infections. She was awarded the 2009 Wain medal. In all her



Gail Ferguson

work Gail never took the easy path, but her imagination provided many novel solutions. One quote sums up the sentiments of many of us: "She was a person that I enjoyed seeing walk into a room, and someone that I hated to see leave. I am glad that I was able to get to know her, and to call her a friend and colleague." Her presence will be greatly missed.'

STAFF

We warmly welcome our new members of staff.



William Burns, J. Atherton

WILLIAM BURNS joins us as our Science Policy Officer. William was educated at Imperial College and NIMR, Mill Hill, and has worked as a science writer, business-to-business journalist and policy analyst in Europe and Asia-Pacific. He is fully conversant with the latest economic approaches to science policy and has particular expertise in food production, the chemical industry and the globalization of R&D. William is keen to talk to members about their policy interests and what SGM can do to articulate them – so please do contact him.

RICHARD SCRASE is the online science producer for Understanding Animal Research where he produces videos and writes science news. He is on secondment to SGM for 6 months to arrange, film and edit educational films. In his previous life Richard was a science teacher and science writer. His formal education includes an MSc in Science Media Production from Imperial College and an MPhil in Mycology from Bath University.



Richard Scrase, J. Atherton

SHARON JOHNSTONE has been appointed as HR Manager for 1 year. Sharon has a solid background in general HR, and has recently advised a number of small charities on their HR policies on a consultancy basis.



Sharon Johnstone, J. Atherton

In December 2011, we said a sad goodbye to **MARIANNE ASBURY**, JGV Editorial Assistant, after 20 years of service to the journals and we wish her a happy retirement. **SARAH FERRIS** has moved over from IJSEM to replace Marianne on JGV. But as we say farewell to one member of journal staff, we say hello to two new Editorial Assistants, **CHRISTINE WHEELER** (JMM) and **LYDIA CHARLES** (IJSEM). Christine is returning to work after bringing up her children, having previously worked in telecommunications. Lydia has spent 3 years working as an audio typist at the Cellular Pathology Department of the Royal Berkshire Hospital before joining SGM. Before that she spent a year working for the Army Legal Department in Herford, Germany.



Lydia Charles, J. Atherton



Christine Wheeler, J. Atherton

PEOPLE

Sir Howard Dalton Young Microbiologist of 2011

The final of the 2011 Sir Howard Dalton Young Microbiologist of the Year Competition, held at the SGM Autumn 2011 meeting, University of York, was unusual in having an all-female line-up. As usual, presentations were at a very high standard and the judges had some tough decisions to make. The contestants joined **SUE ASSINDER** and **SARA BURTON** from the Education Division at the conference dinner where the winners were announced by SGM President **HILARY LAPPIN-SCOTT**.

The first prize was awarded to **MARIAN KILLIP**, a postdoctoral researcher at



Marian Killip, Joanna Verran

FIRST AWARD FELLOW RECEIVES TRAVEL GRANT

In 2011, SGM launched a new grants scheme in collaboration with the African Women in Agricultural Research and Development (AWARD) Project. AWARD is a professional development programme that strengthens the research and leadership skills of African women in agricultural science, empowering them to contribute more effectively to poverty alleviation and food security in sub-Saharan Africa. The SGM scheme offers Scientific Conference Travel Grants for AWARD Fellows.

The first grant from this scheme was awarded to **DR RACHEAL AYE** of the International Livestock Research Institute in Nairobi, Kenya. Rachael presented her work aimed at identifying vaccine candidates in *Mycoplasma mycoides*, the cause of contagious bovine pleuropneumonia, at the Conference of Research Workers in Animal Diseases, held 4–6 December 2011 in Chicago, USA. She found the conference a great experience that generated a lot of new research ideas, and she further used the opportunity to visit Pfizer Research and Development Laboratories. She hopes the trip will lead to new collaborative opportunities to further her research in the future. Rachael has become an International Associate Member of the Society.



Rachael Aye

FIS Annual Scientific Conference

SGM hosted the Federation of Infection Societies (FIS) Annual Scientific Conference held in Manchester, 16–18 November 2011. SGM has been closely involved with the FIS annual conference for several years and this was the second time we have hosted it. The majority of delegates at FIS conferences are medical professionals with interests in microbiology. The conferences offer a mixture of sessions relating to research and clinical practice with opportunities for delegates to present case studies and research papers either orally or as posters.

The 2011 conference attracted over 600 delegates who participated in sessions on a variety of topics including antibiotic resistance, infection control, cystic fibrosis and microbial pathogenesis. In addition, **PROF. DAVID BLACKBOURN** (SGM General Secretary) ran a career development workshop for trainees on critical appraisal of the scientific literature.

Our grateful thanks are due to **PROF. DIETRICH MACK**, Swansea University, who has represented SGM on the FIS scientific committee for a few years and who chaired the committee during 2011. We very much appreciate his hard work that ensured the conference's high quality and varied programme.

SGM PRIZE MEDAL LECTURE

PROFESSOR JULIAN DAVIES is the recipient of the SGM Medal awarded annually to a microbiologist whose work has led to far-reaching impact beyond microbiology. He will deliver his talk, *Molecules, microbes & me* on Monday 26 April 2012 at the SGM Spring Conference at the Convention Centre Dublin.

Julian obtained his BSc (1953) and PhD (1956) at Nottingham University. After 3 years of postdoctoral training at Columbia University (NY) and the University of Wisconsin, he accepted a lectureship in chemistry at the University of Manchester Institute of Technology in 1959.



Increasingly interested in biology, in 1962 Julian procured a fellowship in the laboratory of Bernard Davis (Harvard Medical School) to study the streptomycin mode of action

together with Luigi Gorini and Walter Gilbert, followed by a brief collaboration with H. Gobind Khorana studying streptomycin-induced miscoding. In 1965, an NIH fellowship permitted him to work with Francois Jacob (Pasteur Institute) where he carried out genetic mapping of the regulatory genes of the *Escherichia coli lac* operon. Julian joined the Biochemistry faculty at the University of Wisconsin in 1967, and worked on antibiotic modes of action and the origins and evolution of antibiotic resistance.

In 1980, Julian became Research Director of the Biotechnology company Biogen, in Geneva, Switzerland, eventually becoming President of the Geneva operation. He left in 1985 to become Head of the Microbial Engineering unit at the Institut Pasteur (Paris). The group worked on a variety of topics, including antibiotic resistance, antibiotic production, mechanisms of bacterial and fungal pathogenicity, and horizontal gene transfer in microbes.

Julian became Professor and Head of the Department of Microbiology and Immunology at the University of British Columbia in 1992 before having to retire in 1997. He continues to teach and run a laboratory. He founded TerraGen Diversity, one of the first environmental DNA metagenomic start-up companies in 1996 (subsequently acquired by Cubist Pharmaceuticals).

Julian has served as the President of the American Society of Microbiology from 1999 to 2000 and was President of the International Union of Microbiological Societies in 2003. He is a Fellow of the Royal Societies of London and Canada, and has received a number of other awards and honours. He is Chair of the External Scientific Board of the NIH Human Microbiome Project.

Marjory Stephenson Prize Lecture

PROFESSORS PATRICK S. MOORE and **YUAN CHANG** were jointly awarded the 2012 prize and will deliver their prize lecture on Tuesday 27 March 2012 at the SGM Spring Conference at the Convention Centre Dublin.

Patrick Moore received his medical degree at the University of Utah College of Medicine and a Master's degree in public health from the University of California Berkeley. From the late 1980s through to the early 1990s, Patrick worked at the Centers for Disease Control and Prevention (CDC).

Yuan Chang received her medical degree from the University of Utah College of Medicine and completed a residency in anatomic pathology at the University of California San Francisco, followed



by fellowship training in neuropathology at Stanford. In 1994, as newly appointed faculty members at Columbia University, they jointly discovered Kaposi's sarcoma-associated herpesvirus (KSHV). They subsequently moved to the University of Pittsburgh where Patrick is a Professor of Microbiology and Molecular Genetics in the School of Medicine and Director of the Cancer Virology Program at the University of Pittsburgh Cancer Institute. Yuan is a Professor in the Department of Pathology at the University of Pittsburgh School

of Medicine. Their laboratory discovered Merkel cell polyomavirus in 2007.

They have jointly received numerous honours and awards, including the Robert Koch Prize, the Mott Prize from the General Motors Cancer Research Foundation, the Meyenburg Cancer Research Award, and American Cancer Society Basic Research Professorships.

PRIZES & AWARDS

FLEMING LECTURE

DR BILL HANAGE will deliver his lecture on Wednesday 28 March 2012 at the Society's Spring Conference at the Convention Centre Dublin.

Bill studies the evolution and epidemiology of (mainly) bacterial pathogens. He did his undergraduate degree in biochemistry at the University



of Bath, before a PhD at Imperial College London. This was then followed by postdoctoral work at Imperial College London and the University of Oxford. In 2010, he joined the faculty at Harvard School of Public Health. He is especially interested in subjects that combine clinical importance with fundamental biological questions, such as how pathogens respond to novel selective pressures in the form of antimicrobials and vaccines, and the link between transmission and virulence. He has also worked extensively on the phenomenon of homologous recombination in bacteria, where genetic material is shuffled among lineages, studying how it can be detected and its consequences for how bacteria respond to selection. Increasingly, he is involved with population genomic analyses of large numbers of very closely related pathogen isolates, to probe in detail their patterns of transmission and diversification. He works both with culture flasks and computer simulations, and would hate to have to choose between them!

Next to science, his great passion is Arsenal Football Club, in good times and bad!

Heatley-Payne Travel Award

Named in honour of British scientist Norman Heatley and US scientist William 'Jack' Payne, this scheme is offered jointly with the American Society for Microbiology (ASM). It supports the reciprocal exchange of one early-career member to present their research at the other Society's main conference and visit a laboratory in that country.

The award has been developed to strengthen the bonds between SGM and ASM and to give early-career microbiologists the opportunity to experience the best of microbiology in the exchange country.

DR JOSEPH HYSER, the US-based recipient of the Heatley-Payne Travel Award, will be joining us in Dublin to present his research and will also carry out a short research visit at the University of Leeds hosted by Stephen Griffin.

Full details of the scheme are available on the SGM website. SGM members awarded their PhD after 1 November 2007 and who are interested in applying for a 2013 award to attend the ASM General Meeting and visit a research laboratory in the US, should look out for the announcement and deadline in a future issue of *Microbiology Today*.

SGM CONFERENCE PLANNING – YOUR CHANCE TO GET INVOLVED

SGM conference scientific programmes are planned by a dedicated group of SGM members who form the divisional committee matrix. The divisions include members with interests in some aspect of one of four cross-cutting themes: microbial diversity and evolution; fundamental microbiology; translational microbiology; and infectious disease. Each member joins the committee for 3–4 years. Divisions work independently to plan conference symposia, whilst other symposia are developed with a cross-cutting theme in mind. All SGM members are eligible to join the divisions and we warmly welcome new people to the committees. If you would like to get involved with any of the divisional committees, we would very much like to hear from you.

Divisional Committee Elections 2012

For the 2012 elections, we are inviting nominations to fill 15 vacancies (see below).

Theme	Virology	Prokaryotic Micro- biology	Eukaryotic Micro- biology	Education	Irish
Microbial diversity & evolution	1 vacancy	1 vacancy	1 vacancy	2 vacancies	4 vacancies
Fundamental microbiology	1 vacancy	No vacancy	No vacancy		
Translational microbiology	1 vacancy	1 vacancy	1 vacancy		
Infectious disease	2 vacancies	No vacancy	1 vacancy		
Total	5	2	3	2	4

Nominations for the Virology, Prokaryotic and Eukaryotic Microbiology Divisions must fall into the cross-cutting themes as advertised. The Irish Division does not have cross-cutting themes, so nominations are open to individuals with any scientific interest. The Education Division are looking for people with interests in any aspect of microbiology education.

Nominations should be made using the form available on the SGM website at www.sgm.ac.uk/meetings/divisions.cfm. All nominees must be SGM Members and their nomination must be supported by two Ordinary Members. Nominations may also be made by the divisional committees. If the number of nominations exceeds the number of vacancies, elections will be held in early summer.

Please send your nominations to Jane Westwell, SGM, Marlborough House, Basingstoke Road, Spencers Wood, Reading, RG7 1AG (j.westwell@sgm.ac.uk). The closing date is **20 April 2012**. More information about the divisional committee remits and their current members is available at www.sgm.ac.uk/meetings/divisions.cfm

News from Divisions

In September 2011, 19 new members joined the Divisional Committees and we are delighted to welcome the new Division Chairs whose term of office will run from 2011 to 2013. They take over as we are organizing our first Spring Conference in Ireland and they have already been very busy co-ordinating a packed scientific programme.

Prof. Peter Sudbery

Eukaryotic Microbiology Division

Peter's research focuses on the cellular and molecular mechanisms of hyphal growth in the human fungal pathogen *Candida albicans*. His group combines genetic and biochemical approaches with high-resolution imaging in living cells. Previously he carried out research on the control of mitosis in the acellular slime mould *Physarum polycephalum*, cell cycle control in the budding yeast *Saccharomyces cerevisiae*, and genetic analysis and recombinant protein expression in the methylotrophic yeast *Hansenula polymorpha*. He also has an interest in human genetics and has authored a text book on the subject, now in its third edition. Peter has been at Sheffield University since 1977 where he is now Roper Chair of Genetics. As Chair of the Eukaryote Division, his mission is to use SGM to foster interaction between the disparate communities working on microbial eukaryotes. In addition, he will seek to encourage interaction between these communities and the Prokaryotic Microbiology and Virology Divisions of the SGM to share insights into common biological processes.



Dr Nick Dorrell Prokaryotic Microbiology Division

Nick Dorrell originally trained as a pharmacist at the University of Bath. After 2 years working in hospital



Dr Paul Duprex Virology Division

Paul undertook a PhD working between The

Queen's University of Belfast (QUB) and the Institute for Animal Health (IAH) in Pirbright. Working with Martin Ryan at IAH opened his eyes to the power of reverse genetics and he learned how to recover recombinant foot-and-mouth disease virus in high-containment facilities. Sam Martin and Elizabeth Hoey were his mentors at QUB and there he engineered a non-pathogenic positive-strand RNA virus to express foreign proteins with a view to developing novel veterinary vaccines. Understanding viral pathogenesis and attenuation by manipulating virus genomes has always been a key focus of his studies, and working with Bert Rima in Belfast introduced Paul to negative-strand RNA viruses. It was there that he established and generated reverse genetics systems for a number of paramyxoviruses and transitioned to a Lectureship in Molecular Virology in 1999.

Paul was awarded the Hellen C. Levitt Visiting Professorship in 2001 and spent time working in the group of Roberto Cattaneo in the Molecular Medicine Program at Mayo Clinic in Minnesota, developing replicating paramyxovirus vectors for cancer gene therapy. In 2006–2007, he returned to the USA to work for TransForm Pharmaceuticals/Johnson and Johnson in Boston where he focused on a measles virus vaccine project supported by the Bill and Melinda Gates Foundation. Currently, he is a Senior Lecturer at QUB and an Associate Professor of Microbiology at Boston University School of Medicine. His interests in high-containment virology continue to develop within the National Emerging Infectious Disease Laboratories where he is the Director of Cell and Tissue Imaging.

Paul has always been a strong supporter of SGM; he is currently a member of the Editorial Board of JGV and represents SGM on the European Society of Virology Scientific Advisory Board. He believes education, training, outreach and internationalization are the foundation on which a strong and vibrant virology community is built and aims to make these key themes during his tenure as Chair of the SGM Virology Division.

pharmacy, he returned to Bath to study DNA repair in *Escherichia coli*, completing his PhD in 1993. He joined Brendan Wren's research group at St Bartholomew's Hospital in 1994, and worked on many different aspects of bacterial pathogenicity in *Brucella* and *Yersinia* species and *Helicobacter pylori*. He joined the London School of Hygiene and Tropical Medicine (LSHTM) in 1999 and is continuing to study bacterial pathogenicity in both *H. pylori* and *Campylobacter jejuni*. He has been the Course Director for the LSHTM's Medical Microbiology MSc Course since 2005.

DIVISIONS

Dr Kevin Kavanagh Irish Division

Kevin is a Senior Lecturer in the Department of Biology, National University of Ireland Maynooth where he teaches on a variety of undergraduate programmes. His research interests are centred on how the innate immune system of mammals responds to microbial pathogens. In addition, he has interests in examining structural and evolutionary similarities between the innate immune response of mammals and the immune system of insects. As part of this work he developed the *Galleria mellonella* model as a rapid means of evaluating the virulence of microbial pathogens and for screening antimicrobial drugs.



Dr Sara Burton Education Division

Sara brings experience to the Education Division from many aspects of her varied career in government laboratories, industry and, latterly, in higher education. Her formal teaching experience began with the Open University when she taught in the south-west England region. More recently, she has worked at the University of Exeter, in Biosciences, primarily as a research and teaching fellow and then as Senior Tutor.



Sara is delighted to chair the Education Division in these times of huge change within the Higher Education sector. The Division will support SGM in responding to new HE challenges, particularly with respect to managing student expectations and the employability challenges in the UK and beyond in these demanding times.

GRANTS

MAKE THE MOST OF YOUR MEMBERSHIP

SGM CONFERENCE GRANTS

SGM conferences are the ideal place to develop research ideas, communicate results, catch up on other people's research findings and network with fellow microbiologists. We offer several grant schemes to support attendance at our conferences. The 2012 closing dates are:

Spring Conference (Dublin) –

23 March 2012

Autumn Conference (Warwick) –

31 August 2012

Irish Division (Cork) –

9 November 2012

POSTGRADUATE STUDENT CONFERENCE GRANTS

POSTGRADUATE STUDENT CONFERENCE GRANTS – All PG Student Associate Members, resident and registered for a PhD in an EU country, are eligible to apply for a grant to support their attendance at one SGM conference each year. Grants contribute towards travel, registration and accommodation expenses. The student need not be presenting their research, so it is an ideal introduction to scientific meetings at little or no cost to themselves or their supervisor's budget.

TECHNICIAN CONFERENCE GRANTS

TECHNICIAN CONFERENCE GRANTS – All Associate Members who are technicians are eligible to apply for a grant to support their attendance at one SGM conference each year. Applicants need not be presenting work at the conference. Some microbiology technicians who are not members of SGM may also apply for a grant to attend a Society conference.

UNDERGRADUATE STUDENT CONFERENCE GRANTS

UNDERGRADUATE STUDENT CONFERENCE GRANTS – UG Student Members who have results to present from either their final year or vacation project can apply for funding to attend one SGM conference per year. The grant contributes towards travel and accommodation costs (registration is free), and applicants must have their abstract accepted for presentation. Students need not be the first author, but should be present at the poster session to talk about their work.

RETIRED MEMBERS GRANTS – These cover accommodation and the Society Dinner at one SGM conference a year.

SGM has a wide range of grant schemes to support microbiology. See www.sgm.ac.uk for details. Enquiries should be made to: **Grants Office, SGM, Marlborough House, Basingstoke Road, Spencers Wood, Reading RG7 1AG** (tel. 0118 988 1807; fax 0118 988 5656; email grants@sgm.ac.uk).

TRAVEL & MEETINGS

PRESIDENT'S FUND FOR RESEARCH VISIT GRANTS

PRESIDENT'S FUND FOR RESEARCH VISIT GRANTS – Many researchers reach a point where they would benefit from a visit to another lab to learn a new technique or gain access to specialist equipment and knowledge. SGM recognizes this need and offers grants of up to £3,000 to support early-career microbiologists who are planning a short research visit to another laboratory (minimum visit 4 weeks, maximum visit 3 months). Closing dates for applications: **16 March** and **21 September 2012**

SCIENTIFIC MEETINGS TRAVEL GRANTS

SCIENTIFIC MEETINGS TRAVEL GRANTS – Support for early-career microbiologists wishing to present work at scientific and education meetings in the UK or overseas. Graduate research assistant, lecturers and teaching fellows (within 3 years of first appointment) in the UK and Ireland, and postdoctoral researchers (within 3 years of first appointment) and postgraduate students in the EU are eligible to apply. Retrospective applications are not considered.

SHORT REGIONAL MEETING GRANTS

SHORT REGIONAL MEETING GRANTS – Contribution of up to £2,000 towards the costs of running a regional or specific topic microbiology meeting.

EDUCATION & DEVELOPMENT

NATIONAL

PRACTICAL TEACHING AIDS

PRACTICAL TEACHING AIDS – Small grants to members for developments likely to lead to an improvement in the teaching of any aspect of microbiology relevant to secondary or tertiary education in the UK.

PUBLIC ENGAGEMENT WITH MICROBIOLOGY AWARDS

PUBLIC ENGAGEMENT WITH MICROBIOLOGY AWARDS – Up to £1,000 to support projects that promote public engagement with microbiology.

GRADSCHOOL GRANTS

GRADSCHOOL GRANTS – PG Student Associate Members who are not eligible for a free place on a Vitae (www.vitae.ac.uk) personal development course (National GRADSchool) can apply for a grant from SGM to cover full course fees. Retrospective applications are not considered.

CAREER CONFERENCE GRANTS

CAREER CONFERENCE GRANTS – Contribution towards costs of travel and registration fee for UG Student members to attend a Life Sciences Careers Conference. Next conference: *1 March 2012, University of Westminster, London.*

SEMINAR SPEAKERS FUND

SEMINAR SPEAKERS FUND – Small grants to cover the travel and other expenses of up to two speakers on microbiological topics in annual departmental seminar programmes.

