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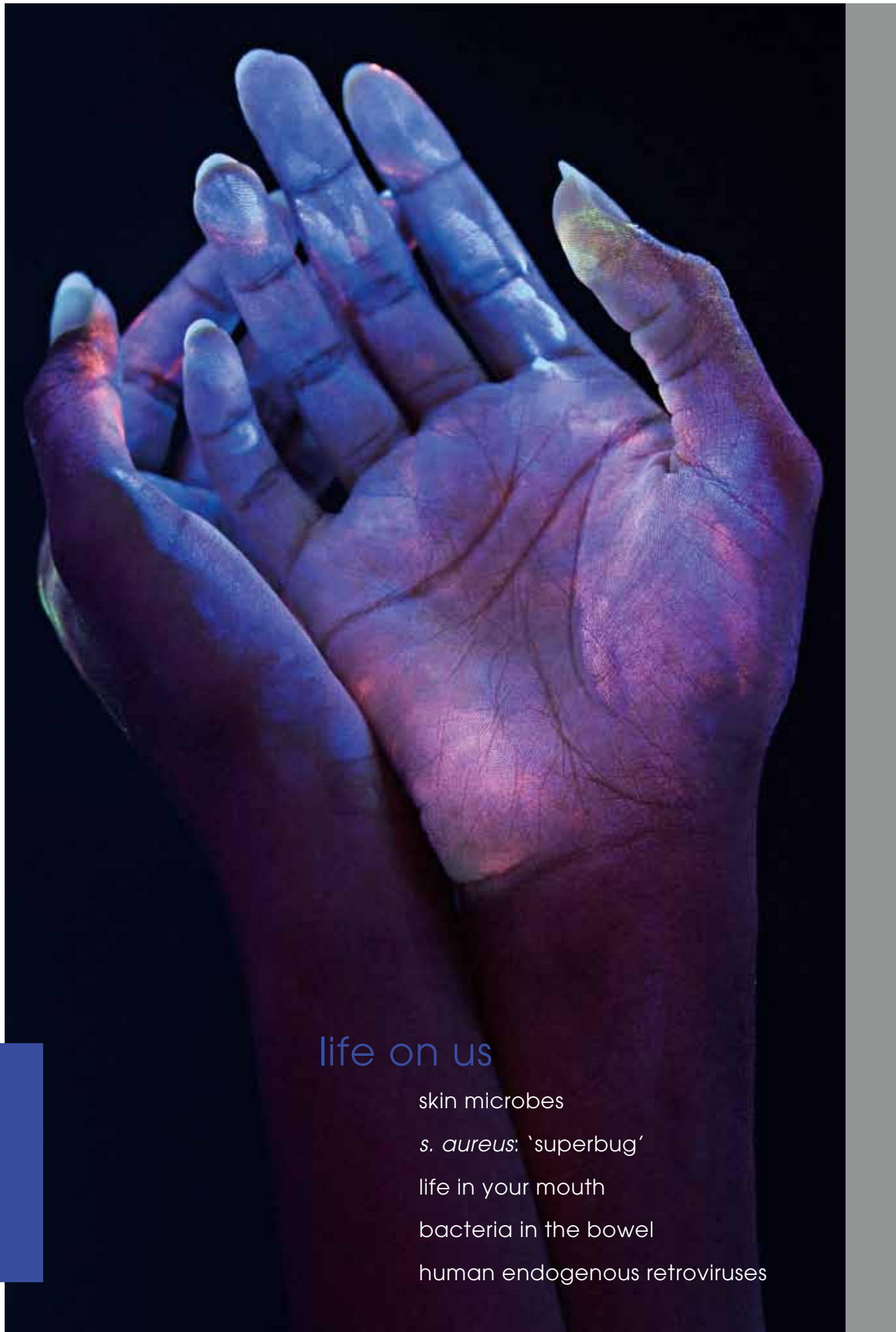

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Cover image Image taken in ultraviolet light of a woman's hands covered in bacteria. *Coneyl Jay / Science Photo Library*

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New Editor-in-Chief for JGV

Professor Richard Elliott

Professor Elliott began his 5-year term of office on 1 January.

I undertook my graduate studies in Oxford, under the supervision of the late Dr David Kelly, investigating protein synthesis by a large DNA virus, frog virus 3. In 1979 I moved to the laboratory of Dr Peter Palese in New York for postdoctoral training, taking a conscious decision to study an RNA virus (influenza virus) and to learn more about viral nucleic acids. At that time cDNA cloning and nucleic acid sequence determination were technologies still in their infancy, and I was involved in cloning influenza genome segments, including the first influenza B virus hemagglutinin gene. In 1981 the opportunity to return to the UK arose at the Institute of Virology in Glasgow with a remit to establish recombinant DNA technologies with negative strand RNA viruses, and from here my interest in bunyaviruses grew. I was awarded an MRC Senior Fellowship in 1986 and then appointed to a Personal Chair by Glasgow University in 1995. I served as Joint Head of the Division of Virology in Glasgow from 1998 until September 2005 when I moved to the University of St Andrews to take up the Chair of Virology. I was elected a Fellow of the Royal Society of Edinburgh in 1999.