STUDENT SOCIETY SPONSORED LECTURES

STUDENT SOCIETY SPONSORED LECTURES – Small grants to cover the travel and other expenses of up to two speakers on microbiological topics at student society meetings.

INTERNATIONAL

INTERNATIONAL DEVELOPMENT FUND

INTERNATIONAL DEVELOPMENT FUND – Supports members to provide training courses, publications and other help to microbiologists in countries with economies defined by the World Bank as low-income or lower-middle-income.

Closing dates: **16 March** and **21 September 2012.**

MEDICAL MICROBIOLOGY SUPPORT GRANTS

ELECTIVE GRANTS

ELECTIVE GRANTS – Funding for medical/dental/veterinary students to work on microbiological projects in their elective periods.

Closing dates: **16 March** and **21 September 2012.**

TRAINEE SUPPORT GRANTS

TRAINEE SUPPORT GRANTS – Funding for SGM members carrying out small lab-based microbiology projects during either foundation or speciality postgraduate medical training. Up to £3,000 is available towards the consumables costs of a project.

Closing dates: **16 March** and **21 September 2012.**

Microbiology Outreach Prize sponsorship

Yakult UK have generously extended their sponsorship of the Microbiology Outreach Prize for a further 3 years.

The prize is awarded to a microbiologist who has undertaken high-quality outreach activities over a period of 2–5 years.

In 2012, the winner will receive a cash prize and give a talk about their activities at the SGM Autumn Conference at Warwick University on 4 September 2012.

If you have been delivering outreach activities and would like to be considered for the award, please check out the prize guidelines at www.sgm.ac.uk/grants/outreach.cfm

The closing date for nominations is **16 March 2012.**



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RNA interference. Studio Macbeth / SPL

Lamarckian challenge to Darwinian evolution

Acquired immunity to viruses can be passed down the generations independently of DNA, according to a new study. Some time before Darwin's theory of evolution by natural selection, Jean Baptiste Lamarck proposed that species evolve when individuals adapt to their environment and pass these new adaptations to their progeny. With the discovery of hereditary genetics, which supported Darwin's theory, Lamarck's ideas became overshadowed. While there is evidence to suggest that acquired traits can be inherited, no one could describe a plausible biological mechanism for this phenomenon. Researchers at Columbia University Medical Centre in the US have shown that RNA interference (RNAi), which turns down the expression of specific genes by destroying mRNA before it is translated, is fundamental to the inheritance of acquired traits. The RNAi mechanism can be triggered by viral RNA, conferring resistance to viral infection, which is passed to the offspring, despite there being no genomic change. In *Caenorhabditis elegans* where the RNAi machinery had been switched off, offspring still had the ability to fend off viral infection so must have acquired the trait from the parent. Lamarckian inheritance could indeed be advantageous since it does not involve a change in the function of a gene and is a reversible process. The fundamental RNAi machinery that underlies this process exists throughout the animal kingdom, including humans, and is the first example of extra-chromosomal inheritance to date.

Cell doi:10.1016/j.cell.2011.10.042

Sarah Maddocks, Cardiff Metropolitan University

Measles targets cancer cell receptor

The specific receptor used by the measles virus to break into throat cells, where it provokes a cough, allowing its spread between hosts, has been identified. Measles is a highly infectious virus that affects over 10 million children each year. One trait underlying the virus's ability to infect and spread so rapidly is the manner in which it crosses epithelial cells to emerge in the trachea, enabling it to be spread as aerosolized particles in coughs and sneezes. Scientists from Europe, Asia, and North America recently discovered that the ability of the measles virus to infect cells correlated with the presence of the epithelial cell receptor, nectin-4, on tracheal cells. The researchers showed that blocking production of nectin-4 in these cells also blocked entry of the measles virus. As well as providing insight into the working of a global killer, this research may also have implications for the treatment of cancer as nectin-4 is commonly found on the surface of lung, breast and ovarian cancers. This raises the possibility of future, measles-based, treatments for these conditions.

Nature doi:10.1038/nature10639

David Guymer, Newcastle University

MICROBIOLOGY TODAY

SOME RECENT

SGM MEMBERS HIGHLIGHT

Fungal culprit for fatal bat disease

A fungus has been confirmed as the agent responsible for the decimation of American bat populations. White nose syndrome (WNS) is a deadly disease that threatens to wipe out bat populations in North America. The disease was first observed among bats hibernating in a cave in 2006, and is estimated to have killed more than a million bats in the subsequent 3 years. It has been spreading steadily and encroached into Canada in 2010. An association between WNS and the cold-adapted



Little brown bat with WNS. Ryan von Linden/New York Department of Environmental Conservation

fungus *Geomyces destructans* was first noted in 2008. A new study by US Geological Survey scientists and partners has confirmed that the fungus is responsible for WNS and not merely opportunistically attacking weakened bats. The researchers also showed that *G. destructans* is spread directly bat-to-bat. Bats are often seen as villains in the spread of infectious diseases, but they are also vital pest controllers, eating up to two-thirds of their body weight in insects every day. Identification of the cause of WNS paves the way for development of control methods.

Nature doi:10.1038/nature10590

Janet Daly, University of Nottingham

Plasma goes viral

Plasma beams have the potential to inactivate and prevent the replication of adenoviruses, according to a recent study. Researchers at the Max-Planck Institut für extraterrestrische Physik and Technische Universität München found that only one in a million viruses could still replicate following exposure to plasma – the fourth state of matter. Adenoviruses predominantly target the respiratory system and are encased in a protein layer which makes them hard to inactivate. For this reason, viral infections are normally treated by tackling the symptoms rather than targeting the virus directly. The researchers exposed different concentrations of adenoviruses to plasma for 240 seconds and incubated them for 1 hour. Treated and untreated adenoviruses were then used to infect two separate cell lines. By monitoring infection via a green fluorescent protein produced by the virus, the researchers found that virtually all of the plasma-treated viruses were inactive. Plasma could potentially be used in a hospital environment as a hand wash, or even inhaled directly to treat viruses within the lungs.

Journal of Physics D: Applied Physics doi: 10.1088/0022-3727/44/50/505201

Elizabeth Andrew, Newcastle University



Plasma. iStockphoto / Thinkstock

If you're interested in writing a Microshort for a future issue of *Microbiology Today*, please contact Laura Udakis on l.udakis@sgm.ac.uk

Climate change: a topic of (bacterial) conversation

Bacterial 'group decisions' could influence the global carbon cycle – and therefore Earth's climate – on a potentially huge scale, according to researchers from Woods Hole Oceanographic Institution (WHOI). Sticky detritus, accumulated from the waste of plankton in the oceans, forms carbon-containing particles heavy enough to sink. This creates a literal 'sink' of organic carbon at the sea-bed – a natural phenomenon that separates carbon away from the atmosphere for up to thousands of years. Many marine bacteria, however, use hydrolytic enzymes to degrade these sinking detritus particles. This provides a source of nutrition for the bacteria and results in lighter-weight particles suspended at shallower depths which are likely to release carbon back into the atmosphere. WHOI marine biogeochemists have discovered that this microbial degradation is under the control of quorum sensing signals that co-ordinate enzyme secretion within bacterial communities. Understanding this bacterial communication mechanism should help to improve future models of carbon flux and climate change.

Environmental Microbiology Reports doi: 10.1111/j.1758-2229.2011.00281.x

Zoe Freeman, University of Bath



Digital Vision / Thinkstock

Uncovering how probiotic yoghurt works

Scientists have uncovered a mechanism by which probiotic bacteria interact with their consumer, i.e. by altering gene expression and metabolism in common gut microbes. Bacteria found in probiotic yoghurt are thought to improve digestive health, but the ways in which these microbes confer such benefits are poorly understood. Recently, the gut microbiomes of healthy adults, and of mice harbouring human gut microbes, were examined over a 4 month period. For 7 weeks of the study, the participants consumed a commercially available yoghurt containing five common strains of probiotic bacteria. The team from Washington University School of Medicine found that the yoghurt did not alter the microbial populations in the participants but instead caused resident gut microbes to increase expression of genes involved in carbohydrate metabolism. The same result was observed in both humans and mice. Although it remains unclear exactly how probiotic bacteria may benefit our health, these results improve our understanding of the ways they can interact with our native gut flora.

Sci Transl Med doi:10.1126/scitranslmed.3003291

Andrew Turner, Princess Royal Hospital, Telford

OTHER EVENTS

IRISH DIVISION FUTURE

Autumn 2012
University of Warwick
3–5 September 2012

University College Cork
14–16 November 2012
Marine microbiology and biotechnology: biodiscovery, biodiversity and bioremediation
Organizer: Niall O'Leary (n.oleary@ucc.ie)

University College Dublin
21–23 March 2013
Gene regulation and microbial pathogenicity
Organizer: Tadhg Ó'Croíinín (tadhg.ocroinin@ucd.ie)

SGM is supporting the following meetings:

10th Meeting on the biology and pathology of hepatitis C virus
Rydal Hall, Cumbria
13–15 April 2012

Bacterial spore formers
Royal Holloway, University of London
16 April 2012
www.sporesconference.com

Geomicrobiology & its significance for biosphere processes
Manchester Interdisciplinary Biocentre
19–20 April 2012
www.minersoc.org/pages/meetings/EMG-SGM/EMG-SGM.html

Young microbiologists symposium on microbe signalling, organization & pathogenesis
University College Cork, Ireland
21–22 June 2012

How bugs kill bugs: progress and challenges in bacteriocin research
University of Nottingham
16–18 July 2012
www.biochemistry.org/MeetingNo/SA140/view/Conference

European Microscopy Congress
Manchester Central Conference Centre
16–21 September 2012
www.ms.org.uk/events/EMC2012



SCIENCE

WWW.SGM.AC.UK/MEETINGS — DELIVERING MODERN MICROBIAL SCIENCE

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SPRING 2012

CONVENTION CENTRE DUBLIN, IRELAND

26–29 MARCH 2012

www.sgmublin2012.org.uk



If you have a device that can read QR codes, access the conference website here.

Symposia

Viral zoonoses | Biocontrol of diseases | International cardiovascular infection symposium (joint with Royal College of Surgeons in Ireland) | Medical devices and biomaterials | Innate barriers to disease | Climate change and infectious disease | Hospital-acquired infections | Bacterial zoonoses | Food-borne pathogens: survival of the fittest | News on the nitrogen cycle | Waste management and recycling | Imaging the living microbe | Phylogeography | Recoding in microbial gene expression | E-learning | UK Clinical virology network meeting

Workshops

New media
Virology workshops: *DNA viruses, Innate barriers, RNA viruses, Retroviruses, Plant virology*

Prize Lectures

Mon 26 March SGM Prize Medal *Dr Julian Davies* | Peter Wildy Prize in Microbiology Education *Dr Vincent Racaniello*

Tue 27 March Marjory Stephenson Prize Lecture *Profs Patrick Moore & Yuan Chang*

Wed 28 March Fleming Lecture *Dr William Hanage*

Early-career microbiologists

Enhance your conference experience by attending the pre-conference networking workshop and supper on Sunday 10 April. Take part in some fun activities to improve communication skills and add value to your conference experience.

Sir Howard Dalton Young Microbiologist of the Year Competition

Short-listing for a place in the 2012 finals of the *Sir Howard Dalton Young Microbiologist of the Year Competition* takes place in Dublin. Eligible postgraduate students and postdoctoral scientists, presenting in selected sessions, will be able to nominate themselves when they arrive in Dublin.

Grants

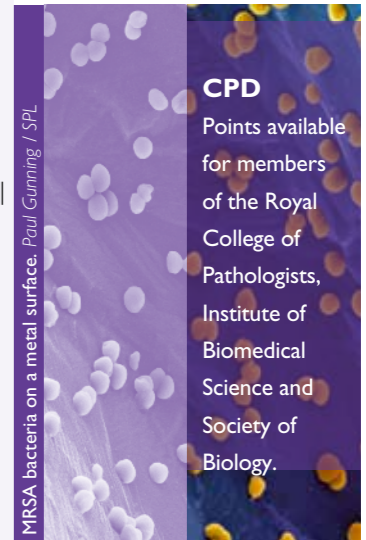
Conference grants are available to eligible SGM Associate Members who are postgraduate students, technicians or retired and to Undergraduate Members who are presenting work at the conference.

Venue

The Convention Centre Dublin is Ireland's first purpose-built conference centre which offers state-of-the-art technology and also meets the highest standards of environmental sustainability. Its stunning design includes a glass atrium with views across Dublin and beyond.

Social event

The conference dinner will take place on Tuesday 27 March at Ireland's number one tourist attraction, the Guinness Storehouse (www.guinness-storehouse.com). The ticket price will include a drink on arrival, self-guided tour of the venue and a three-course buffet.



CPD
Points available for members of the Royal College of Pathologists, Institute of Biomedical Science and Society of Biology.

Accommodation

Accommodation can be booked through Reservation Highway who have secured a range of rooms to suit all budgets throughout Dublin at discounted rates. All participants are strongly advised to book early, as Dublin is hosting several other events during this time.

Registration

Register online at www.sgmublin2012.org.uk or complete (and return) the downloadable PDF. Earlybird registration rate deadline: **24 February 2012**. Registration fees include refreshments, lunch, drinks receptions, the abstracts CD, exhibition entry and all conference literature. Specially discounted rates are available for: SGM Associate/Postgraduate Student Associate Members.



DAVID J. GRIFFITHS

Retroviral placental syncytins: old dogs with new tricks

"I don't want to achieve immortality through my work ... I want to achieve it through not dying."

Woody Allen

Although retroviruses are often associated with disease, retroviral DNA sequences have been found in many mammalian genomes. Far from being inert pieces of junk DNA, some of these sequences have acquired important functions, for example in the development of the placenta.

WE MIGHT NOT THINK of retroviruses as immortal but they are certainly ancient. We recognize them today as deadly pathogens that cause diseases such as AIDS and cancer. However, our evolutionary ancestors were also subjected to recurring waves of retroviral infection. Some of those viruses managed to infect germ cells and became permanently fixed (or 'integrated') in the germ-line of their hosts so that they were passed on through successive generations. The genomes of modern-day mammals now contain large numbers of these inherited retroviral sequences, which are known as endogenous retroviruses (ERVs).

ERVs can be grouped into several families, each representing a different ancestral infection. In addition, some ERVs were further amplified after their initial integration and are now present as many hundreds of copies scattered at multiple chromosomal sites. Therefore, ERVs now comprise a large proportion of the genome in many species; for example, human ERVs (HERVs) account for 8% of the human genome. This is a significant amount, given that only around 1.5% of the genome is thought to encode proteins.

In the time since they integrated, the large majority of ERVs have lost the ability to make viral proteins due to the accumulation of mutations and deletions. Until a few years ago, a commonly held view was that ERVs were simply 'junk DNA'; inert souvenirs of our ancestors' encounters with retroviruses. However, recent studies have revealed that some of these ancient viruses have acquired

new roles and now serve important physiological functions for their hosts. Most notably, ERVs in a number of species contribute to the development of the placenta.

PLACENTAL CELL FUSION

The placenta is a transient and complex organ that provides the interface between mother and fetus. It has several important functions, including nutrient and waste exchange, immune regulation and the production of hormones that help to sustain pregnancy. Different lineages of mammals have evolved placentas with markedly diverse structures. A key feature of the human placenta is a continuous multinucleated cell layer called the syncytiotrophoblast, which forms the outermost layer of fetal-derived tissues and makes direct contact with the maternal blood. During pregnancy, the syncytiotrophoblast is continuously renewed by the fusion of underlying fetal cytotrophoblast cells.

Cytotrophoblast cell fusion is a carefully regulated process involving numerous proteins, but the fusion event itself appears to be driven by envelope (Env) glycoproteins encoded by ERVs. For infectious retroviruses, Env is used to fuse viral and cellular membranes so that the viral capsid can enter the cell. Env-mediated fusion requires a virus-specific receptor on the plasma membrane of the target cell. In the placenta, ERV Env proteins are utilized to induce cell-to-cell fusion. Because they drive the formation of syncytia, these proteins have been named syncytins.

HUMAN SYNCYTINS

Two human syncytins have been identified, designated syncytin-1 and syncytin-2. Each is encoded by a single provirus of the HERV-W and HERV-FRD families, respectively. Syncytin-1 is functionally conserved in all ape species and is thought to have integrated into the primate lineage around 25 million years ago.

Fluorescence deconvolution micrograph of a cross-section through a mature chorionic villus, showing connective tissue (green), proteins associated with embryo development (red) and syncytiotrophoblasts, (blue). The syncytiotrophoblast layer forms the outermost fetal component of the placenta and contributes to its barrier function.
R. Bick, B. Poindexter, UT Medical School / Science Photo Library

Syncytin-2 is older and probably entered the germ-line over 40 million years ago because it is conserved in Old and New World primates. Notably, the other retroviral genes in these proviruses are inactivated by numerous mutations. Only the Env (syncytin) genes and the viral promoters that control their expression remain intact and functional.

Evidence that syncytins have retained their fusogenic activity comes from cell culture models in which freshly isolated cytotrophoblast cells spontaneously generate multinucleated syncytia after 2–3 days in culture. If syncytin-1 and -2 expression is inhibited in these cells, fusion is blocked. Experimental induction of syncytin expression in non-placental cell lines also results in syncytium formation. In other studies, the cellular receptors for both syncytins have been identified. The two syncytins and their receptors are preferentially expressed at high levels in the placenta. Syncytin-2 in particular has a highly specific expression pattern as it is detectable only in cytotrophoblast cells, while its receptor is produced only in the syncytiotrophoblast.

Collectively, these findings strongly suggest a role for syncytins in placental development, but because the HERV-W and HERV-FRD families are present only in primates, syncytin-1 and -2 cannot be studied easily in animal models. Nevertheless, the case for their involvement in placental development has been further strengthened by analysis of disorders in which syncytiotrophoblast development is impaired, such as pre-eclampsia and Down's syndrome. These conditions are associated with abnormal expression of syncytin-1 and -2. It appears that this is due to dysregulation of the differentiation pathways that regulate their production, rather than mutations in the syncytin proteins.

SYNCYTINS IN MICE

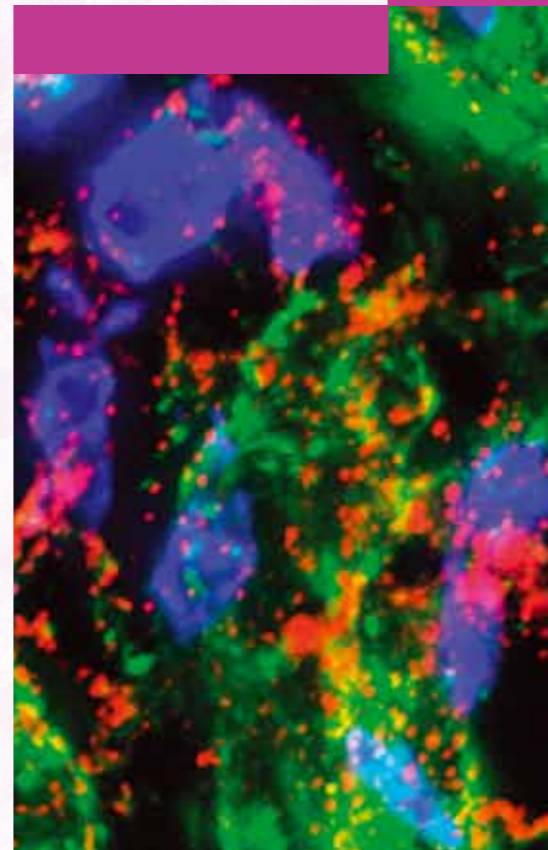
As syncytins-1 and -2 are present only in primates, this begs the question: how do other species manage to produce placental syncytiotropho-

blasts? Remarkably, it appears that different retroviral Env proteins have been captured independently to fulfil this role in different mammals. So far, syncytins have also been described in rodents (including mice, rats, hamsters and voles) and lagomorphs (rabbits and hares), with each mammalian order exploiting an entirely different ERV.

Studies in mice have been particularly informative. Like humans, mice have two fusogenic syncytin proteins, denoted syncytin-A and -B, which show placenta-specific expression. They are encoded by otherwise defective ERV proviruses and probably entered the Muridae lineage around 20 million years ago. Crucially, the mouse syncytins are unrelated to each other, and to the human syncytin proteins, and therefore represent independent infections that were acquired at completely different times in the evolutionary past.

The availability of techniques to delete, or 'knock-out', specific genes in mice has allowed the role of murine syncytins to be examined in more detail in pregnant animals. Whereas human placentas have a single syncytiotrophoblast layer, in mice there are two. If syncytin-A is deleted, mice die during mid-gestation due to a failure of one of the syncytiotrophoblast layers to develop. Mice lacking syncytin-B display a less severe phenotype that is not always lethal, but the offspring that survive have reduced birth weight. Notably, syncytin-B knockout mice have a defect in the second syncytiotrophoblast layer. Therefore, each mouse syncytin has been co-opted by the host to contribute independently to the generation of a different fused cell layer.

A normal sheep fetus and placenta at mid-gestation. The ovine placenta contains multiple placentomes (ring-shaped structures in the figure), which are the sites where fetal and maternal tissues attach. Dr David Buxton, Moredun Research Institute



“It is therefore possible that these ancient viral proteins could be making multiple contributions to mammalian physiology.”

ERVS IN SHEEP

ERVs have also been associated with placentation in sheep. However, the ovine placenta does not have a syncytiotrophoblast, but instead has smaller patches of fused cells, called syncytial plaques, which are hybrids of fetal and maternal cells. One family of ovine ERVs is closely related to an exogenous retrovirus called Jaagsiekte sheep retrovirus (JSRV) that still infects sheep today and causes lung cancer. The endogenous relatives of JSRV, denoted 'enJSRV', are expressed primarily in the reproductive tract of the ewe. Experiments in which enJSRV Env expression is blocked *in utero* result in the failure of implantation and early fetal death, demonstrating that enJSRV Env has an essential role in ovine pregnancy. However, it is not yet clear whether one or several enJSRV proviruses are involved or whether the essential function contributed by enJSRV is the supply of fusogenic activity.

DO SYNCYTINS HAVE OTHER ROLES?

Env proteins of ERVs are thought to play additional roles in their hosts, including resistance to viral infection and immune regulation. Immune responses in the placenta are tightly controlled to prevent rejection of the fetus, which is 'foreign' to the maternal immune system. Many retroviral Env proteins, including syncytin-2 and syncytin-B, have immunosuppressive properties in addition to their fusogenic activity and it is possible they also play a role in fetal immune tolerance. Moreover, mammals have some other uses for multinucleated cells, such as in the development of bone and skeletal muscle, and initial results have suggested that syncytins are involved in those processes too. It is therefore possible that these ancient viral proteins could be making multiple contributions to mammalian physiology.

THE IMPACT OF SYNCYTINS

Research on syncytins has revealed that mammals have appropriated ERV proteins on several independent occasions to provide essential functions in the placenta, providing a remarkable example of convergent evolution. This work has opened new avenues for studying and understanding placental physiology and pathology, and provides a fascinating insight into the relationship of these ancestral ERVs with their modern-day hosts. In addition, it has been speculated that the specific properties of the different ERVs were perhaps instrumental in driving the evolution of the diverse placental structures that are found in different orders of mammals. Retroviruses now have an indispensable role in many species. Perhaps they have achieved a kind of immortality after all.

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Background SEM of placental villi. Steve Gschmeissner / Science Photo Library

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Microbiology of the neonate: getting to the guts of it

Coloured scanning electron micrograph of *Bifidobacterium pullorum*. Scimat / Science Photo Library

THE HUMAN gastrointestinal (GI) tract is one of the most heavily populated microbial ecosystems, hosting a complex bacterial community (termed the 'gut microbiota') comprising millions of millions of bacteria (10^{12} cells per gram of colonic contents). Numerically at least, humans are more microbial than mammal – with ten times more bacterial cells than mammalian cells in the human body (perhaps size does matter after all). Importantly, however, these microbial members of our guts impact our health, well-being, utilization of some dietary components and response to certain medical treatments; and are not simply unwelcome squatters or parasites.

ACQUISITION OF THE GUT MICROBIOTA

Traditionally it was thought that prior to birth the infant gut was sterile, microbiologically, and that initial acquisition of the microbiota occurred during birth – with exposure to maternal and/or environmental microbes. Recent data challenges the sterile fetal gut theory, suggesting *in utero* microbial exposure (especially related to the levels and types of bacteria in amniotic fluid) has been associated with pre-term birth and neonatal outcomes.

Due to the non-invasive nature of sample collection, faecal samples

ANNE MCCARTNEY

Bottle-feeding. Photodisc / Thinkstock

It is a well-known fact that most of the cells in the human body are not mammalian but microbial, but when and how do we acquire our microbiota?

have generally been used for studying gut microbiology, including the acquisition and development of the infant gut microbiota. A number of factors (both host and environmental) have been shown to affect the infant gut microbiota – including gestational age, birth mode, host genetics, exposure to bacteria, country of origin, life-style and diet.

STAGES OF DEVELOPMENT: GLOBAL UNDERSTANDING

Acquisition and bacterial succession of the neonatal gut microbiota have generally been categorized into three distinct stages.

1. Initial exposure during the birthing process and first few hours of postnatal life, resulting in a microbiota largely comprised of facultative anaerobes which actively reduce the redox potential of the gut and facilitate colonization by strict anaerobes.

2. During exclusive milk feeding, with differences observed between breast-fed and formula-fed infants;



Bifidobacterium is generally associated with this phase.

3. Introduction of solid food into the diet, i.e. weaning, which is a transitional period that leads to diversification of the gut microbiota, which stabilizes to a 'climax' community (or adult-like microbiota) around 2 years of age.

TIMING (GESTATIONAL AGE) AND MODE OF BIRTH

Colonization of the gut of pre-term (<37 weeks gestation) and extremely low birth weight (ELBW) neonates is generally delayed and often comprises less diversity than that of full-term neonates. This probably reflects the administration of antibiotics to pre-term and ELBW neonates and their being in the neonatal intensive care unit rather than maternity wards or home. However, it may also reflect the relative immaturity of the pre-term gut compared to that of full-term neonates. Additionally, *in utero* exposure and/or maternal microbiota or host genetics (factors correlated with pre-term delivery) may also play a role.

Caesarian section (CS) delivery has also been associated with delayed acquisition and development of the infant gut microbiota. In addition,

CS-born infants have been shown to harbour lower levels of clostridia than vaginally delivered infants (not only during early infancy, but even at 7 years of age). Natural birthing (vaginal delivery) includes neonatal exposure to the maternal microbiota of the birth canal, vagina and gut (due to defecation during delivery). Accordingly, the maternal microbiota is a major source of bacterial exposure during natural childbirth. Furthermore, the length of time in labour (i.e. birthing of the baby) during vaginal delivery is considered to be another factor in microbial acquisition – with longer labour meaning increased exposure time.

HOST GENETICS

Whilst each individual harbours their own unique gut microbiota, greater similarity is seen between the microbiotas of genetically related individuals than those of unrelated healthy humans. Environmental factors are likely to be involved, but genetically determined host factors also play a role. Mucin production and receptor sites on the epithelial surface of the gut are the prime candidates for host-related factors that impact the acquisition and development of the gut microbiota. Blood group has been shown to be linked to mucin glycopolypeptides and distribution of the carbohydrate structures of GI mucus, which have been shown to impact adhesion of probiotic bacteria and are suggested to have a protective role in relation to a number of microbiologically related GI illnesses (e.g. peptic ulcers, *Helicobacter pylori*; diarrhoea, *Vibrio cholerae*, *Escherichia coli*).

COUNTRY OF ORIGIN AND LIFE-STYLE

Infants born in developing countries generally display both faster acquisition and greater diversity of

their gut microbiota than those in developed countries. More detailed studies comparing specific bacterial populations (e.g. *Bifidobacterium* species; enterobacterial populations) harboured by infants from different countries have also shown distinctions between developed and developing countries. More recently, the EU INFABIO project demonstrated that country of origin (Germany, Italy, Scotland, Spain or Sweden) had the greatest impact on the faecal microbiota of 6-week-old infants, with greater *Bifidobacterium* predominance associated with infants from northern European countries. Feeding regime, mode of delivery and exposure to antibiotic treatment were also shown to impact neonatal gut microbiota in the EU INFABIO project.

Lifestyle choices affect the gut microbiota, particularly those relating to antibiotic usage (or restriction thereof, as is the case with an anthroposophic life-style), diet and exposure to farm animals. Infant diet is probably the most studied factor with respect to development of the gut microbiota, most notably during exclusive milk feeding (i.e. pre-weaned infants).

MICROBIOTA OF BREAST-FED AND FORMULA-FED INFANTS

Human milk not only provides the neonate with its nutritional requirements, but also contains a number of other components that impact the gut microbiota, health and well-being of the infant (including human milk oligosaccharides and maternal antibodies). There is some discrepancy between different studies comparing the gut microbiota of breast-fed and formula-fed infants; however, the general consensus is that they are distinguishable. Breast-fed infants are associated with a *Bifidobacterium*-predominant microbiota, while formula-fed infants generally harbour a more diverse microbiota with significantly higher levels of certain *Clostridium* clusters (e.g. clusters XIV, I and II). Relatively recent fortifications of infant formulae, particularly prebiotic supplementations, have aimed to elicit gut microbiota and/or function (stool frequency and/or consistency) that better resemble that of breast-fed infants, and this is reflected in the commercial availability of such formulae.

WEANING AND DEVELOPMENT OF 'CLIMAX' MICROBIOTA

Introduction of solid food to the infant diet (which normally occurs between 4 and 6 months of age) delivers a more complex range of nutrients and results in diversification of the infant gut microbiota. It is also worth noting that this transitional period (of diet and microbiota) coincides with the maturation of infant immune function, and may also correspond with reduction or withdrawal of breast milk from the diet of breast-fed infants and/or introduction of follow-on formula milk to the diet. In general, weaning is associated with a shift in the gut microbiota, with *Bacteroides*, *Clostridium* and *Eubacterium* species becoming more predominant members.

Few studies have examined the impact of weaning on the infant gut microbiota in detail. Two such studies, recently published, demonstrated that the initial weaning period was a transitional period microbiologically and that convergence of the infant gut microbiota was evident after initial weaning towards that of the so-called 'climax' or adult-like gut microbiota. Interestingly, weaning has also been shown to elicit diversification within the *Bifidobacterium* population of infants.

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Coloured scanning electron micrograph of a Y-shaped *Bifidobacterium* sp. BSIP / Science Photo Library

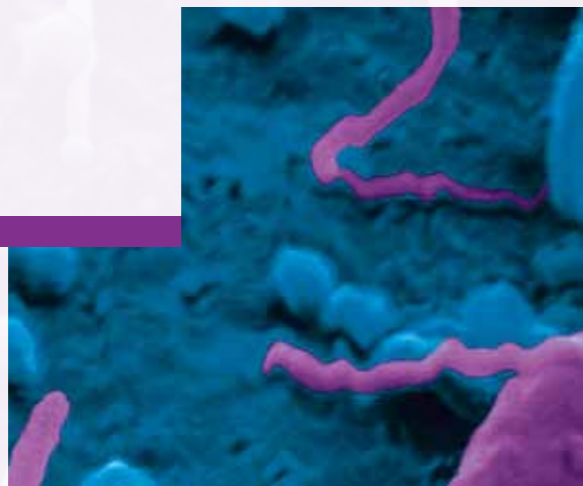


“Breast-fed infants are associated with a Bifidobacterium-predominant microbiota, while formula-fed infants generally harbour a more diverse microbiota with significantly higher levels of certain Clostridium clusters.”



Breast-feeding, iStockphoto / Thinkstock



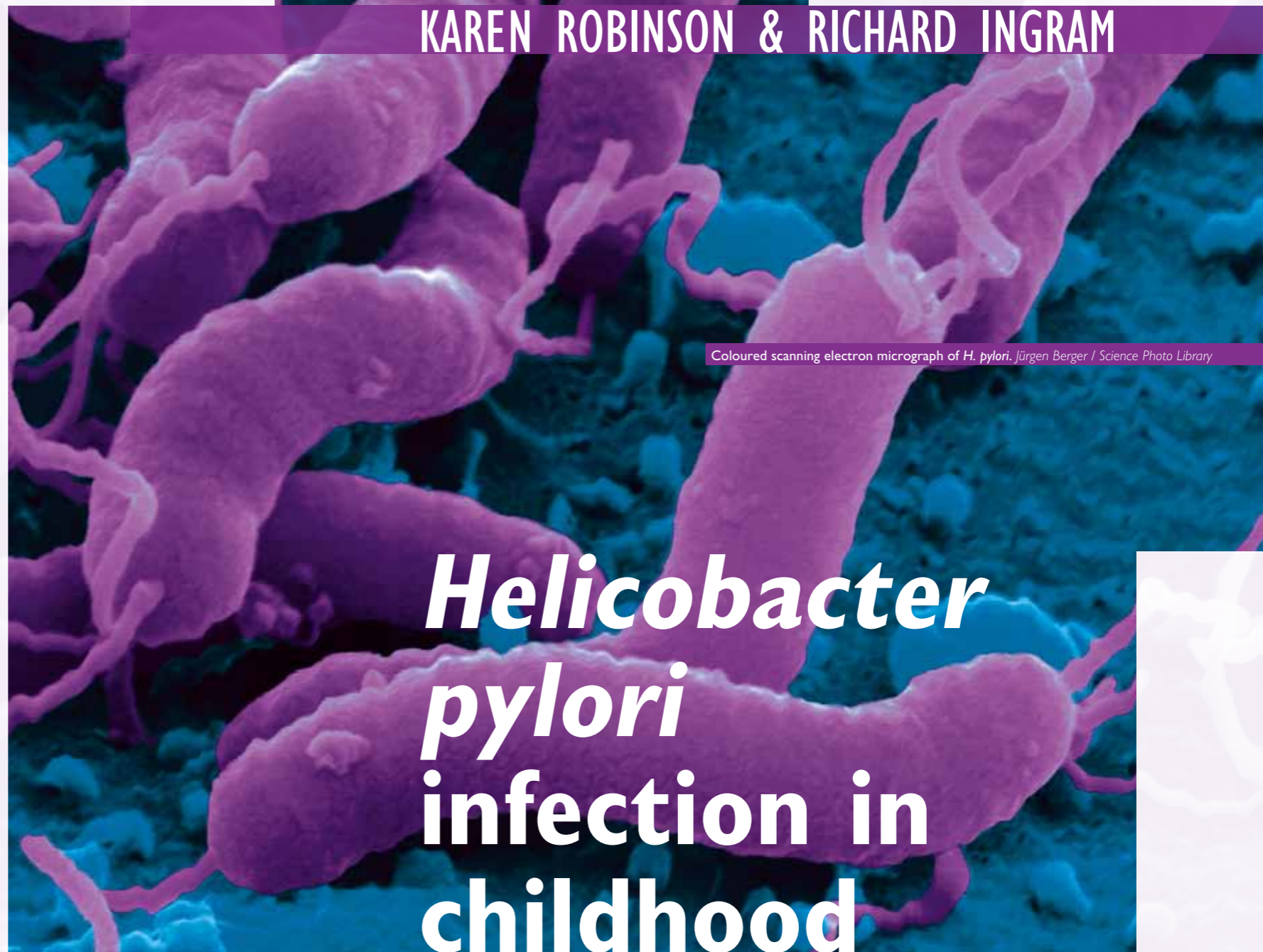


HELICOBACTER PYLORI colonizes the stomachs of nearly half the world's population, making it one of the most common chronic bacterial infections affecting mankind. Warren & Marshall first cultured the organism and identified it as the main cause of gastric inflammation and peptic ulcers only 25 years ago, for which they were awarded the 2005 Nobel Prize in Medicine. Their discovery, along with the development of acid-suppressing drugs, completely revolutionized the management of ulcers. *H. pylori* has had a very interesting history, but our understanding of many of its basic features is still incomplete.

The route of *H. pylori* transmission is not fully understood, but it is known to require close contact and probably occurs by direct exposure to *H. pylori* present in someone's mouth or perhaps by ingesting aerosolized vomit – yummy! There is a higher likelihood of infection in large families, and genetic studies have shown that strains tend to be passed from mother to child or from older siblings. The bacteria deploy an amazing armoury to prevent them being cleared by the immune system, including the expression of molecules that mimic host antigens, and even its cell envelope lipopolysaccharide has been modified to be much less stimulatory than other bacteria. Once colonized, the infection usually remains in place for life unless the child is treated with antibiotics. In the UK, usually a triple

Although our understanding of *Helicobacter pylori* is not complete, we do know that infection with this bacterium can cause problems in both children and adults. But is there a positive side to colonization by this bacterium?

KAREN ROBINSON & RICHARD INGRAM



Coloured scanning electron micrograph of *H. pylori*. Jürgen Berger / Science Photo Library

Helicobacter pylori infection in childhood

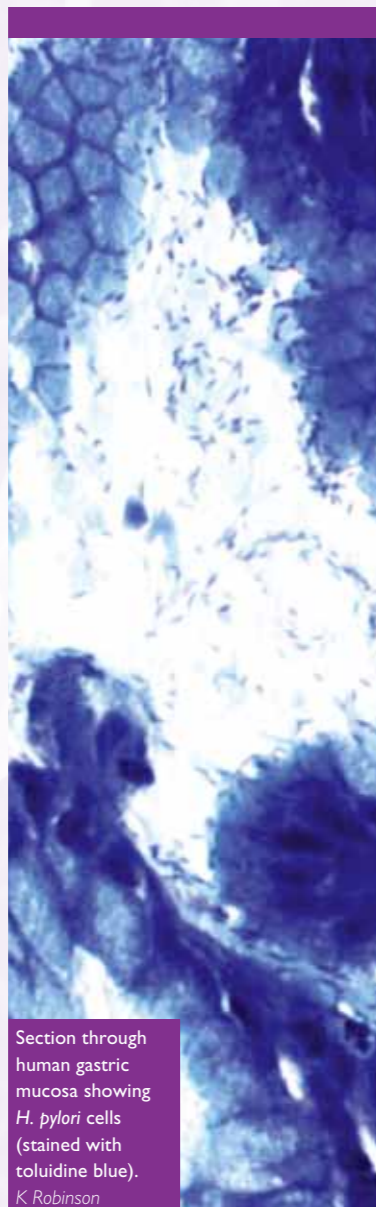
therapy of clarithromycin, amoxicillin or metronidazole, and an anti-acid drug is taken for 1–2 weeks.

This elegant spiral-shaped organism is uniquely adapted to survive in the harshly acidic environment of the stomach. It burrows into the thick mucus layer of the stomach lining for protection where it interacts with epithelial cells, causing inflammation and damage. Infection is normally established in childhood and persists for life. Adults cannot normally become infected as their gastric environment

is more hostile to colonization. *H. pylori*-associated peptic ulcer disease and stomach cancer are more commonly encountered in later life, but the infection is an important factor in a variety of conditions in children. Interestingly and controversially, studies are beginning to show that, as well as causing disease, infection with *H. pylori* might also be of benefit to its host, especially in childhood.

DISEASE OR NO DISEASE – THAT IS THE QUESTION

Gastric diseases are relatively rare in children – less than 5% of infected children get peptic ulcer disease compared with 15–20% of adults. There have been no reported cases of adenocarcinoma-type gastric cancer in childhood.



Section through human gastric mucosa showing *H. pylori* cells (stained with toluidine blue).
K Robinson

unexplained IDA should be tested and treated for *H. pylori*.

A study of 7- to 11-year-old Scottish girls in 1994 was the first to report adverse effects of the infection on growth; *H. pylori*-positive children were shorter. This and associations with lower birth weight, delayed puberty and reduced cognitive function (lower IQ) have been shown in infected children by groups around the world. Some have also shown alterations in the concentrations of growth-related hormones. Poor growth in infants might be due to reduced maternal milk production or impaired nutrition. *H. pylori* is known to disrupt the absorption of nutrients and eradication has been shown to increase nutritional parameters such as serum protein levels. However, such associations and effects have not been observed in all studies and still need to be clearly proved.

Another area, which in contrast has strong evidence and international consensus, is the role of *H. pylori* in a blood clotting disorder called idiopathic thrombocytopenic purpura (ITP). This condition results in lowered platelet numbers, preventing blood clots from forming properly, and leading to extensive bruising and petechiae on the skin and mucous membranes. Studies have reliably shown that patients with ITP are more likely to be infected, and eradication therapy results in significant and long-lasting



Illustration of immune thrombocytopenic purpura showing coating of platelets (orange) with IgG auto-antibodies (yellow), which renders them susceptible to phagocytosis by a macrophage (purple). DNA Illustrations / Science Photo Library

increases in platelet counts in over half of cases. Molecular mimicry has been proposed as a mechanism for induction of ITP, where *H. pylori* induces antibodies that cross-react with platelet membrane glycoprotein epitopes.

DISEASE OR BENEFIT – THAT MAY BE THE REAL QUESTION

We have presented evidence that *H. pylori* is a pathogen and causes harm, but around 80% of infected people do not suffer any ill effects. Humans have co-evolved with *H. pylori* for at least 50,000 years and colonization has arguably been the ‘natural’ state. There is a very high prevalence of *H. pylori* infection in certain parts of the world. In developing countries such as India and Vietnam, about 80% of the population are infected by the time they reach 20 years of age, compared to less than 30% in the UK and northern Europe. The frequency of infection has been declining globally over the last three to four decades, whilst there is a dramatic increase in the incidence of immune, inflammatory and metabolic diseases. An increasing body of evidence, albeit controversial, suggests that the absence of *H. pylori* is an important contributor to the development of these conditions. Several epidemiological studies, including our own, have shown that children with *H. pylori* are less likely to have allergies. It is difficult to say with confidence that *H. pylori* is protective as it could simply be a marker for

other exposures in early life. However, in one study people infected with more virulent CagA-positive strains were found to be 37% less likely to have had childhood asthma. Recently, some direct proof was shown in a study from Anne Müller’s group in Switzerland. They showed that in an animal model of allergic asthma, *H. pylori*-infected mice had less severe lung damage. They also found stronger protective effects when the mice were infected at an early age, mimicking the establishment of *H. pylori* in young children. As early childhood is the time when our immune systems are developing, perhaps *H. pylori* provides some of the signals needed for a healthy immune system.

IN CONCLUSION

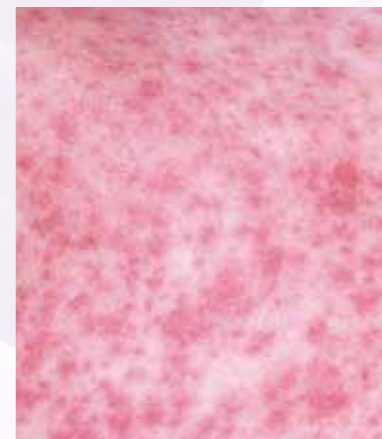
We have outlined our increasing understanding of *H. pylori* in childhood – it has infected most and still infects many, it harms some early on and may seriously harm others in later life, but could colonization also be beneficial?