European Society for Clinical Virology

SGM has been delighted to accept the invitation of the ESCV Council to provide administrative and financial services with effect from 2008. The Society will be processing all membership subscription applications and renewals and provide banking and accounting services for ESCV. The membership database will be hosted on the secure SGM server and online credit card payments will be available, a new facility for ESCV members. Most communications will be carried out by email. ESCV officers will continue to organize the Society's very successful scientific meetings and manage their website (www.escv.org).

ESCV President, Bruno Lina, commented, '*Through this agreement, ESCV's management can become more professional than in the past, and Executive Officers of ESCV will be able to focus on scientific matters rather than having to worry about administrative problems. Also, regular replacements of Officers, as required by the Society's Constitution, can take place without creating breaks in continuity of administration.*'

The ESCV arose from the merger of two European virology organizations in 1997. It aims to bring together scientists and clinicians throughout Europe and to promote public health and advance education, particularly medical education, in Clinical and Basic Virology. ESCV became a charity registered in England and Wales in 2003 and its registered office is already at Marlborough House, SGM's headquarters.

People

Congratulations to…

Professor Clive Ronson (Otago University, New Zealand) who has been elected as a Fellow of the Royal Society of New Zealand.

Brian G. Spratt (Professor of Molecular Microbiology at Imperial College London) on the award of a CBE for services to science in the New Years Honours 2008.

Professor Duncan Maskell (University of Cambridge) has been appointed a member of the General Advisory Committee on Science by the Food Standards Agency. This body will offer independent advice on how the FSA collects and uses scientific evidence.

Deaths

The Society notes with regret the death of **Dr K.R. Cameron**, Cockermouth, Cumbria (member since 1965).

SGM Council

Nominations 2008

Professors **Iain Hagan** and **Bert Rima**, retire from Council in September 2008. Nominations are invited to fill three vacancies on Council. All nominations must include the written consent of the nominee and the names of the proposer and seconder, both of whom must be Ordinary Members.

Members submitting nominations should indicate the main area of microbiological interest of their nominee, who must have been an ordinary member of the Society for at least two years. Nominations should be sent to the SGM General Secretary, Dr Ulrich Desselberger, c/o SGM Headquarters to arrive no later than **30 April 2008**.

SGM Council November meeting highlights

SGM Prizes 2008

Council approved the following awards:

Fleming Prize

Dr Cameron Paul Simmons, Oxford University Clinical Research Unit, Hospital for Tropical Diseases, Ho Chi Minh City, Vietnam, for his achievements in exploiting cutting-edge molecular and immunological techniques in the study of human tropical and emerging infections.

Marjory Stephenson Prize

Professor Alan B. Rickinson FRS, University of Birmingham, for outstanding achievements in research relating to the molecular biology and immunology of Epstein–Barr Virus and the immune surveillance and evasion of Burkitt's lymphoma.

Peter Wildy Prize

Dr Christopher Smith, University of Cambridge, for developing innovative ways of publicising and explaining achievements and problems in science, especially microbiology, to the public, such as radio broadcasts, the Nature podcasts and The Naked Scientists website.

A more detailed appreciaton of the prizewinners' work will appear elsewhere in *Microbiology Today*. The lectures will be delivered at SGM meetings in 2008.

The SGM Medal

Council agreed to create a new prize, called the SGM Medal. This will be bestowed annually (starting in 2009) on an individual from anywhere in the world whose research is of internationally high reputation and has been of significance in reaching beyond microbiology. The Prize comprises a Medal and a cheque for £1,000. The winner will deliver a lecture at an SGM meeting. A search committee will accept nominations until **31 August 2008** (udesselberger@btinternet.com) and make a recommendation to Council in November 2008.

Associate Membership

It was reported to Council that the new Associate Membership category, to include recent graduate and postgraduate students, early career postdoctoral fellows, clinical trainees, technicians and retired members, had been approved at the Society's Annual General Meeting in September 2007 and was now part of the bye-laws.

Joint ASM/SGM Burnet/Hayes Postgraduate Travel Awards

The Australian Society for Microbiology (ASM) and the SGM have agreed to establish ASM/SGM Burnet/Hayes Postgraduate Travel Awards. This scheme is designed to benefit PhD students in both countries by giving them the opportunity of travelling overseas to present their work and experience the best of microbiology in the partner country. Two awards will be made per annum, one in each country. Excellent PhD students will be able to apply to attend the annual meeting of either the ASM or the SGM where they are expected to make a presentation. They will then be able to visit laboratories which carry out research relating to their interests. The society of the country of origin will cover the costs of international air fare and national travel (as appropriate), accommodation and subsistence; the society of the host country will provide the conference registration fee. Details will be announced on the SGM website.

SGM Finances

At the end of the third quarter of 2007 SGM's finances were in a healthy state. Council decided to choose the tobacco-restricted option of Charishare for the UK equity component of the Society's investments portfolio.