Gastric cancer usually develops in old age and only in 1–8% of infected adults depending on where in the world they live and virulence of the infecting *H. pylori* strain.

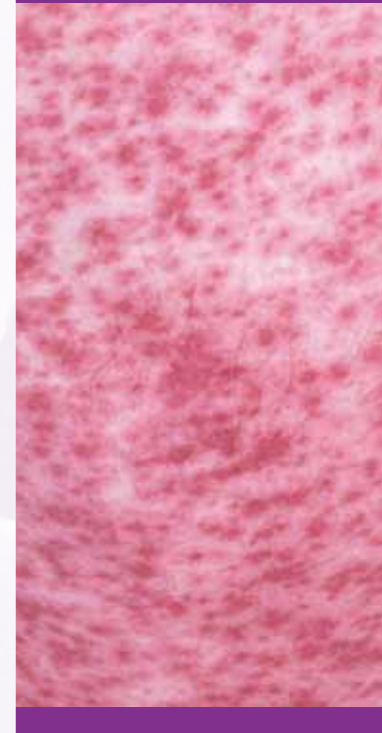
Incredibly, this stomach-dwelling bacterium has also been linked with a variety of extra-gastric conditions in children.

Iron deficiency anaemia (IDA) is a massive problem, particularly affecting children. There is increasing evidence that *H. pylori* infection is an important cause of this condition. Iron is more easily absorbed in soluble form – both when reduced from ferric to ferrous iron by gastric acid or converted into complexes by vitamin C. *H. pylori* infection is known to reduce levels of both gastric and ascorbic acid in the inflamed stomach. The bacterium may also take up iron from gastric tissue, thereby competing for nutrients with its host. Eradication therapy, both with and without iron supplementation, can improve blood iron levels and red cell counts. International consensus guidelines recommend that children and adults with

“Eradication treatment is warranted to avoid developing ulcers or cancer; however, perhaps this interesting organism still has a lot more to teach us about health benefits that we can derive from our colonizing microflora.”



Purpuric rash on a patient's back. Dr MA. Ansary / Science Photo Library



Eradication treatment is warranted to avoid developing ulcers or cancer; however, perhaps this interesting organism still has a lot more to teach us about health benefits that we can derive from our colonizing microflora.

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Myths and legends of *Cronobacter* – a new bacterial pathogen of babies?

IN INFANTS, *Cronobacter* has been reported to cause various clinical conditions, such as necrotizing enterocolitis, bacteraemia and even meningitis. The pathogenesis of *Cronobacter* meningitis is different to that of both *Neisseria meningitidis* and neonatal meningitic *Escherichia coli*, and is similar to that of the closely related bacterium *Citrobacter koseri*. *Cronobacter* has been isolated from the hospital environment and clinical samples: cerebrospinal fluid, blood, bone marrow, sputum, urine, inflamed appendix, conjunctivae and neonatal enteral feeding tubes. Infants can be colonized by more than one strain of *Cronobacter* and therefore multiple isolates need to be characterized in epidemiological investigations. *Cronobacter* infections can be fatal and, if the infant survives meningitis, then they are likely to be severely neurologically damaged for life. Hence, the need to search for a means to reduce the risk of infection is self-evident.

I actually know the time and place when I first heard of this bacterium. It was at Leatherhead Food RA on Tuesday, 2 November 1999 at a workshop on microbiological criteria. Terry Roberts was talking about the International Commission Microbiological Safety of Foods revised risk categories of food-borne pathogens. He referred to *Cronobacter* as being of concern for immunologically deficient individuals and especially neonates. I thought, 'Oh my goodness, there is going to be another *Listeria hysteria!*' The risk of *Listeria* in cheese and pâté to pregnant women had been big news at that time and I thought there would be a similar reaction to *Cronobacter*. However, it took another 4 years, when in 2003 the organism hit the headlines following a neonatal death in Tennessee (which actually occurred in 2001). This had been investigated by the FDA, with their report published in the journal *Emerging Infectious Disease*. Using pulse-field gel electrophoresis (PFGE) to genotype iso-

lates, the same strain detected in the cerebrospinal fluid was also found in a previously unopened tin of powdered formula. Several other neonates in the intensive care unit were also colonized by the strain. Hence, instead of a general 'nosocomial'-attributed cause of infection, a source could be named and therefore a control measure could be applied. Consequently, the Codex Alimentarius Commission (CAC) started a review of the microbiological guidelines for powdered infant formula, and in 2004 the FAO/WHO undertook the first of three risk assessment evaluations of *Cronobacter*. At that time, the microbiological criteria for powdered infant formula had a strict two-class plan for *Salmonella*, and a three-class plan for coliforms. Hence, *Cronobacter* would be within the coliform count. These criteria had been set in 1979, and the Tennessee outbreak prompted their re-evaluation. In fact, *Cronobacter* was not the only organism reviewed with respect to possible infection through reconstituted infant formula. Since *Salmonella* infections of powdered infant formula had also been reported, the 2006 FAO/WHO meeting reviewed the current relevant knowledge. It was plausible that lactose-fermenting *Salmonella* colonies on commonly used isolation media could be overlooked. Despite the FAO/WHO risk assessment meetings starting in 2004, the CAC microbiological criteria did not change until 2008. These now apply to powdered infant formula for a target age of up to 6 months. The criteria were not applied for formula commonly known as 'follow-on formula' which is used at weaning as there was insufficient epidemiological evidence.

Despite the recent recognition of the organism, there are a number of myths that have already grown up around it which need to be dispelled before moving on to recent developments.

MYTH 1: *E. SAKAZAKII* IS NOW CALLED *C. SAKAZAKII*

Initially, the isolates in the reports above were identified as *Enterobacter sakazakii*. But with further studies it became apparent that they differed sufficiently from *Enterobacter* to be considered as a separate and novel genus to which the name *Cronobacter* was applied. This genus currently contains seven species: *C. sakazakii*, *C. malonaticus*, *C. turicensis*, *C. muytjensii*, *C. dublinensis*, '*C. universalis*' and '*C. condimenti*'. However, these names have not been adopted consistently in the literature. At times the '*E.*' genus prefix appears to have been simply replaced by '*C.*', and the method of speciation has not been given. Phenotyping using commercial biochemical profiling kits is insufficient to differentiate between the seven *Cronobacter* species; instead, DNA sequence-based methods are necessary. But even PCR-based methods developed just a few years ago may not be fully reliable as the number of recognized *Cronobacter* species has increased from five to seven since 2007.

MYTH 2: ALL *CRONOBACTER* STRAINS ARE 'YELLOW'

Unfortunately, this description appears in some internationally approved detection schemes (i.e. ISO). When the original species '*Enterobacter sakazakii*' was described it was applied to yellow-pigmented strains of *Enterobacter cloacae*. However, as new strains have been collected from diverse sources, this trait is no longer recognized as a reliable or useful criterion. The yellow pigmentation is not unique to *Cronobacter* as it is also found in related organisms such as *Pantoea* and, possibly more importantly, about 10% of strains are non-pigmented. Also, the pigmentation is temperature-dependent with some strains being pigmented when grown on soya-based media at room temperature, but not when grown at 37 °C.

MYTH 3: *CRONOBACTER* TARGETS INFANTS

Why was the genus named '*Cronobacter*'? It is derived from *Kronos*, the Greek god famed for eating his off-

Many microbiologists in recent years have heard of the 'novel' bacterium *Cronobacter*, probably because of its association with severe, often fatal, infections of babies. However, accurate information on this organism is not so easily acquired as its emergence has been convoluted and prone to misunderstanding.

Coloured scanning electron micrograph of *Cronobacter sakazakii*. Scimat / Science Photo Library



Bottle feeding. Lifesize / Thinkstock

STEPHEN J. FORSYTHE



Baby with a feeding tube in a neonatal intensive care unit. iStockphoto / Thinkstock

MYTH 5: CRONOBACTER INFECTIONS ARE DUE TO POWDERED INFANT FORMULA

This myth is clearly not true given that the majority of infections are in adults. Powdered infant formula has been identified as the source in some neonate intensive care unit (NICU) outbreaks and is thus a target for control. Nevertheless, the level of intrinsic contamination has never been reported to exceed 1 c.f.u. g⁻¹. So temperature abuse could be a significant factor in enabling bacterial growth and increased risk of infection. Additionally, the formula can be extrinsically contaminated due to unhygienic

spring. This may seem apt, but in fact *Cronobacter* infects more adults than infants. The data can be found in the 2008 FAO/WHO risk assessment concerning follow-on formula, and was provided by Paul Cook from the HPA surveillance scheme in England and Wales.

MYTH 4: CRONOBACTER IS NOT FOUND IN THE ENVIRONMENT

This myth is due to an early paper that reported looking for *Cronobacter* in mud, soil, etc., without any luck. However, at that time the isolation medium used was for *Enterobacteriaceae* in general and was not specific for *Cronobacter*. These days a number of chromogenic agars have been marketed which enable the isolation of *Cronobacter* from a mixed *Enterobacteriaceae* flora. In fact the organism is ubiquitous. A highly plausible ecosystem for *Cronobacter* is plants. This would account for the yellow pigmentation (to reduce oxygen-radical damage on exposure to sunlight) and capsule production (which could help adherence to the plant surface). It may also account for its desiccation tolerance, possibly linked to resistance to essential oils via efflux systems, and facilitating the organism's persistence during starch production. Starch is used in both casein- and soy-based powdered formula, as well as other foods, and this explains the high incidence of *Cronobacter* in seasonings (30%) and powdered foods (25%), as well as cereals such as wheat, corn, soy, rice, vegetables and salads. The organism has been isolated from a range of other foods, including cheese, meats and milk powder.

practices. *Cronobacter* is found in faeces and therefore transfer via hand contamination could occur. Breast milk is not sterile and the *C. malonaticus* type strain was isolated from a breast abscess. It may be pertinent to point out that whereas powdered infant formula production is subject to microbiological criteria, mastic breast milk is still used to feed newborn babies who lack a developed immune system and competing gut flora.

The population most at risk of *Cronobacter* infection are low-birth-weight neonates. These do not have a developed suckling response and so are fed via nasogastric tube. *C. sakazakii* has been isolated from biofilms in feeding tubes removed from neonates receiving breast milk and also ready-to-feed (not reconstituted) formula. The Tennessee outbreak, and others in France, have involved neonates fed using an automatic dispensing syringe device which over several hours slowly pumped the feed through the nasogastric tubes into the stomach. The syringes were at ambient temperature (which in NICUs is normally raised to ~25 °C) and were not chilled. Hence, there was the opportunity for bacterial growth before ingestion. In addition, the neonatal stomach is less acidic than an adult's, and so less bacterial kill will occur before the feed enters the intestines.

Ironically, despite changes in international practices, an outbreak such as the one in Tennessee could happen again. Why? Because the outbreak was due to the use of formula not intended for infants, which is not subject to the revised CAC criteria. Also, the FAO/WHO-recommended reconstitution of powdered infant formula with water at >70 °C is not followed in the USA. Dipping a thermometer into reconstituted formula would have its own inherent problems of contamination, and so the advice has been to use water which has been boiled and left to cool for 30 minutes. This may be feasible for feeding full-term infants, but is very difficult for premature babies requiring small volumes of formula. Finally, the term 'powdered infant formula'

“Ironically, despite changes in international practices, an outbreak such as the one in Tennessee could happen again.”

includes 'breast milk fortifiers'. These products are added to supplement the nutritional value of mother's milk and so are not reconstituted with water.

As stated above, most *Cronobacter* infections occur in adults, possibly primarily due *C. malonaticus*. The source of infection may be through ingestion, as the organism is ubiquitous in food; however, it is also plausible that the source is nasopharyngeal infection, like *Neisseria meningitidis*, which would explain cases of pneumonia and isolation from sputum.

MYTH 6: ALL 'E. SAKAZAKII' INFECTIONS WERE DUE TO CRONOBACTER

It is reasonable to expect that the taxonomic revisions would encompass the previously identified '*Enterobacter sakazakii*' isolates. However, the identification of strains in early outbreaks did not use methods with the same reliability as those used currently. We have studied strains from early cases and have re-identified them as *E. cloacae* and, more often, *E. hormaechei*. This includes a strain published as a 'quinolone-resistant *E. sakazakii*', which is actually *E. hormaechei*.

MYTH 7: CRONOBACTER IS THE MOST THERMOTOLERANT ENTEROBACTERIACEAE

This is one of the first myths that appeared and it is possibly due to the surprising idea that a non-spore-forming bacterium could be found in powdered infant formula, which would have been exposed to various stresses such as heat and drying. It comes from one publication in which the decimal reduction time (the time required to kill 90% of the organisms being studied) of 1.3 min at 72 °C had been calculated. However, even though later experiments published by the same authors revised the thermal resistance to a more accurate value, the initial story persisted through secondary references. In support of the myth, a protein was identified in *Cronobacter* with a high decimal reduction time of ~400 s at 58 °C which had a nearest match to the Archaeal *Methylobacillus flagellatus* KT protein and hence it was proposed that the gene encoding this protein in *Cronobacter* was a thermoresistance gene. These days, it is well known that this protein is widespread amongst bacteria, and its presence does not correlate with thermoresistance. *Cronobacter* can be killed by normal pasteurization processes and is more thermosensitive than *Listeria monocytogenes*.

SO WHERE ARE WE NOW?

We know with respect to infants that ingestion is a route of infection, and one means of reducing the risk

is hygienic preparation of all feeds. We are at the early stages of understanding the mechanisms of how *Cronobacter* invades the intestinal epithelial layer, persists in macrophages and penetrates the blood-brain barrier.

The sequenced genomes of *Cronobacter* species have revealed an array of adhesins, outer-membrane proteins, efflux systems, iron-uptake mechanisms, haemolysins and type VI secretion systems. Yet there is considerable diversity within the genus. Following a multiple-strain *Cronobacter* outbreak at a French NICU it became apparent that possibly not all *Cronobacter* strains are equally virulent. Although PFGE and PCR are useful for small-scale studies, they are limiting when studying bacterial populations, and DNA sequence-based databases are more applicable. Fortunately, a seven-allele multilocus sequence typing (MLST) scheme has been established and is hosted by the University of Oxford through Professor Keith Jolley. The protocols and database are publically accessible online at www.pubMLST.org/cronobacter. Additionally, the same approach has been used for multilocus sequence analysis (MLSA) for phylogenetic construction of the *Cronobacter* genus. The concatenated sequences of the seven loci give a total sequence length of 3,036 bp. MLSA supported the recent naming of two novel species: '*C. universalis*' and '*C. condimentii*'. Further interrogation of the MLST database for patterns of sequence type and source have revealed that the majority of meningitic cases in the past 30 years across six countries were due to one sequence type (ST4) out of the currently recognized 85 sequence types. This remarkable discovery indicates that the organism is clonal and gives a clear direction for further *Cronobacter* meningitis research. Applying MLST to non-clinical strains has shown that ST4 was isolated from milk powder in 1951, hence giving us a 60-year timescale of stability. To date, there does not appear to be such a clear link between sequence type and other *Cronobacter* infections, such as necrotizing enterocolitis. Nevertheless, by a combination of regulatory improvements and laboratory research we have come a long way in a short time in our understanding and control of neonatal *Cronobacter* infections.

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FURTHER READING

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FEW PEOPLE IN BRITAIN under 40 have had measles, and even those who have are unlikely to have experienced its secondary bacterial effects, which in the developing world still often kill young children. Yet before the antibiotic era measles was as serious in the developed world as it remains in those countries where obstacles to the use of vaccine persist and secondary respiratory and other infections are likely to go untreated. In Britain a century ago, measles was often followed by pneumonia and middle-ear disease. While diphtheria was then treatable by antiserum, scarlet fever was steadily losing its virulence and smallpox was no longer epidemic, measles remained an inescapable and potentially dangerous fever of young children. Without vaccination measles was, and has remained, a serious disease of childhood.

Smallpox was globally eradicated over 30 years ago; but what about measles?

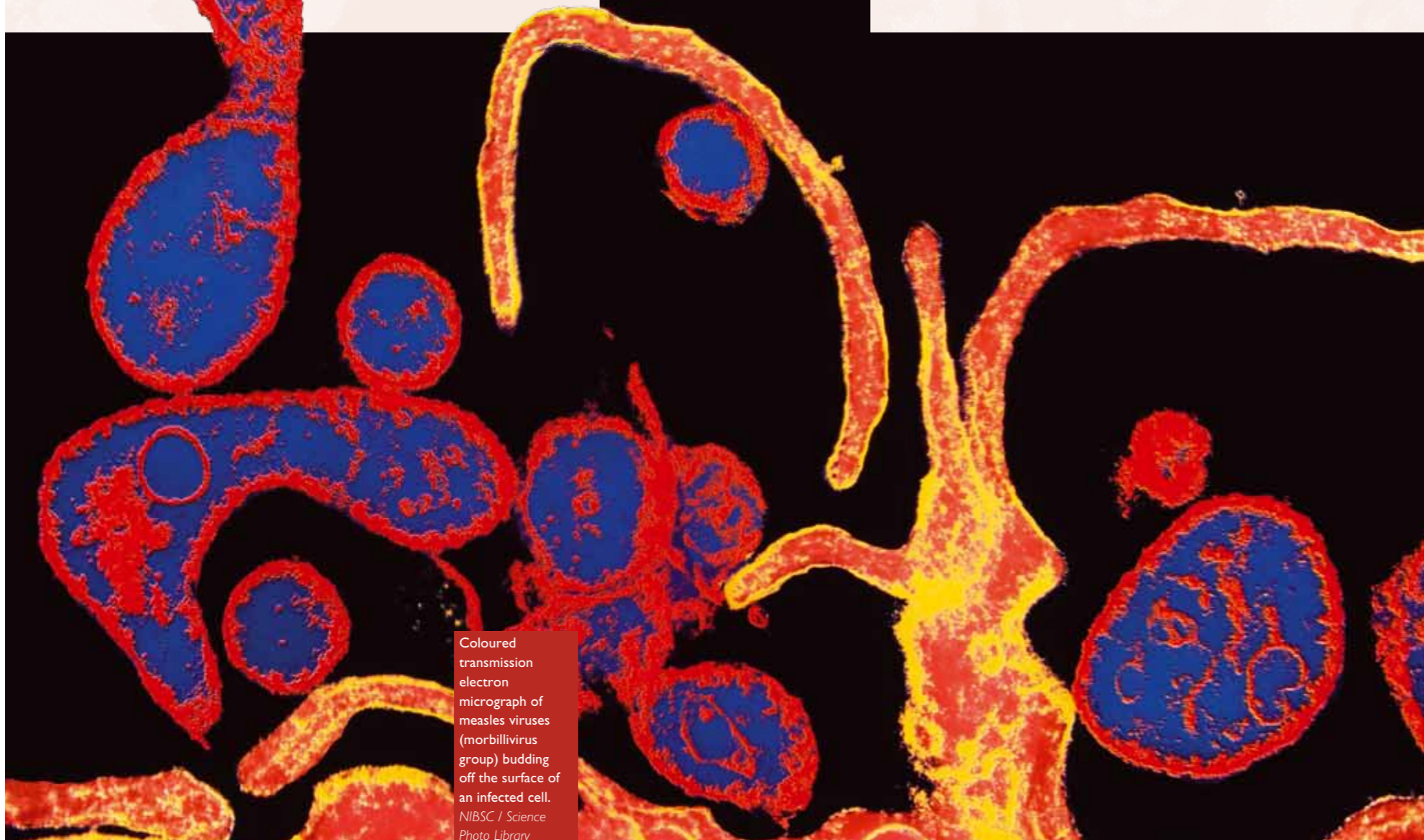
VACCINE THE KEY

To combat measles, convalescent serum or immunoglobulin were used initially, but the protection they gave was short-lived. Then, in 1954, the measles virus was isolated in cell culture and, in 1965, an inactivated vaccine was released for use. This vaccine unfortunately caused an ill-understood reaction in some recipients when they were subsequently exposed to measles ('atypical measles syndrome'), and the vaccine was withdrawn. Soon after, however, attenuated vaccine strains became available; these were immunogenic and had only occasional side effects, all of them consistent with mild natural measles infection. A further refinement was to add to the attenuated measles vaccine similar vaccines against mumps and rubella (MMR vaccine). That mixture, developed by Hilleman

and colleagues at Merck laboratories, could be given without any loss of efficacy. The measles component of the vaccine offered the prospect of countries, continents, and even the entire world being rid of measles just as, since 1977, the world has been rid of smallpox. The two diseases are analogous in several respects. Like smallpox, measles is in almost all cases clinically identifiable and has a proven effective vaccine, vaccine reactions are uncommon, especially in the re-vaccinated, there is no permanent animal reservoir, and vaccination

Can measles

be eradicated?



Coloured transmission electron micrograph of measles viruses (morbillivirus group) budding off the surface of an infected cell. NIBSC / Science Photo Library

PHILIP MORTIMER



Florid measles in a young child: note the feverish and miserable appearance and the intense rash. Lowell Georgia / Science Photo Library



Administering the MMR vaccine. Dr P. Marazzi / Science Photo Library

is not needed once the disease has been eradicated. It was the successful global eradication of smallpox that first suggested that measles' grip on young children might soon be loosened worldwide. The ensuing years have shown, however, that global eradication of measles will be a harder task.

WHAT ARE THE BIOLOGICAL CONSTRAINTS ON MEASLES ERADICATION?

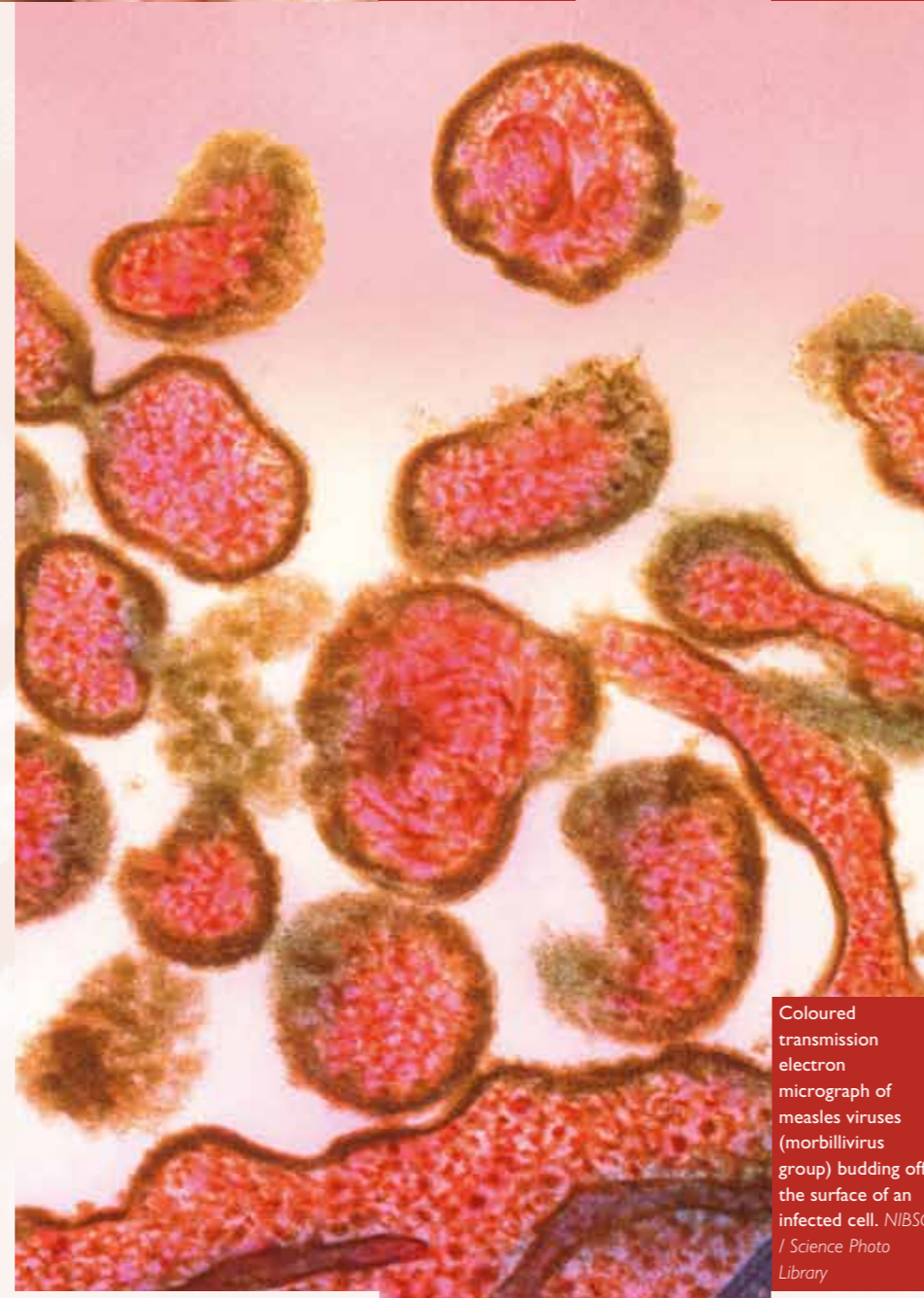
On the face of it, measles and smallpox have much in common. Both spread through the air and by close contact, and both enter the body by the respiratory route. At a predictable interval, susceptible contacts develop fever and then a rash. Each case sheds abundant virus, especially early on, generating a new crop of cases from 10 to 14 days later. Almost all infections are clinically expressed unless there has been a previous vaccination. Attenuated vaccine, given pre- or immediately post-exposure, can prevent both diseases. No animals have been found that might act as a reservoir of either virus following its eradication in humans.

However, the two viruses are genetically entirely different – smallpox is a DNA virus and measles is an RNA virus – and they do not behave in exactly the same way. Measles remains very widespread and it is very infectious, so isolation is less effective. It targets young children, so a susceptible cohort accumulates quickly. The incubation period

for measles to the point where virus begins to be shed is a few days shorter than for smallpox, leaving less time in which to intervene to protect contacts. Measles is also more infectious than smallpox: its reproductive rate (R value) typically exceeds 10 in an unimmunized community. Once maternally acquired immunity to measles has waned (a matter of months and probably less if the mother owes her immunity to vaccination), exposed children almost invariably contract measles, and under-5s, exposed within the family or at a nursery are particularly vulnerable to its complications. Measles vaccine, though superior from a regulatory point of view, is more expensive, more labile and more difficult to administer than the smallpox vaccine. It is preferable to give two separate doses of measles vaccine, a further logistic challenge. In addition to this, measles vaccine has during its relatively short existence attracted as much dissent as smallpox vaccine ever did; so its benefits have to be constantly re-asserted.

STILL AN OPPORTUNITY TO ERADICATE MEASLES

Those are some of the obstacles to measles eradication. Yet in 2010, veterinarians were able to announce that rinderpest, a cattle disease virologically and clinically very close indeed to measles, had been globally eradicated. That is an indication that measles too could be eradicated worldwide, though it will not be as



Coloured transmission electron micrograph of measles viruses (morbillivirus group) budding off the surface of an infected cell. NIBSC / Science Photo Library

“Because of its high infectivity, the global eradication of measles will be harder than was the case for smallpox, and it will demand circumstances that do not yet prevail internationally.”

readily dealt with as rinderpest – people can't be herded as cattle can.

So the idea that measles can, like smallpox and rinderpest, be eradicated should not be dismissed. The hurdles are mostly political ones. To overcome smallpox worldwide during the Cold War era of the 1960s and 1970s, the United States and the Soviet Union embarked through the World Health Organization on a huge collaborative effort. Diplomatic pressures were exerted, multi-national personnel were recruited, smallpox vaccine was procured on a scale sufficient and sustained enough to overcome logistical difficulties, freeze drying was used to stabilize the vaccine, and a novel needle was invented that delivered the vaccine safely. The shrinking pockets of smallpox in South Asia and East Africa were gradually eliminated, and after a decade of intensive effort and further years to ensure that no other human cases or animal reservoirs existed, smallpox was declared eradicated and vaccination was redundant. In theory the same could now be achieved for measles.

Ultimately, the challenge is to interrupt virus transmission from every case of measles, and this requires almost universal acceptance of vaccine by each new cohort of susceptible children. So far, wariness about the safety of measles vaccine and the motives of vaccinators has not been overcome, and the reservations of some parents have often been compounded by administrative shortcomings that have denied children the opportunity

of immunization. Other countries have simply lacked the resources to support a vaccination programme.

Such failures of vaccine delivery have long-term effects. For example, the consequences of a single, now discredited, 1998 *Lancet* paper by British authors about the side effects of MMR are still being reflected in inadequate UK acceptance rates, as well as cases of measles in never-vaccinated young adults. Across Europe there is currently a surge of measles; 30% of these cases are in young adults who for various reasons were denied measles vaccination in the 1990s.

Global measles eradication is predicated on good governance that will deliver universal immunization. Yet it is hard to imagine that what is now being achieved in terms of disease elimination in developed and some developing countries can be established in every single weak or failed state, or even that every functional state can rapidly eliminate measles. The influx of refugees and asylum seekers to these countries includes recently measles-exposed and measles-susceptible children, and they may disseminate the disease in their destination country before they can be vaccinated. Meanwhile, in developing countries allocation of available funds is often uneven so that while measles eradication remains the long-term goal, initial but premature efforts may turn out to be unsustainable. It is probably preferable to concentrate international resources in politically stable countries, immunizing the youngest children who are most likely to be susceptible.

CONCLUSION

There is a safe attenuated measles vaccine, the primary tool of an eradication programme. Measles can therefore be eliminated in any country where a political will, sustained vaccine delivery and continuing disease surveillance are in place. Because of its high infectivity, the global eradication of measles will be harder than was the case for smallpox, and it will demand circumstances that do not yet prevail internationally; but excessive cynicism on this score can easily undermine the vaccine campaigns already being pursued by national governments and international philanthropic organizations. A constructive realism and a goal-orientated policy will ensure that resources continue to be best used and a point reached when global eradication is perceived as a realistically attainable goal. Once achieved, the benefits from this would exceed even those that the eradication of smallpox have brought over the last 30 years.

PHILIP MORTIMER is a retired Health Protection Agency (HPA) virologist. (The views expressed in this article are not necessarily those of HPA.)

FURTHER READING

- Allen, A. (2007). *Vaccine: the Controversial Story of Medicine's Greatest Lifesaver*. New York, London: Norton.
- Cotrell, S. & Roberts, R.J. (2011). Measles outbreak in Europe. *Br Med J* 342, 1374–1375.
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IN MARCH 2011, video clips of practical microbiology techniques were introduced to the SGM's video portal. Ranging from preparation of working surfaces to effective methods of culture transfer, the videos could be used by educators to demonstrate basic techniques to their students.

After a second jaunt to the 'studio' we have added a number of new investigations to the portal, including using and testing antimicrobial agents, preparing a Gram stain, microscopy and serial dilutions.

These videos feature Dr John Schollar from the National Centre for Biotechnology Education

NEW PRACTICAL MICROBIOLOGY TECHNIQUE VIDEOS



who runs the SGM's practical microbiology courses. This is a dynamic resource and will be added to regularly over the coming years; keep an eye on the portal and in *Microbiology Today* for updated content.

The portal can be accessed through www.microbiologyonline.org.uk/what-s-hot/podcasts-and-vodcasts



ALGAE: A PRACTICAL RESOURCE FOR SECONDARY SCHOOLS

THIS RESOURCE has been developed to provide a series of well-tested practical activities for use in secondary schools. Algae are safe to handle, easy to source and maintain, and cheap to use. They have great potential to illustrate numerous scientific concepts, such as photosynthesis, phototaxis, bioluminescence, eutrophication and gas cycling.

Each pack contains all the information needed to carry out five practical activities, including teacher, technician and student guides, as well as video clips and images, a PowerPoint presentation and a poster. The resource is free to SGM School Members and Ordinary Members involved in outreach work. The price for non-members is £15. To order your copy, please contact y.taylor@sgm.ac.uk

To support the algae pack, the Culture Collection of Algae and Protozoa (CCAP) have designed a kit comprising a leaflet with culturing advice and relevant samples. The cost will be £25 plus VAT plus p&p. Go to www.ccap.ac.uk to order or for more information.



MICROBIOLOGY EDUCATION TODAY AND TOMORROW



From 4 to 7 January 2012 the University of Liverpool hosted this year's Association for Science Education (ASE) conference. The SGM had a stand in the exhibition marquee, offered a practical microbiology session, contributed to a series of talks sponsored by the NUCLEUS group and launched a new practical resource pack.

Above Yvonne Taylor, SGM Communications Administrator, manning the SGM stand. Right John Schollar (right) demonstrating to an attendee. V. Symington



FIGHTING FIT AND ALGAL ANTICS: SGM AT THE ASE IN LIVERPOOL

SGM'S STAND at ASE this year was hugely popular; we were showing off SGM's educational resources, including the new algae pack and practical microbiology videos (see p. 38). We talked to teachers from all over the world from Denmark to New Zealand and from Brazil to Merseyside! All were very keen to learn what SGM had to offer the mini-microbiologists in their classrooms.

John Schollar (National Centre for Biotechnology Education) ran a practical microbiology workshop on behalf of SGM as part of a collaborative project. Attendees learned how to build their own microbial fuel cells, prepare a Gram stain, enumerate their cultures and paint snowmen using bacteria!

NUCLEUS is a group of learned societies, and similar not-for-profit organizations, who are involved with promoting bioscience education. Amongst other things, members of NUCLEUS get together to sponsor a 1-day symposium at the ASE conference entitled *Biology in the Real World* (BitRW). Eminent speakers from the different societies' disciplines give talks which aim to bring the biology curriculum to life while maintaining a link to the biology specifications. Each year the series follows a specified theme; this year it was

Right: Two examples of the press coverage following Mike Gleeson's talk. Below: Dariel Burdass (right) launching the new SGM algae resource with the author James Redfern (left). V. Symington



A *Sporting Chance*. The talk from SGM, in collaboration with the British Society for Immunology and MISAC, was given by Professor Mike Gleeson (Loughborough University) and was entitled *Fighting fit – how exercise affects your immunity and susceptibility to infection*.

Mike began by introducing the 'J-shaped' curve which describes the relationship between physical activity and the risk of upper respiratory tract infections (URTIs), i.e. moderate training conveys a below average risk to URTIs, while high-intensity exercise carries a higher risk. He went on to talk about how this applies to the general population and to elite athletes; he cited anecdotal examples, including one of Alberto Salazar who confessed to having 12 colds in 12 months during his preparation for the 1984 Olympic marathon! This was ultimately related to immune function and performance after physical activity, revealing a delicate 2-hour post-performance

Exercise keeps the colds at bay (as long as it's gentle)

REGULAR exercise reduces the risk of getting a cold – but only if it is not too vigorous, researchers say. Brisk walks every day have been shown to boost the immune system and reduce the chance of catching a cold by up to 30 per cent. But prolonged strenuous activity, such as running a marathon, could leave participants between two and six times more likely to succumb to upper respiratory infections including colds, flu, sinusitis and tonsillitis, it is claimed. Professor Mike Gleeson, an expert in biochemistry at Loughborough

By Tamara Cohen
Science Reporter

University, said the immune system contains natural killer cells which try to eliminate infected ones. Moderate exercise enhances their activity but stressful endurance can turn them down. Speaking at the Association for Science Education's annual conference in Liverpool, he concluded that 'moderate' exertion is best for the immune system. 'It's all about finding a happy medium,' he said.

Risk of flu in too much exercise

1000 people over the course of the study. On the other hand, vigorous exercise can help to reduce the risk of catching a cold. Moderate exercise strengthens the immune system and reduces the risk of catching a cold. However, too much exercise can lead to a higher risk of catching a cold. This is because the immune system becomes exhausted after intense exercise. The risk of catching a cold is highest in the first 72 hours after intense exercise. This is because the immune system is still recovering from the stress of the exercise. The risk of catching a cold is lowest in the 72 hours after moderate exercise. This is because the immune system is still recovering from the stress of the exercise.

the chance of catching a cold. Prof Gleeson said the immune system contains natural killer cells which try to eliminate infected ones. Moderate exercise enhances their activity but stressful endurance can turn them down. Speaking at the Association for Science Education's annual conference in Liverpool, he concluded that 'moderate' exertion is best for the immune system. 'It's all about finding a happy medium,' he said.

window where endurance athletes are susceptible to infection due to reduced lymphocyte function. To conclude, Mike warned that this information should be kept in perspective! High-intensity training may mean a few more colds, but it is good for reducing risk of chronic metabolic and cardiovascular diseases.

A summary of Mike's talk, together with the resource produced for the event, was circulated to the press and we were delighted by the response. The timely nature of the story meant that it was covered in the local and national press, and the story travelled around the world with coverage in Ireland, Australia and America; the *British Medical Journal* also got in touch and an article appeared there too. Andrew Lansley, Secretary of State for Health, remarked on the press coverage the talk received while visiting Mike at Loughborough University the following week!

We are very grateful to Mike for his contribution to our success at this year's conference. He has kindly helped us update an article (*Couch potato or elite athlete? Finding the happy medium*) which was originally published in *Microbiology Today* in August 2009 and is now being distributed to SGM School Corporate Members – order your copy at www.microbiologyonline.org.uk/teachers/resources. To see Mike's presentation slides and others in the series – go to www.societyofbiology.org/bitrw. To listen to an SGM podcast with Mike, go to www.sgm.ac.uk/NEWS/podcast.cfm

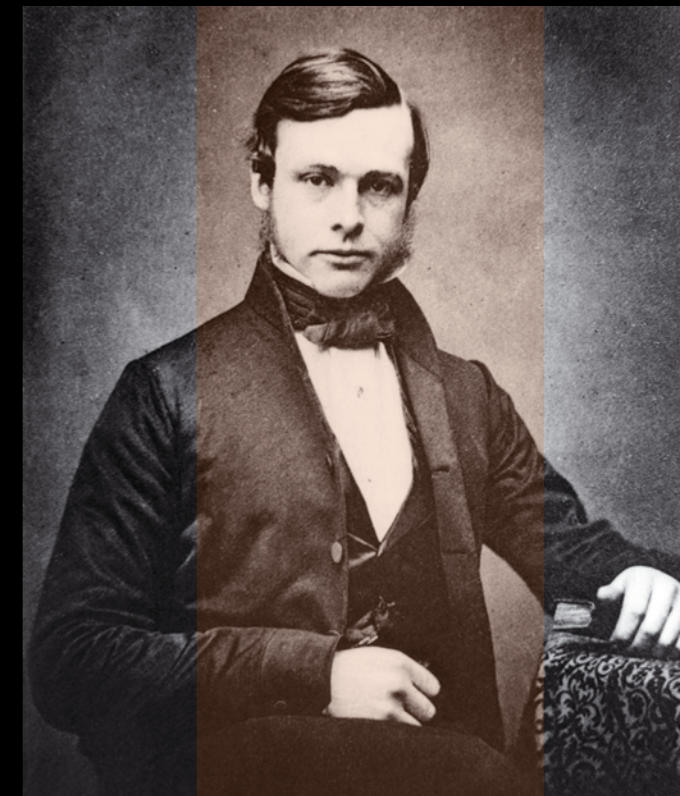
At 4.30 pm, after BitRW closed its doors for another year, the SGM were on hand to offer light relief and refreshments to science educators interested in taking algae into their classrooms. *Algae: a Practical Resource for Secondary Schools* (see p. 38) was given its official launch with an audience of more than 50 delegates, all of whom we armed with instructions to return to their classrooms to become acquainted with the finer details of bioluminescence, phototaxis and gas cycling, all of which can be demonstrated safely in the classroom as detailed in the pack!

VICKI SYMINGTON is Education and Outreach Officer at SGM

Learning from Lister: antiseptics, safer surgery and global health

Conference 22–24 March 2012

King's College London is hosting a major conference on Lister's life, methods and ideas, to examine the significance of his techniques in their historical context, and the enduring impact that Lister has had on 20th and 21st century medical and surgical practice.



Joseph Lister (1827–1912), SPL

MARKING THE 100TH ANNIVERSARY of Lister's death, this conference will be of interest to academic historians, clinical and healthcare scientists and practitioners, bioscience, health policy and management professionals, and those with an interest in Lister, Listerism and the development of antiseptic surgery.

The conference will be run in association with the Royal Society and the Hunterian Museum at the Royal College of Surgeons, and events will take place at both of these institutions and at the King's College London Strand Campus.

The conference is supported by The Lister Hospital, The Lister Institute of Preventive Medicine, the Society for the Social History of Medicine, the Royal College of Surgeons of England, the Wellcome Trust, the Royal Society, King's College London and King's College Hospital Charity and the Hunterian Museum.

Further details of the programme and information on how to register to attend are at www.kcl.ac.uk/cultural/listers2012/index.aspx

2012 will see the 10th winner of *The Sir Howard Dalton Young Microbiologist of the Year* competition. In this Gradline, Karen McGregor talks to finalists from the previous 10 years about their experience of the competition and the importance of developing good communication skills for a career in science. Previous winners give their tips for success to the 2012 finalists.



UPDATES AND ADVICE FOR EARLY CAREER MICROBIOLOGISTS

Sir Howard Dalton

THE AWARD promotes and rewards excellence in science communication and gives early-career members an opportunity to develop their presentation skills. First run in 2003 as the *SGM Young Microbiologist of the Year Competition*, this annual competition sees up to eight eligible early-career microbiologists give judged short oral presentations on their research to compete for cash prizes.

Sir Howard Dalton, a distinguished and influential microbiologist and SGM president from 1997 to 2000, achieved early international recognition for his pioneering work on the biology of methane oxidation and went on to become a recognized expert in the fields of global warming, biofuels and animal diseases. He was a highly respected science communicator – communicating with politicians (in his role as Defra’s Chief Scientific Adviser to the UK Government), fellow researchers (in more than 250 published scientific papers), students (as a professor at the University of Warwick where he was described as a quirky and engaging lecturer)



“The competition is based around clearly presenting your most exciting data to a general audience. This is a key part of every scientist’s working life and it’s not easy to do well. Entering this competition is a great way to practice.”

Edward Hutchison
1st prize 2007

and lay people (in blogging his experiences of a visit to the British Antarctic Survey in 2006) – making it highly appropriate that the competition was renamed in his honour following his death in 2008. Two previous winners were PhD students at the University of Warwick at the time of their win (Josh Neufeld, 1st prize 2006; Rich Boden, 1st prize 2008 – and both were supervised by Colin Murrell, himself a former PhD student of Professor Dalton). The 10th winner will be aptly awarded at the SGM Autumn Conference taking place at the University of Warwick in September 2012.

See p. 7 for the names of the 2011 finalists and prize winners.

“This prize gave me something to write in the ‘Recognition and Esteem’ section of my CV and on job application forms.”

Kirsty Ross
3rd prize 2007

“Good communication skills are essential for your success as a scientist – they will allow you to convince people to collaborate with you, to hire you and to give you funding.”

Nolwenn Jouvenet
1st prize 2004

HOW THE COMPETITION WORKS

Entry is open to SGM members who are postgraduate students or postdoctoral researchers (having gained a PhD in the last 2 years). Full details of eligibility criteria are available at www.sgm.ac.uk/meetings/SgmPrize.cfm

Step 1. Eligible members tick the appropriate box on the abstract submission form for the SGM Spring Conference to nominate themselves for consideration.

Step 2. Judges view the posters or offered papers of nominees at the SGM Spring Conference. Selected finalists are notified in the summer.

Step 3. Finalists give a 10-minute oral presentation (with 5 minutes for questions) in a special session at the SGM Autumn Conference.

Step 4. Cash prizes are awarded to the three best entries at the conference dinner: 1st Prize £500, 2nd Prize £200, 3rd Prize £100.

Young Microbiologist of the Year Competition

WHERE ARE THEY NOW?

1st winner	2003	Stephen Griffin	Senior Research Fellow and Group Leader at the Leeds Institute of Molecular Medicine
2nd winner	2004	Nolwenn Jouvenet	Chargée de Recherche at the Pasteur Institute, France
3rd winner	2005	James Edwards	Teaching Fellow at the University of York
4th winner	2006	Josh Neufeld	Associate Professor at University of Waterloo, Canada
5th winner	2007	Edward Hutchison	Junior Research Fellow at Worcester College and Postdoctoral Researcher at the University of Oxford
6th winner	2008	Rich Boden	Lecturer in Ecology and Environmental Microbiology at Plymouth University
7th winner	2009	Tim Blower	Postdoctoral Researcher at University of Cambridge
8th winner	2010	Nabil Wilf	Postdoctoral Fellow at the MRC Laboratory of Molecular Biology
9th winner	2011	Marian Killip	Research Fellow at the University of St Andrews
10th winner	2012	<i>Could it be you?</i>	

Didn't nominate for the 2012 competition? It's not too late. Contact k.mcgregor@sgm.ac.uk

“Consider who is in your audience and that they won't necessarily be familiar with the science you present and the acronyms you use.”

James Edwards 1st prize 2005

STEPHEN GRIFFIN

1st prize 2003

EDUCATION AND EMPLOYMENT HISTORY

- BA (Hons) Biological Sciences, University of Cambridge
- PhD, University of Cambridge
- MRC Postdoctoral Researcher, University of Leeds
- Wellcome Trust Postdoctoral Researcher, University of Leeds
- MRC New Investigator Independent Fellow, University of Leeds
- Senior Fellow and Group Leader (Antivirals and Viral Oncology), Leeds Institute of Molecular Medicine

I was in the first year of my first postdoc when I entered the competition, mainly motivated by the thought 'why not' and was hugely surprised when I won. The prize is well regarded, particularly in the UK, but as well as giving me CV points I found that winning the prize really helped me focus on my research and my career. The response I got following participating in the competition showed me that my research was something people had an interest in and that others felt that there were 'good legs' on it. This made me more confident and assertive, and spurred me on to co-write my first grant application for my next job. This provided a basis on which to establish this research and subsequently develop my career to a stage where I've become relatively well established within my field.

As a scientist, communicating what you are doing through talks and papers is as an essential part of the job; I would say it accounts for about a quarter of what I do, far more if you include writing grant applications. It is really important that you are able to present information clearly and concisely in science. This becomes even more important as time goes on (e.g. going for fellowship interviews) – if you can't get your ideas across in a 10-minute slot then you may struggle to progress.

ADVICE FOR 2012 NOMINEES HOPING TO BECOME FINALISTS

Be confident ... don't be overwhelmed by the fact that it is a big meeting. I was totally surprised to be selected (and flabbergasted when I won) – there's no reason why anyone else can't do the same!



TIPS FOR 2012 FINALISTS

Practise, practise and then practise some more! This was mentioned by all of the previous finalists I spoke to. It will help you feel more relaxed and comfortable on the day and will also help you achieve the second-most mentioned tip – keep to time!

To learn more about what makes a good conference presentation, see the Gradline article in the February 2010 issue of *Microbiology Today* for advice from Rich Boden (1st prize 2008): www.sgm.ac.uk/pubs/micro_today/pdf/021009.pdf

KIRSTY ROSS

3rd Prize 2007

EDUCATION AND EMPLOYMENT HISTORY

- BSc (Hons) Biological Sciences: Microbiology and Infection, University of Glasgow
- PhD, University of Glasgow
- Postdoctoral Research Assistant, University of Glasgow
- Postdoctoral Research Associate, University of Strathclyde

I knew early in my undergraduate studies that I wanted a career pursuing scientific research. Keen to take charge of my career, I tried hard to make the most of my PhD experience; attending skills and development courses offered by the university, demonstrating to undergraduates and joining learned societies. In the second year of my PhD I entered the *Young Microbiologist of the Year* competition. I had given oral presentations as part of my undergraduate and postgraduate education and felt relatively confident about my presentation skills. That said, my competition presentation was only the second one I had ever given at a conference and I was definitely shaking while I delivered it!

Participating in this competition made me feel a lot more confident in my ability to present my data to a diverse audience. Good communication skills are vital to my current work as a researcher in communicating with colleagues and collaborators as well as with the children I meet as part of my outreach activities.

ADVICE FOR 2012 NOMINEES HOPING TO BECOME FINALISTS

Stay by your poster at all opportunities to give the judges a chance to talk to you. Have a 1- to 2-minute 'elevator' pitch prepared summarizing your work and why it is important.



TIM BLOWER

1st prize 2009

EDUCATION AND EMPLOYMENT HISTORY

- MA and MSc in Natural Sciences, Robinson College, University of Cambridge
- PhD, Department of Biochemistry, University of Cambridge
- Postdoctoral Research Assistant, Department of Biochemistry, University of Cambridge

Members of my lab had been finalists (and prize winners) in previous years so I was familiar with the competition and how it worked. When it came to the point where I felt I had made sufficient progress with my work (towards the end of the third year of my PhD) it seemed natural that I should enter – nothing to lose by doing so! I found the atmosphere of the competition session more receptive than at a general conference. I felt the audience appreciated the additional nerves associated with the competitive aspect and it felt as though the audience collectively wanted all the finalists to perform well. The organizers provided useful constructive criticism on my presentation and the whole experience of participating in the competition provided me with confidence to push myself in presenting my work outside of my lab and the UK. It was good to know that my work was considered of sufficient interest and quality by other academics.

ADVICE FOR 2012 NOMINEES HOPING TO BECOME FINALISTS

Make your poster big and bold – keep text to a minimum and make figures large enough to be visible without the reader having to stand and squint.

Media training

Opportunities for Society for General Microbiology (SGM) members



sense about science

SENSE ABOUT SCIENCE: ASK FOR EVIDENCE

Are you fed up with inaccurate science reporting in the media?

Do you want to ensure microbiology is represented in the best possible way?

SGM supports the following courses to help prepare you for talking to journalists:



– Standing up for Science media workshops



– Introduction to the news media

To organize your place on one of these free courses, please contact:

Laura Udakis, Press & Social Media Officer, SGM

t +44 (0)118 988 1843; e l.udakis@sgm.ac.uk

Every day we are bombarded with claims in publications, adverts and policy announcements, some of which lack the scientific evidence to back them up. When the evidence is not readily available we, as consumers, voters and patients, can ask for it.

THE VOICE OF YOUNG SCIENCE (VOYS) network, coordinated by Sense About Science, has done this before. In 2009, they asked companies for the evidence behind claims they were making for detox products, special diets, tonics and supplements. None of the companies contacted were able to provide any evidence, or give a comprehensive definition of what they meant by 'detox' and were often surprised to be asked. Worryingly, many of their claims about how the body works were wrong and in some cases the suggested remedies were potentially dangerous. Sense About Science concluded that 'detox', as used in product marketing, is a myth. We reviewed progress in 2011 and found that while some of the original products VoYS investigated are no longer claiming to 'detox', there are plenty of new products that do, including detox dental treatments and methods for mental detox.

Claims like this will keep cropping up. The only way to make a permanent difference is for everybody to ask for evidence for every claim they see. Sense About Science launched a campaign called 'Ask for Evidence' that encourages anyone, regardless of their level of expertise, to demand the evidence for a claim. By doing this we hope that anyone making a claim will ultimately expect to be asked.

VoYS is a network of early-career researchers who want to play an active role in public debates about science. Following Sense About Science's first 'Standing up for Science' workshop in 2005, participants kept in touch, forming the beginning of the network, which continues to grow.

Sense About Science coordinates four workshops each year; the most recent was the first to be held in Glasgow. Amongst the group of 45 attendees, some of whom were members of SGM, there was a feisty and dynamic discussion about the responsibilities of scientists and journalists in reporting science. Participants told us that the workshop provided the chance to share experiences with other people at the same stage of their career and to hear from journalists about how the media works, and get tips on how to get involved. Drawing on the experiences of our panel of scientists, the participants discussed the barriers to communicating science to the public, including what happens when research



Listen to what SGM members who attended a recent Standing up for Science media workshop thought.



MICROBIOLOGY IN THE PRESS, ON AIR AND ONLINE

announcements go wrong, statistics are manipulated, risk factors are distorted, or the discussions become polarized. The next workshop will be held in Manchester on Friday 23 March – see www.senseaboutscience.org/a4e for details. In the meantime, you can join in with the evidence hunting. Head to the website to read other people's stories, find tips and advice on how to ask for evidence, and get in touch to let us know how you got on.

For help asking for evidence or to find out more about VoYS go to www.senseaboutscience.org. Follow on Twitter @senseaboutsci and @voiceofyoungsci. Tweet about the campaign #askforevidence

If you want to know whether a claim made in a policy, newspaper article, advert or product is backed by scientific evidence, use Sense About Science's postcard to ask for evidence. Or fill in their online form www.senseaboutscience.org/ask.php. SGM supports the Ask for Evidence campaign. Visit our campaigns page at www.sgm.ac.uk/news/campaigns.cfm



If you would like to attend a VoYS Standing up for Science workshop, please contact Laura Udakis (l.udakis@sgm.ac.uk).

WHAT SGM MEMBERS THOUGHT...

'There are many nonsense science stories around and the workshop has helped me realized some of the reasons for this and what scientists can do to change that and put across their side of the story. A lot of work is being done is by young scientists and I think it's important that they get out there and communicate with the public.'

Adam Kucharski University of Cambridge

'As young scientists there are ways for us to get involved in promoting good science – organizations such as Sense About Science have a lot of opportunities to get involved.'

Avika Ruparell University of Nottingham

'A great opportunity to meet other researchers from different institutes and share our experiences.'

Alpesh Thakker University of Nottingham

'The workshop gave an insight into the different approaches of the media and science - journalists have only a short time to finish an article and scientists usually spend years on the same project'

Jana Hiltner University of Strathclyde

MICROBLOG WAS BORN

in Matt's garden on a sunny afternoon in early May. I had popped round for tea with my former supervisor and his family. We were chatting about microbiology and how my science writing was going at the Wellcome Trust. 'We should start a microbiology blog', Matt suggested. I'd been looking for a new writing project so I quickly agreed.

Often these ideas come to nothing, but we've kept going – 9 months and counting. We've made some errors, but with Matt's impeccable microbiology knowledge, and my writing and editing skills, I think we're on the right track. Indeed, it was Matt who came up with the name for our project. Easy to remember, I'm sure you'll agree. Except there



M I C R O B E L O G



was a small problem. When we were starting out, if you were to Google 'Microbelog', the search ending would assume you meant to look for 'Microblog' and direct you to microblogging sites. Thankfully we're over that now and if you search for us, there we are...

We built the site on Wordpress; it's very easy to set up and post articles. Most importantly it's free! We have a budget of exactly zero. As such, we've had to be clever – for example, we tend to use images from Flickr or the Public Library of Science that are freely available under a Creative Commons license. We also have Kirsty, one of my former colleagues and an amazing editor, who fixes any grammar errors that slip past Matt or myself. The distance from good to great is small, and she's really helping us to get there. Of course we utilize social media (@themicrobelog), but whether this helps to drive traffic is debatable. Recently, Microbelog has been accepted into ResearchBlogging.org, an aggregator site for blogs that contain peer-reviewed science. It's great for us and shows that we produce quality content.

But why are we doing this? Most importantly, we both love microbiology – it makes writing and

research much easier! Second, we both really want to produce accessible content about microbiology that's suitable for everyone, be they researchers or the general public. Science papers can be really complicated – sometimes unnecessarily so – but every lab has an interesting story to tell. We really want to highlight these for a more general audience, and deal with other issues that are important to those working in the field. We recently had an article by Professor Iain Sutcliffe, Editor-in-Chief of the journal *Antonie van Leeuwenhoek*, about the declining impact factors of microbiology journals.

Ultimately we want to create a community resource with content made by us

HTTP://MICROBLOG.WORDPRESS.COM

and, more importantly, other microbiologists. We've had several articles written by friends or former colleagues but we need more. We need you! Whether you're a PhD student or a head of department, you have a story to tell. For students wanting to get into science communication, practising your writing is invaluable (and looks good on your CV), and for faculty, well, it counts as an outreach activity...

So have a read. Go to the site and see what you think. If you like it and would like to get involved, get in touch. We'd be delighted to hear from you!

BENJAMIN THOMPSON is a science writer and co-founder of Microbelog

MATT HUTCHINGS is a Lecturer in Microbiology at the University of East Anglia

Q How did you make the move from lab-based work to policy? Was it a difficult decision?

A This is always a gradual process, but quite natural for scientists who are interested in seeing their research having a wide range of impacts. We generate knowledge and we have an interest in both how that knowledge is produced and how it is used. One of my first 'policy' experiences was on the Council of SGM and it was very valuable understanding both how to support this area of science and who we needed to tell about what we do (government, policy-makers, industry, etc.). For the past 10 years I have been on the Natural Environment Research Council (NERC) and again, this has been a great experience both meeting scientists from different areas of environmental research as well as setting strategies and supporting the case for future Government funding for environmental science. Of course, my most recent role as the first Chief Scientific Adviser for Scotland was the biggest change as that was a 4-day-a-week secondment and meant that

Chief Scientific Advisor to the European Union President: Professor Anne Glover

As highlighted in the News section, we are very pleased to announce that SGM member Professor Anne Glover, former Chief Scientific Advisor for Scotland, has been appointed as Chief Scientific Advisor to the European Union President. In this interview for *Microbiology Today*, Anne gives an insight into her new role.

I could not spend so much time on my research. However, the overwhelming positive is that you know what you are doing is making a significant difference. It wasn't a difficult decision to make, I saw it as a welcome challenge to add something different to what I do.

Q What were the most significant achievements of your time as Chief Scientific Advisor for Scotland?

A I was able to raise the agenda around science, engineering and technology in Scotland. I was able to provide robust independent analysis of Scotland's global contribution in these areas



(relative to our GDP, Scotland is No. 1 in the world in terms of research impact) and helped to ensure that the contribution Scotland's science base could make in the UK was visible. I was also able to raise the profile of SET in Scotland, giving it a loud voice, and I translated science issues for politicians, allowing them to identify the value SET has for our society and economy. Ensuring that we both attract and retain women in SET has been an important issue for me too and I am pleased that this agenda is being pursued with enthusiasm in our HEIs and by Government. These are just some highlights for me.

Q What are the most enjoyable and frustrating parts of your role in science policy?

A It really is all enjoyable. There are times that evidence is ignored in policy-making or at best has a very minor input, but as long as the reasons for this are clearly transparent, e.g. the evidence suggests X but for social reasons we are doing Y, then I think we have a strong process. It is important that we move to a more evidence-led policy cycle, so that is a continuing challenge.

Q What will be your top challenges in your new role as Chief Scientific Advisor to the EU President?

A At the moment, it is my great pleasure to be finding out about the European Commission, the science it funds and the challenges it faces. It would be premature of me to set out my agenda at this early stage, but I want to

see Europe recognized as the leading global force in science and I would like to see the citizens of Europe benefitting from that in a wide range of ways.

Q Is it difficult to work in areas of science outside your area of expertise? And how does the process work – do you work with a team of advisors?

A No one can be an expert on everything, so it is necessary to have a wide network of experts who are willing to provide advice and analysis, sometimes at short notice. I have been delighted at the generosity with which other scientists and engineers do this. In particular I have been supported by the Scottish Science Advisory Council, the Royal Society of Edinburgh and the Royal Society as well as by a wide range of individual scientists and engineers and other learned societies.

Q When there is clear scientific evidence supporting a specific issue, how difficult is it to translate this into government policy?

A Sometimes this is easy in as much as the evidence is compelling and there is little difficulty in determining the resulting policy; for example, this would apply to the policy relating to the legislation around passive smoking. In other areas, it is more difficult because there are conflicts with political imperatives or because there are vociferous, although not always representative, pressure groups who feel strongly about an issue which can distort the policy environment. But this is democracy. One of the roles of a Chief Scientific Adviser is ensuring that evidence also has a very strong voice.

Q What advice would you give a scientist wishing to get involved in science policy, at any level?

A Well of course I would encourage them to get involved. Many scientists are happy to complain about funding or regulation but do nothing to contribute constructively to the argument. If they venture into science policy, they can help to change things; that has to be an attractive option. It is always good to 'dip your toe in the water' first and so it's useful to speak to colleagues who are already involved to see how they find it. For a first-hand experience it is usually possible to ask if you can attend as an

PROMOTING UNDERSTANDING AMONGST PARLIAMENTARIANS

observer on various Boards or strategy groups. (The UK Resource Centre for Women organize a very valuable mentoring/shadowing scheme like this for women to attend senior Boards, although I am very disappointed that the UK Government intends to withdraw 100% of their funding for UKRC by 2012.)

Q I know that you are a strong supporter of Women in Science, Engineering and Technology? How do you see your personal role in this and what should others do to help?

A For me this is a basic equality issue. Women and men who have a passion and ability for science, engineering and technology should be encouraged and supported to pursue this. If we as a society do this (and we need to encourage our best and brightest young people to consider a career in SET if we are all to have a successful future), then we are foolish in the extreme if we do not continue to support the best to progress in their careers after our initial investment. This means supporting particularly women to be able to have families but to continue with their careers. I think it is time to examine our approach to parental leave to have a more equitable and enlightened view about what is appropriate for the 21st century.

With regards to my personal role, I think my visibility is important as other women can see that it is possible to do anything you want, you just have to be determined. In practical terms, I am also very careful when asked for a suggestion for a committee, a nomination for a prize or an interview, etc., that I try to take a few minutes to make sure that my list includes able women and men in equal measure.

'Antibiotic Action' –



Professor Laura Piddock, BSAC President, with The Rt Hon Kevin Barron, MP for Rother Valley, Chair of the Health Select Committee and parliamentary sponsor of *Antibiotic Action*. BSAC

FROM THE OUTSET *Antibiotic Action* has been unapologetic in its aims. *Antibiotic Action* is a UK-led initiative that calls for global action to ensure replenishment of the drug discovery and development markets for antibacterial agents. It makes clear that nothing less will do, that the time for discussion is over and that the time for action is now. A campaign strapline ends 'determined to succeed', and the first 3 months of activity certainly seem to signify that these were not hollow words.

Two recent papers published in *The Lancet Infectious Diseases* and *Science in Parliament* present succinct scientific and lay overviews of the pending crisis of no new antibiotics, and say more about the need for *Antibiotic Action* than can be reported here.

The initiative was soft-launched in mid-October by the release of the *Antibiotic Action* website – www.antibiotic-action.com. The initial response was overwhelming. In the 3 weeks prior to a

House of Commons launch on 9 November, the online petition received almost 5,000 signatures, easily exceeding the 500 required for delivery to 10 Downing Street. UK parliamentarians aside, the initiative captured the interest and imagination of colleagues across the globe. The American Society of Microbiology, Infectious Diseases Society of America and numerous European societies, including the Inter-regional Association for Clinical Microbiology & Antimicrobial Chemotherapy (IACMAC), Société Française de Microbiologie (SFM) and the Hellenic Societies, signified their support within a matter of days of the website being launched. Patient organizations, charities and industry also pledged support and asked how they could be involved, not forgetting members of the public who showed their support by sharing stories and signing the petition.

Sponsored by Kevin Barron MP, former chair of the Health Select Committee, the parliamentary event resulted

securing the future of antibacterial development, determined to make a difference

in swift and strong support, including the tabling of an Early Day Motion and support for a new all-party parliamentary group to support antibiotic research and development. At the launch event, attended amongst others by Shadow Health Secretary, Andy Burnham, Mr Barron urged parliamentary colleagues to support the initiative, and they have done so cross-party.

Spearheaded by Professor Laura Piddock, work is underway to consolidate and build on the early successes of the campaign. *Antibiotic Action* is looking forward to an exciting 2012 and maintaining the incredible momentum that all involved helped achieve in 2011. To this end the petition on the website will remain open and used as a tool to maintain pressure on governments and policy-makers to address the various issues in antimicrobial chemotherapy and not just re-generate antibacterial drug discovery, research and

In October 2011, the British Society for Antimicrobial Chemotherapy (BSAC) launched *Antibiotic Action*, providing a platform informing all about the need for discovery, development and appropriate use of antibiotic agents.

development. Next steps will include:

- taking forward the establishment of an all-party parliamentary group in the UK, for which BSAC will provide administrative support
- working with the World Health Organization to help deliver their action plan on antibiotic resistance under a Memorandum of Understanding to be signed at the end of January 2012
- working with like organizations in other countries to establish the *Antibiotic Action* initiative to help deliver their national

priorities in antimicrobial chemotherapy

- establishing a network of *Antibiotic Action* 'champions' to work together to raise the profile of the campaign, continue gathering signatures on the petition and effect action locally, nationally and internationally.

Antibiotic Action's long-term aspiration is the establishment of a public-private global alliance to meet its aims, and particularly a model by which antimicrobial development can be successfully taken forward. There is no intention to 'reinvent wheels', but rather capitalize on or adapt those that are already in existence – such as the models employed by the GAVI Alliance and Bill & Melinda Gates Foundation for the successful delivery of vital vaccines worldwide.

Antibiotics are the medical miracle that changed the face of modern medicine; *Antibiotic Action* with support and help from all concerned, is ready to do battle to ensure they remain so.

Join now at www.antibiotic-action.com

LAURA PIDDOCK is President of BSAC and Professor of Microbiology at the University of Birmingham

'Antibiotic Action' launch at the House of Commons

IMAGINE A WORLD where we have a severely reduced life expectancy, where there are high levels of infant and adult mortality resulting from simple, everyday infections, where routine surgical procedures are life-threatening, and where cystic fibrosis patients rarely live beyond 5 years of age. Well, we all know that this world doesn't exist anymore don't we? Surely, it's just a description of what life was like in the pre-antibiotic era, not a vision of life in 10–20 years time?



House of Commons. BSAC

Life in a post-antibiotic era is unthinkable in an age when we take our ability to control infection for granted, but a return to the pre-antibiotic age is just around the corner if no action is taken now to develop new antibiotics. This is the message that the BSAC was sending out at the launch of its new global initiative *Antibiotic Action* at the House of Commons on 9 November 2011.

In the shadow of a huge inflatable ball printed with the names of many of the 4,500 people who had signed a petition in support of *Antibiotic Action*, attendees sat down to listen to a series of impassioned short talks.

Following a welcome address by **KEVIN BARRON MP**, sponsor of the event, **PROFESSOR LAURA PIDDOCK** (BSAC President) presented the opening talk '*Antibiotic Action*' – *why this, why now?* Laura described how, despite an increase in antibiotic development up to the 1990s, only four antibiotics had been developed in the last 4 years. She commented how problems with cancer drug availability causes public outrage, but that the desperate need for new antibiotics does not. She described how no new antibiotics had been developed recently because funding of basic research is inadequate, licensing and regulation is currently not fit for purpose, and there is no return on investment for development. There was a need to encourage the private sector by making it quicker, easier and cheaper to license and regulate drugs, and a global alliance was required to fund public-private partnerships to the cover the cost of development (as much as £500m for a single drug), with some of the profit going back into the system. Laura finished by reminding us that the success with vaccine development over recent years needs to be repeated with antibiotics.

DR HILARY JONES, a GP and TV personality, highlighted what he saw as the three main problems we need to tackle urgently – the rise of resistance, emergence of multidrug-resistant infections and failure of pharmaceutical markets. He emphasized that there was a real danger that we will soon return to a 'pre-' antibiotic era where even simple wounds result in fatal infections.

Dr Jones' talk was followed by a moving presentation from cystic fibrosis (CF) patient, **SHARON BRENNAN**, who described what antibiotics meant to her life. CF used to be considered a childhood disease; in the 1960s, life expectancy with this condition was just 5 years. Sharon is now 30, but only thanks to antibiotics.



Professor Laura Piddock, BSAC President, Dr Matthew Dryden, BSAC General Secretary, and Dr Hilary Jones, GP and TV doctor, delivering the petition to 10 Downing Street. BSAC

She is currently on a regime of three different antibiotics, with four intravenous treatments per year and two 2-week courses 6 times a year. This course has to be constantly reviewed as microbes become 'cleverer' and R&D struggles to keep pace. Sharon also talked about how fewer people in work due to illness would be bad for society and the economy, not to mention the financial burden to the NHS.

The final talk, a call for action, came from **DR MATTHEW DRYDEN**, a physician from Winchester and General Secretary of BSAC. He started by asking for a show of hands – only one person in the room had never had a course of antibiotics. He highlighted how antibiotics have saved more lives than any other group of drugs, and how although good infection control is fine, it only holds back the inevitable tide of resistance. Collaboration between public, private, academic and regulatory bodies was urgently required, and this would also bring economic benefit to UK pharmaceutical companies.

The session was concluded by a stimulating question and answer session in which many topics were discussed by interested parties and MPs in the audience. The potential cost burden for the NHS if

nothing is done was highlighted, as was the necessity for more interaction between academia and industry. One member of the audience presented the alarming statistic that female UTIs caused by *Escherichia coli* affect 50% of women in their lifetime. Such infections could become untreatable and life-threatening within 10–20 years. Regulation had to be eased and costs for companies must be made easier to bear. It was pointed out that now we understand the evolution of resistance and resistance mechanisms better, future antibiotics could be developed that would be less badly affected by resistance.

Whitehall and Westminster were eerily quiet as Professor Piddock and her colleagues travelled to Downing Street to deliver the signed petition to the Prime Minister (the police had cordoned off the entire area in preparation for a protest march later that afternoon), but with the petition successfully delivered, an Early Day Motion tabled, and parliamentary support for an All Party Parliamentary Group to tackle these issues, the day could be considered as a great success and will provide a firm platform from which to proceed.

IAN ATHERTON is Design Manager and Managing Editor of *Microbiology Today*

WITH ITS €2 BILLION

funding scheme, IMI is the world's largest public-private partnership in drug research. IMI's ambitious projects give researchers from academic groups, small and medium-sized enterprises (SMEs), and other organizations in the health sector the opportunity to work with top scientists from industry and translate their know-how and expertise into innovative therapies. With their strong focus on open collaboration, partners in IMI projects gain access to clinical data and research results that would otherwise be unavailable to them. The antimicrobial resistance project is not IMI's first foray into infectious disease research. The RAPP-ID project (www.rapp-id.eu) is working to develop point-of-care tests (POCTs) that will provide clinicians with information in less than 2 hours on the cause of a patient's infection and, in the case of bacterial infections, whether the bacteria are resistant to certain drugs. This will allow doctors to administer the right medicines sooner. A project on tuberculosis is in the pipeline.

'Antimicrobial resistance is a major challenge to all involved in drug development', commented IMI Executive Director Professor Michel Goldman. 'I am optimistic that by bringing together Europe's leading experts from academia and industry, IMI will contribute to the generation of urgently needed new medicines in this area.'

Initial results from ongoing projects demonstrate the success of IMI's public-private model at boosting drug development.

The Innovative Medicines Initiative (IMI) will launch a major Call for proposals in the first half of 2012 in the area of '*Tackling resistance to antibiotics: building partnerships to progress the discovery and development of novel antibiotic drugs to treat the most urgent infections*'. The IMI Call represents a key pillar in the EU's recently launched action plan on antibiotic resistance. More information on IMI's antimicrobial resistance Call for proposals is available on the IMI website.

IMI to launch major antimicrobial resistance project

For instance, the IMIDIA project (www.imidia.org) has generated an innovative tool which will help researchers to understand diabetes and to test potential new drugs in laboratory conditions. This achievement is recognized by the scientific community as a major breakthrough in diabetes research. In the drug safety field, eTOX (www.etoxproject.eu) has developed an innovative computer model that predicts if a candidate drug is likely to cause serious heart problems in patients. The new eTOX system provides better results than the computational systems currently used, and should help researchers pick up drug safety problems earlier on in the drug development process.

Finally, IMI's Education & Training projects provide courses and information for students and scientists that wish to develop their career in the area of drug safety, preclinical testing or any other aspect of drug discovery and development. This will allow industry and other research organizations to recruit better-trained scientists. Find out more about IMI at www.imi.europa.eu

FURTHER READING

http://ec.europa.eu/dgs/health_consumer/docs/communication_amr_2011_748_en.pdf



FOR SEVERAL YEARS,

undergraduate microbiology students at Manchester Metropolitan University (MMU) have undertaken a cross-disciplinary project where they explore the relationships between microbiology and art. The outputs from this project have really demonstrated the skills and interests that students possess in addition to science, showing expertise in artwork, photography, textiles, models, music, animations, education materials and so on. To celebrate this work, I curated an exhibition during the 2011 Manchester Science Festival where several of the pieces were displayed. I was successful in an application for an SGM Public Understanding of Microbiology grant, which allowed me to produce a catalogue to accompany the exhibition. The catalogue described the background to the project, as well as the microbiology underpinning all of the exhibits on show (some copies of the catalogue are available on request).

The preview took place on the evening prior to the beginning of the Manchester Science Festival, at 'MadLab' (Manchester Digital Laboratory) in Manchester's 'bohemian Northern Quarter'. In addition to several large student exhibits and a continuous projection of the entire output, three projects were showcased: the winner of a microbiology and art competition open to the public; the MMU AIDS



Microbiology at the Manchester Science Festival

banner, produced in 2009 as a collaborative community project between myself and a textile artist; and the Manchester Microbe Map, an interactive Google map depicting contamination of bus stops in popular routes in Manchester. This map represented the results of the first project taken on by the Citizen Science Manchester DIYBio project, sponsored by the Wellcome Trust. The exhibition continued over 3 weeks and attracted

enthusiastic interest from audiences that, perhaps, tended not to be typical for public engagement with microbiology.

MMU held a more traditional 'family fun day' during the Festival, which included a microbiology session developed by my PhD students (Sarah Jackson and James Redfern, assisted by Katherine Suddards, Anne Leahy-Gilmartin and David Wickens), focusing on *Plaque attack!* Members of the public – particularly families – were invited to disclose their plaque, look at their plaque biofilm under the microscope, investigate the effect of the biofilm matrix on plaque removal using shaving gel, Fimo modelling clay and a spray bottle (many thanks to Nicola Stanley-Wall at



Photos from the exhibition preview evening at 'MadLab' Jo Verran



Dundee for advice and ideas), and construct the components of a biofilm using Model Magic clay. Using a small Polaroid printer bluetoothed to a laptop, we were able to provide all our guests with pictures of their own plaque before they left the lab, as well as toothpaste and a toothbrush courtesy of Unilever at Port Sunlight. We also intend to build the biofilm for display on our website. It was pretty labour-intensive, but feedback was really excellent, including the statement that *'this should be compulsory at all primary schools!'*

Our public engagement activities for 2011 ended on World AIDS Day (1 December), where a month-long exhibition displaying the MMU AIDS banner was



launched at the Manchester People's History Museum. SGM had supported the initial collaboration which led to the production of the banner (see the February 2010 *Microbiology Today* article at www.sgm.ac.uk/pubs/micro_today/pdf/021010.pdf), and Lynn Settingington, the textile artist, and I wanted to show the framed 4' x 4' piece to a wider public (it was also displayed at the SGM symposium in York in September 2011). We put together information about HIV/AIDS (including SGM's literature) together with the story of the development of the banner, advertised the event on the World AIDS Day website, and got sponsorship for the event from the School of Healthcare Science at the University. Well over 50 people came to the launch, including the different communities (schools, students, women's groups, visitors to the Whitworth art gallery) who had helped to produce the banner, and several members of Manchester Age Concern's LGBT group who had walked across the city from the AIDS memorial, prior to attending the vigil that evening. The Bad Bugs Bookclub held a meeting where we discussed *'28 stories of AIDS in Africa'* by Stephanie Nolen: each story describing one individual's experience of HIV/AIDS, and each representing 1 million people who had been affected by AIDS (see the meeting report and reading guide at www.hsri.mmu.ac.uk/badbugsbookclub). It was heartening to accompany and update the book with the relatively positive UNAIDS World AIDS Day report for 2011 (<http://bit.ly/zVmVd>) which could envision *'a world with zero new HIV infections, zero discrimination and zero AIDS-related deaths.'*

These activities take time, but the rewards are significant: student participation enriches their skills portfolio and improves communication skills and science literacy; different public audiences can engage with science in novel ways; everyone enjoys hands-on learning; and scientists are reminded of the value and impact of their work, and the need to tell other people about it. Well, that's how I feel – and it's fun too! Thanks again to all concerned.

JOANNA VERRAN is SGM Education and Public Affairs Officer, Dept of Biology Chemistry & Health Science, MMU, Chester Street, Manchester M1 5GD (email j.verran@mmu.ac.uk)

It was always intended to donate the completed banner for appropriate display, so we are delighted that its new home will be the Terrence Higgins Trust.

Polio end game

Polio is a viral disease that occurs in childhood and leaves the debilitating effects of post-polio syndrome for life. Humans are the only natural host for this virus, which is an important reason why it should be possible to totally eradicate the disease. Thanks to vaccination programmes that provide lifelong protection, the world is now at a point where the wild poliovirus is near eradication, after only 1,349 cases in 2010 (compared to 350,000 in 1988). Philip D. Minor from the National Institute of Biological Standards and Control in the UK has recently reviewed whether the world can take the final step to end polio disease. There are serious political, logistical and financial obstacles, for example in delivering vaccines in war zones, but there are also important biological issues with long-term implications, involving the nature of the disease and the vaccines that have led to near-eradication. His review shows that decisions with long-lasting consequences must soon be made by the world public health community.

Most people infected by poliovirus experience no symptoms, or at worst a fever and sore throat, with the virus remaining confined to the gut and passing away in faeces. However, in around 1% of people it damages motor neurones and then the muscles waste away, giving weakness or paralysis that continues throughout life. There are still around 120,000 people in the UK suffering from severe after-effects, even though new infections have ceased due to very effective vaccination programmes started in the 1960s. By the mid-1970s, polio had been controlled and essentially eradicated in developed countries like Europe and the USA. Two types of vaccine were involved: an injected inactivated polio vaccine (IPV) developed by Salk, which induced immunity and prevented virus spread within the body, and an oral polio vaccine (OPV) containing live but attenuated virus developed by Sabin that broke transmission through the environment.

Following successful mass-vaccination campaigns in South America in the 1980s, the World Health Organization proposed to free the world of polio by 2000. National Immunization Days were a major strategy, followed by surveillance for new cases and poliovirus in sewage with further rounds of vaccination as necessary. As an impressive result, by 2000 the only

regions where wild poliovirus remained were in the Indian subcontinent and central Africa. The regions with endemic infections are now even smaller, but there have been several outbreaks in countries including China, Indonesia, the Russian Federation and Yemen, in part caused by wars, political differences and worker migration, as well as poor surveillance.

With wild virus sources on their way out, attention has turned more carefully to man-made sources of the disease, particularly the live-attenuated vaccine OPV. Right from the start it was known that the virus could increase its neurovirulence after passage through a human gut, and since the 1980s there has been solid evidence that OPV causes disease in about 1 in 500,000 first-time vaccinees. For this reason, vaccination in developed countries has changed to IPV, where the virus cannot cause disease. Analysis using molecular biology is also now being applied to the live-attenuated vaccine strains to identify the mutations causing their distinct properties and discover whether better strains can be created.

The circumstances that generate vaccine-derived polioviruses to circulate in the community are unclear, although the presence of non-immunized children is essential. Researchers want to know how the attenuated strains regain virulence in the human gut. There is no way to prevent virus shedding into faeces and, very rarely, some

Minor, P.D. (2012). The polio-eradication programme and issues of the end game. *J Gen Virol* 93, 457–474.



Ancient Egyptian carving from 1500 BC showing a priest with a shrivelled leg typical of polio. Carlsberg Museum, Copenhagen

immunodeficient people have continued to shed virus for over 25 years that can infect unvaccinated individuals. Surprisingly, the type of gut cell infected by poliovirus is still unknown. Several virulent strains have developed, some by mutation, others by recombination with currently unidentified enteroviruses. Improved understanding of the evolutionary pressures on poliovirus in the gut could help with construction of improved vaccine strains, as well as with treatments for those few individuals who remain infected.

Since most poliovirus infections have no apparent effect, eradication means total removal of the virus and not just the disease, otherwise it will spread again. The issue of the vaccine-derived virus giving rise to new virulent strains is therefore of increasing importance now that wild poliovirus is nearly eliminated. If vaccination programmes stop, these new strains will start to circulate

widely and begin to undo all the good work of the last 50 years. Even though the human and monetary value of eradicating the disease can readily be calculated to be higher, continuing vaccination and surveillance in 2011/12 will cost around US\$1 billion. With the progressive reduction in cases, there has to be a decision on what to do if polio disease is to end.

The official definition of eradication is that 3 years should have elapsed since the last case of wild-type polio disease, so the world is still several years from this point. In addition, strategies to limit outbreaks from vaccine-derived virus, especially if mass vaccination ceases, are still uncertain. However, decisions about vaccine production, especially if new vaccines are to be developed, must be made many years in advance of need. The use of IPV is increasing: production of IPV depends on the use of wild-type paralytic strains of polio and improving the safety of production systems, for example by using safer strains of virus, is a major area of interest. With the only four licensed producers located in western Europe, companies in India and China have become interested in entering this market.

Understanding the pathogenesis and virology of the infection is of major significance as the programme nears its end and a major danger to total eradication becomes the protective vaccine itself. This must be solved to bring over half a century of global endeavour to a satisfactory conclusion.

Communication block

Ever since researchers realized that cell-to-cell communication was essential for some bacteria to cause disease, they have tried to block it to provide new antimicrobial treatments. The phenomenon, called quorum sensing, involves bacteria using the concentration of chemicals they secrete to assess their numbers. Once a pre-determined level is exceeded, all the bacteria activate the formation of biofilms and production of toxins, enzymes and other factors that enhance pathogenesis. This simultaneous assault from a large number of bacterial cells is much more effective in overwhelming the infected host's defences.

Developing therapies that stop quorum sensing is not straightforward. Some of the most effective chemicals for this purpose are too toxic for pharmaceutical use. However, researchers have been using nature as inspiration, searching for animals and plants that have evolved natural blocking systems. It turns out that some plants contain compounds that block quorum sensing. These give starting points for the development of effective pharmaceuticals. Testing extracts from plants already documented to have medicinal properties seems obvious, but an international team of researchers have now found a promising lead from a tree where antimicrobial properties had not been recorded before.

The attractive *Lagerstroemia speciosa* tree originates from tropical South-East Asia, but has now been planted around the world in suitable climates, resulting in many local names including Jarul, Banaba, Pride of India and Crepe myrtle. It has spectacular sprays of large crinkly-petalled white, pink or lilac flowers followed by oval, brownish fruit that remain on the tree, splitting open into decorative dry pods. Extracts from all parts of the tree are used in traditional herbal medicine for anti-obesity and anti-diabetic treatments.

A team of researchers, led by the Director of the National Botanical Research Institute at Lucknow, India, Dr Chandra Shekhar Nautiyal, and involving researchers from India, the USA and Saudi Arabia, has tested ethanolic extracts of *L. speciosa* fruit for anti-quorum sensing

activity. The researchers monitored test bacteria for production of biofilms, enzymes involved in pathogenesis and coloured metabolites, all of which only occurred after detection of quorum sensing signalling molecules. In these tests, the fruit extract inhibited signalling between the bacteria but did not inhibit their growth.

The team is now working to discover the nature of the anti-quorum sensing chemical in the fruit extract, but they speculate that it may be structurally similar to the unstable chemical furanones already known

Singh, B.N., Singh H.B., Singh, A., Singh, B.R., Mishra, A. & Nautiyal, C.S. (2012). *Lagerstroemia speciosa* fruit extract modulates quorum sensing-controlled virulence factor production and biofilm formation in *Pseudomonas aeruginosa*. *Microbiology* 158, 529–538.

to block quorum sensing. However, the molecules in the fruit extract appear to be more stable, opening up new opportunities for pharmacology.



Crepe myrtle flowers. iStockphoto / Thinkstock

Under pressure

Life at volcanic deep-sea hydrothermal vents and so-called black smokers thrives under conditions very different from those at the surface. Piezophiles, also called barophiles, thrive at high pressure and can die once depressurized. Retrieving samples from the ocean's depths while maintaining them at the correct temperature and pressure is a substantial technical challenge. Trying to isolate and characterize bacteria from these samples under their native pressures has rarely been attempted. However, in March 2007 researchers led by Daniel Prieur from Université de Bretagne Occidentale on the Serpentine cruise of the research vessel *Pourquoi pas?* achieved this using a remotely operated submersible and custom-built incubation equipment.

The samples came from a depth of 4,100 m at the Ashadze site in the Mid-Atlantic Ridge. It is the deepest hydrothermal vent field located so far. The pressure here is around 42 MPa, compared with around 0.1 MPa at sea level. Back in the

Birrien, J.-L., Zeng, X., Jebbar, M., Cambon-Bonavita, M.-A., Quérellou, J., Oger, P., Bienvenu, N., Xiao, X. & Prieur, D. (2011). *Pyrococcus yayanosii* sp. nov., an obligate piezophilic hyperthermophilic archaeon isolated from a deep-sea hydrothermal vent. *Int J Syst Evol Microbiol* 61, 2827–2831.

laboratory, samples of rock suspensions collected into a sealed box near the black smoker were briefly depressurized for distribution into culture vessels and then incubated in the absence of oxygen at temperatures from 85 to 105°C at 42 MPa. Within 2 days the researchers could see bacterial growth and they isolated the organism through repeated dilutions while maintaining the high-temperature and high-pressure environment conducive to growth.

The bacterial cells were slightly irregular spheres with a tuft of flagella at one end to enable them to swim. Their preferred growth environment was strictly oxygen-free, at 98°C and at a pressure of 52 MPa in a solution containing 3.5% salt, sodium sulfide, ammonium sulfate and a complex mixture of proteins, carbohydrates and vitamins obtained from meat and yeast extracts. The researchers tested growth on a wide range of single-carbon, nitrogen and sulfur compounds to characterize the growth requirements more precisely. They also managed to extract DNA so that they

could compare the signature sequence within the ribosomal 16S rRNA gene with previously studied bacteria, and went on to sequence the whole of the bacterial genome.

The DNA analysis showed that the bacteria belonged to the genus *Pyrococcus* and were 99.4% similar to *P. furiosus*. Very high levels of similarity are typical of this genus, so to decide whether the bacteria were *P. furiosus*, or another species, they compared all the ribosomal protein sequences. This clearly showed that the bacteria belonged to a distinct species, notable because of its very thermophilic nature as well as its piezophilicity. It has been named *P. yayanosii* in honour of Aristides Yayanos, a pioneer of the study of piezophilic bacteria.

The environmental requirements of this species have posed a problem for bacterial systematists, since a novel species usually has to be deposited in two recognized bacterial culture collections in two countries for safe-keeping. For the moment *P. yayanosii* is only available from the Japanese Collection of Micro-organisms and the much smaller 'Souchothèque de Bretagne' collection in France as efforts continue to find another larger international culture collection that can cater to its unusual requirements.

Anemones at the Ashadze hydrothermal vent (13° N 44° 56' W). Ifremer / Serpentine 2007



Gonzales-Marin, C., Spratt, D.A., Millar, M.R., Simmonds, M., Kempley, S.T. & Allaker, R.P. (2012). Identification of bacteria and potential sources in neonates at risk of infection delivered by Caesarean and vaginal birth. *J Med Microbiol* 61, 31–41.

Neonatal detectives

Current routine bacterial identification depends on the ability to grow micro-organisms in the laboratory; however, researchers have known for decades that most bacterial species are difficult, or impossible, to grow. Fortunately, new methods that use bacterial DNA for identification bypass the need for growth and are revealing new insights into how bacteria affect us.

Researchers at Queen Mary University of London, working with colleagues at the Royal London Hospital, the Eastman Dental Institute and the Wolfson Institute of Preventive Medicine, have recently used DNA identification techniques to identify bacteria present during one of the most sensitive times in our lives, the birth. Various complications occur in around 11% of pregnancies in the UK and the relevance of bacterial infections to the outcome is often unclear. This makes it difficult to devise strategies to manage these pregnancies better.

As a step towards solving this problem, neonatal gastric aspirates (NGA), collected using a sterile tube passed into the stomach of the newborn in the first few hours after delivery



Newborn baby. iStockphoto / Thinkstock

and before feeding, are routinely screened in UK hospitals to investigate infections associated with adverse pregnancy outcomes. This viscous fluid should originate from swallowed amniotic fluid and be sterile, but bacteria could be acquired during or after delivery from the mother or hospital surroundings. The types of bacteria present could also be affected if the mother was receiving antibiotic therapy for an infection during pregnancy.

The researchers gained approval to carry out a systematic study to describe the bacterial prevalence in NGA from 240 newborns considered at risk of infection born through Caesarean and vaginal delivery. The routine microscopic and growth tests indicated that about 25% had bacteria present and provided some information on their identity. The researchers followed this with two molecular DNA tests which identified bacteria in 41% of the samples, representing 51 species. Many had not been detected in NGA before. The

researchers were given access to the medical records of the mothers and babies to help with interpretation of the results. There were some obvious conclusions, such as a good correlation between detection of bacteria in the routine hospital tests and the molecular analyses. This study also confirmed that prolonged rupture of membranes was a very good indicator of the presence of bacteria in the newborn, which is why antibiotic therapy has been provided routinely for this condition since the 1990s. The researchers also determined that some bacteria were acquired as contaminants during collection of the samples, rather than from the babies themselves.

However, this study has also indicated some practical areas for future research that, if confirmed, could improve the outcome of pregnancies. For example, an association has been proposed between gum infections and adverse pregnancy outcomes and this study adds evidence through detecting bacterial species in NGA that are known to be involved in gum disease. Another possible association was between antibiotic prophylaxis given to the mother and the bacterial species in NGA. Overall, the study certainly shows that building up data from further NGA analysis using molecular DNA methods will increase the value of current investigative screening in UK hospitals.

Emerging Trends in Antibacterial Discovery: Answering the Call to Arms

Editors A.A. Miller & P.F. Miller

Publisher Caister Academic Press (2011)

Details £180.00 | pp. 480 | ISBN 978-1-90445-589-9

Reviewer Matt Hutchings
University of East Anglia

This excellent volume is in itself a call to arms, or rather a call to the world's governments and research funding bodies to reduce the red tape involved in bringing a new drug to market and to start investing seriously in antibiotic discovery. As the excellent chapters on natural product antibiotics make clear, these are still our best hope of finding effective new antibacterials; we just have to be smarter about how we screen for new antibiotics to avoid the problems of rediscovery. This is backed up by scientists involved in the antibiotic discovery programmes of big Pharma which make it clear that high-throughput screening of compound libraries, to find inhibitors of essential bacterial enzymes, is no longer considered a viable route to develop new antibacterials. This book is an essential reference for anyone interested in antibiotic resistance or discovery but also contains interesting chapters on the human microbiota and on current strategies for vaccine development. I highly recommend that you add this to your shelves.

Kucers' The Use of Antibiotics, 6th edn

Editors M. Lindsay Grayson, S. Crowe, J. McCarthy, J. Mills, J. Mouton, R. Norrby, D. Paterson & M. Pfaller

Publisher Hodder Arnold Education (2010)

Details £450.00 | pp. 3,000 | ISBN 978-0-34092-767-0

Reviewer Karen McGregor, SGM

Alvis Kucers, one of Australia's leading infectious diseases physicians, wrote the first edition of *The Use of Antibiotics* in 1972, primarily to assist new registrars in understanding how best to use these agents in the treatment of infection. Kucers helmed the revision and updating of the book, aided by increasing numbers of contributing authors, through to the 5th edition in 1997. Although involved in the initial preparation of the 6th edition, Kucers died in 2007 prior to its publication. The 6th edition has been renamed in his honour.

The 6th edition is a major revision; consisting of more than 3,000 pages across two volumes with over 250 chapters written by 200 or so internationally renowned authors with expertise in all aspects of antimicrobial agents. Divided into four sections (antibacterials, antifungals, antivirals, and antiparasitic and antimalarial drugs), there is information on existing and new therapies as well as emerging drugs (not yet fully licensed). The comprehensively referenced information on each drug is easily navigable and highly structured according to a standard format and includes susceptibility and relevant resistance issues, formulations and dosing, pharmacokinetics and pharmacodynamics, and toxicity and drug distribution. Although not examined by this reviewer, it is worth noting that the 6th edition of the book includes access to an electronic version of the print material (accessible either online or on your personal computer), offering search functions, live references and the ability to customize the content by the addition of notes.

The book remains true to its original purpose and represents an invaluable and incomparably detailed reference for infectious disease clinicians at a time when issues of antimicrobial resistance are of paramount concern. It would also make a useful addition to the bookshelf of academic researchers working in this field and to medical school libraries.

Microbial Toxins Methods and Protocols

Editor O. Holst

Publisher Humana Press (2011)

Details £85.50 | pp. 235 | ISBN 978-1-61779-101-7

Reviewer Gavin Paterson, University of Cambridge

This book provides detailed step-by-step protocols for a wide variety of techniques related to microbial toxin detection and characterization. The protocols are easy to follow and are very well supported by key references, example data, extensive explanatory notes and troubleshooting advice. The chapters provide accessible insight far beyond that in journal references and are obviously written by authors highly familiar with the methods. Although each protocol is focused on a particular microbe or toxin, most of them could be readily modified for your favourite microbe or target of choice. In this case the book will be a helpful reference for those wishing to introduce these or related methods to their laboratory. Alternatively, some of the protocols are only appropriate for laboratories with specialized or expensive equipment. The book would nonetheless be very helpful should you be collaborating with such a laboratory. Probably only one or two chapters would be of interest to any single laboratory and so this book is most suitable for institutional purchase.

Stress Response in Pathogenic Bacteria

Editor S. Kidd

Publisher CABI Publishing (2011)

Details £85.00 | pp. 320 | ISBN 978-1-84593-760-7

Reviewer Conor O'Byrne, National University of Ireland

This timely volume fills a significant gap in the literature for a review of the mechanisms of stress tolerance that exist within pathogenic bacteria. Other books have dealt with microbial stress responses in more general terms, but there was a need for a review of stress responses specifically in the context of bacterial infections. It brings together a series of well-written reviews, authored by highly regarded researchers, which span a range of bacterial pathogens and different stresses that are relevant to infection. It covers molecular responses to the stresses associated with acid, oxygen, nitric oxide and metal ions particularly well. Obvious omissions include responses to osmotic stress and mechanisms of bile tolerance, both of which almost certainly play important roles in gastrointestinal infections. Despite this shortcoming, researchers with an interest in the molecular mechanisms that underpin the adaptations to the sometimes harsh environments encountered within the host will find this a very useful volume. Indeed, students embarking on research projects in this area will find this an invaluable starting point.

Marine Microbiology: Ecology and Applications, 2nd edn

Author C. Munn

Publisher Garland Science, Taylor & Francis Group (2011)

Details £41.00 | pp. 320 | ISBN 978-0-81536-517-4

Reviewer Russ Grant, University of York

This useful student-targeted text book provides a good general overview and useful detailed elements in marine microbiology. Focusing specifically on ecology and applications, it provides information rather than specific methods of ecology. It may not be the best to look at with a two-toned blue colouring and a few colour plates at the rear, but its strength lies within its simple layout. Starting with an excellent breakdown of its contents, it makes great use of sidebars and boxes to show relevant, interesting information, facts and research focuses, and ends with a well-written glossary.

The material is very up to date for a text book, taking account of relevant events in 2010 and with recent references, but does suffer from occasional grey literature usage and falls into the trap of the 'next generation' technologies moniker which are very much technologies of today (on writing), dating the book for future usage. The cost is a little high for those outside of the field, but provides an appropriate rounded overview of marine microbiology today.

Reviews on the web

Reviews of the following books are available on the website at www.sgm.ac.uk/pubs/micro_today/reviews.cfm

NF-κB in Health and Disease

Author M. Karin

Publisher Springer-Verlag GmbH & Co. KG (2011)

Details £126.00 | pp. 263 | ISBN 978-3-64216-016-5

Essentials of Clinical Mycology, 2nd edn

Editors C.A. Kauffman, P.G. Pappas, J.D. Sobel & W.E. Dismukes

Publisher Humana Press (2011)

Details £90.00 | pp. 568 | ISBN 978-1-44196-639-1

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microbiology careers

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www.sgm-microbiologycareers.org.uk



Professor Harry Smith CBE FRS (07.08.21–10.12.11)

PROFESSOR HARRY SMITH CBE FRS died peacefully at the age of 90 on 10 December 2011. He was a former President, Treasurer and Meetings Secretary of SGM, and an Honorary Member of the Society. When he was Meetings Secretary, the Society had only one member of staff based in the Institute of Biology in London, and SGM Officers completed their tasks from their home institutions. While he was the SGM Treasurer, he made the Society far more professional, but to avoid paying high London rents and wages, he persuaded Council to buy Harvest House in Reading, where property was still cheap and salaries were lower.

Harry Smith was the guru of microbial pathogenicity, renowned for pioneering studies of how bacteria survive *in vivo*. He first studied Pharmaceutical Chemistry at University College, Nottingham, when it was still an outpost of the University of London. His PhD involved the first chemical synthesis of a dinucleotide, and was examined by Professors Todd and Ingold. His intention had been to follow a career in chemistry, starting as



Courtesy Prof. Peter Owen, Microbiology Department, Moyné Institute, Trinity College, Dublin



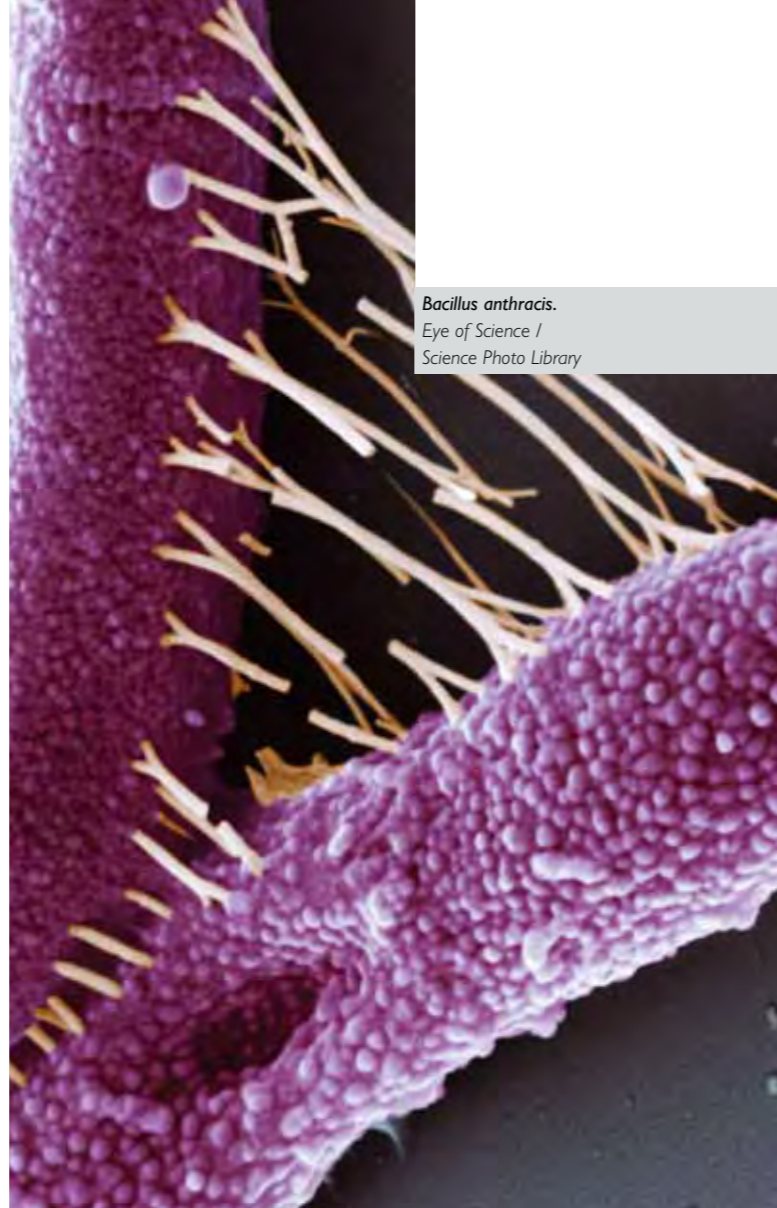
Harry was instrumental in the purchase of SGM's first permanent office, Harvest House, on London Road, Reading.
I. Atherton

a full Lecturer in Nottingham, where by now he had met and proposed to his lifelong partner, Janet. However, Lord Todd was instrumental in recruiting him to Porton Down, encouraged by the offer of a house and an extra £100 a year to his proposed £700 annual salary in Nottingham. He was assigned Dr David Henderson's Microbiology Section, where interest was turning to studies of microbial pathogenicity.

First, Harry investigated the virulence-enhancing properties of mucin. This led to the discovery that sometimes multiple factors combine synergistically to produce their biological effect. In the case of mucin, three factors were involved in the interaction: heparin, chondroitin sulfate, and blood group substance, none of which was active alone.

The anthrax project immediately followed the mucin studies. Extracts of *Bacillus anthracis* isolated from infected animals were not toxic, but plasma from these animals caused oedema when injected subcutaneously, and killed mice and guinea pigs when injected intravenously. The toxin was subsequently produced in culture and shown to consist of three components, none of which was toxic when injected alone. This was the first chemical analysis of bacteria harvested from infected animals: it had three repercussions. First, it stimulated fresh interest in a subject that had become moribund; second, it showed that toxins can be multi-component; and finally, it confirmed Harry's lifelong interest in microbial pathogenicity. Subsequent studies of plague and brucellosis at Porton are summarized in his 1958 article in *Annual Review of Microbiology* (12, 77–102).

In 1964, Harry successfully applied for the Chair in Microbiology at the University of Birmingham where he established a department focusing on plant, microbial and viral pathogenicity. A major project for many years was to determine the molecular basis of gonococcal serum resistance: in short, how do gonococci survive in the human body? It was almost 20 years later that, in collaboration with the author, we identified the nucleotide CMP-NANA as the host factor that protects the gonococcus against complement-mediated killing by sialylating its lipo-oligosaccharide. *Neisseria gonorrhoeae* is an obligate human pathogen: its ability to exploit host-derived CMP-NANA is an elegant example of molecular mimicry.



Bacillus anthracis.
Eye of Science /
Science Photo Library

Ten years after leaving Porton, an outbreak of anthrax in Russia caused alarm that the Russians were still working on germ warfare. He was appointed advisor to the UK Government on biological warfare and he recruited the virologist David Kelly to help him. Harry was possibly the last person to speak to Dr Kelly on the fateful day of his death. His CBE was awarded for services to the Ministry of Defence.

His many academic awards included the Stuart Mudd award in 1994, and visiting professorships at UCLA, Berkeley, Seattle, Ann Arbor and the University of Malaya, Kuala Lumpur. He loved interacting with young people. He also spotted talent, and went to extraordinary lengths to promote young, talented scientists.

He is survived by his lifelong partner, Janet, on whom he depended for wise council and moral support.

JEFF COLE, University of Birmingham

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RECENT REPORTS in the media raise concerns about the appropriateness and the accuracy of terminology used by microbiologists to communicate with the public and each other. A key issue has to be the casual and indiscriminate use of 'bug', which is prevalent in its use within the community of microbiologists. The term has a broad range of definitions, including (but not restricted to) an annoyance, a surveillance device and all sorts of small organisms, from viruses and bacteria to insects in the garden. More significantly, to the general population, 'bug' is the umbrella term of choice used to describe a variety of illnesses, including food poisoning, which may be caused by different types of organisms – notably, viruses and bacteria.

The outbreak of food poisoning centred in northern Germany during spring 2011 was picked up by the British media and reported widely. This story joined the notorious list of similar *Escherichia coli* outbreaks that have hit the headlines, with their associated morbidity and mortality issues. If a positive outcome of these recurring headlines could exist, then perhaps the repeated reporting of these stories would lead to factual accuracy. However, this last outbreak showed that such factual accuracy is not always apparent. Media reports described the steps being taken by the authorities to track down the bacterium responsible for this outbreak – the deadly but elusive '*E. coli* virus (*sic*)'. This highlights incidents where virus and bacterium are used interchangeably, revealing a clear misunderstanding of the differences between these organisms, or 'bugs'!

The reporting of this story highlights concerns about the use of 'bug' as appropriate scientific terminology. Rather than laying the blame at the door of sloppy reporting and the lack of scientific education of the average journalist, perhaps the problem lies closer to home? Turning the spotlight back on ourselves as microbiologists, let us examine the fine line between pedantry and literary ease, associated with the prevalence of and our willingness to describe all microscopic life forms as 'bugs'. The attraction of this term is clear: it is a shorthand, umbrella term that is accepted and understood by our peers and the public. Or is it? Perhaps by using this term we have blurred the key distinctions between microbial life forms. Therefore, should we be shocked when we have this lack of clarity reflected back at us?

But should it really be a concern that there is a lack of exactness in using this term? Let us review one of the key microbiological issues affecting humanity today: the growing number of disease-causing bacteria that are developing antibiotic resistance. This has been exacerbated by indiscriminate, mis-prescribing of antibiotics for infections caused by 'bugs'. To counteract this issue, many campaigns have been directed at governments, healthcare workers, the food industry and the general public with an emphasis on developing an

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A listening device, an annoyance, an illness or an indiscriminate term for various types of microbe – what is a 'bug', and does it have any place in microbiological parlance?

LAURA BOWATER

COMMENT

Stop bugging me!

understanding of the differences between microbiological taxonomy, especially those of viruses and bacteria. These campaigns show signs of success. For example, there is growing acceptance among healthcare workers and the public that a sore throat caused by a virus is a self-limiting illness that should no longer be treated with antibiotics, but a 'Strep' throat may be. Would this advance have been achieved if all sore throats had been described as 'throat bugs'? Compare this to our subtle acceptance of the phrase 'tummy bug' as a catch-all term and our consternation when it is replaced by the '*E. coli* virus'.

Clearly, a tension does exist when communicating with the public. Describing detailed and complex scientific concepts in a straightforward manner while maintaining integrity and accuracy is a challenge. However, I would argue that, as a scientific community, we are not meeting this challenge by our reliance on the term 'bug'. Instead, at the very least, as an umbrella term to describe any member of the microbiological kingdoms we should replace 'bug' with 'microbe' and instantly remove the confusion associated with illness, insect, annoyance and listening device. In fact, I would like to suggest that if we know our microbe's kingdom, its phylogeny or its classification then let's be bold and accurate, and use that instead. I agree that it will take a few more characters on the keyboard, a renewed confidence in the public's scientific literacy, as well as an additional clarity of thought from within the community of microbiologists. However, a positive outcome as far as I am concerned is that replacing the indiscriminate use of 'bug' with clearer and more accurate terminology will reduce the microbial confusion that is bugging me!

LAURA BOWATER, Norwich Medical School, University of East Anglia, Norwich Research Park, Norwich NR4 7TJ (email laura.bowater@uea.ac.uk)

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

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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: September 2011.
See Chloramphenicol Summary of Product Characteristics for full prescribing information.

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

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