Review of Council composition and functions

A short oral report was received from Petra Oyston who is chairing the working group looking at the responsibilities of members of Council, the roles of SGM officers and Elected Members, and the functioning of Council. A written report will be presented at the next Council meeting in February 2008 for detailed consideration.

Journal of General Virology

Professor Richard M. Elliott, University of St Andrews, will start as Editor-in-Chief of the *Journal of General Virology*, in January 2008 for a period of 5 years (for profile see p. 2). The President Robin Weiss thanked the retiring Editor-in-chief, Professor Geoffrey L. Smith, for all his efforts in maintaining and increasing the quality of the journal, and for his contributions to Council.

Safety of computer-stored information

Council learned that the Society was in the process of upgrading its email and internet connections and had taken further measures to increase the security of its computer-stored information.

***Ulrich Desselberger**, General Secretary*

Prize Lectureships

Fleming Lecturer

Dr Cameron Simmons will deliver his prize lecture, entitled *Understanding emerging pathogens: H5N1 influenza and Dengue in Vietnam*, on Wednesday 2 April 2008 at the Society's meeting at the Edinburgh International Conference Centre. The Fleming Lecture is awarded for outstanding research by a microbiologist in the early stages of their career.

Cameron Simmons is a Reader in Tropical Medicine at Oxford University and based at the Hospital for Tropical Diseases in Ho Chi Minh City, Vietnam. Cameron completed his PhD at the University of Melbourne (Australia) and postdoctoral studies at Imperial College (UK). Since arriving in Vietnam in 2001, Cameron's research focus has been in understanding the pathogenesis of important diseases in the region, beginning with tuberculous meningitis and, more recently, H5N1 influenza and dengue. These studies have been deliberately holistic and encompass investigations of the pathogen, immune response and host genetics. It is hoped these investigations will provide the foundation for improved patient management and new clinical interventions in these diseases. Cameron's contributions to dengue research have seen him appointed to various WHO advisory committees.

Cameron has three small children and loves tennis, football (Fulham), travel and (rarely) sleeping in on a Sunday morning!

Marjory Stephenson Prize Lecturer

Professor Alan B. Rickinson, FRS will deliver his prize lecture, entitled *Studies with an oncogenic virus: how to survive a lifetime with EBV*, on Tuesday 1 April 2008 at the Society's meeting at the Edinburgh International Conference Centre. The Marjory Stephenson Prize Lecture is awarded for an outstanding contribution of current importance in microbiology.

Alan Rickinson did his PhD in Radiotherapeutics in Cambridge and, after spending 3 years as a postdoctoral fellow in Sydney, moved to Bristol in 1972 to join Tony Epstein's group working on Epstein–Barr virus. There he began to study EBV's interaction with the B lymphoid system, the role of the virus in B cell lymphomagenesis, and later (in collaboration with Denis Moss in Brisbane) the host's cellular immune response to virus infection. Since moving to become Head of the CRUK Institute for Cancer Studies in Birmingham in 1983, he has continued these themes, working in the area where virology, immunology and oncology meet, and is currently developing and testing therapeutic vaccines against EBV-positive malignancies. He was elected a Fellow of the Royal Society in 1997.

SGM/RMS meeting – microscopy and microbes

Getting inside cells: the pathogens progress

This one-day meeting is a new collaboration between the Royal Microscopical Society (RMS) and SGM. The synergy is an obvious one – there are not a lot of microbes that can be seen with the naked eye – but up until now there have been no formal joint ventures.

The event takes place on Tuesday 24 June 2008 in London and will concentrate on the cellular aspects of infection and will consider both cell invasion processes and the 'tussle' that takes place once a pathogen has started to replicate within the host cell and the cell tries to fight back. Dr Mark Jepson (University of Bristol) will be looking at the way in which bacteria invade cells and then Prof. Urs Greber (University of Zürich) will describe interactions of viruses with the cell cytoskeleton. Dr Michelle S. Swanson (University of Michigan) will introduce the role of autophagy (a normal cellular process that helps cells last through times of nutrient stress), in bacterial and viral infection. There is some uncertainty as to whether pathogens induce autophagy as an aid to replication or the process is used by the cell as a defence mechanism.

There will be ample opportunity for delegates to present their own work either by poster or oral contribution. Oral contributions will be selected from the submitted abstracts.

Held alongside the prestigious Microscience 2008 exhibition and conference, at the Excel conference centre, delegates to the SGM-RMS meeting will have access to the exhibition where over 100 exhibitors will cover almost all aspects of light and electron microscopy. If it is new and exciting in microscopy – it will be there. SGM members will be able to register at RMS member rate (£45).

For further information email **paul.monaghan@bbsrc.ac.uk** or **clare@rms.org.uk**. For full details of the Microscience 2008 programme see **www.microscience.org.uk**

Organizers: ***Dr Paul Monaghan*** *(RMS) and* ***Professor Joanna Verran*** *(SGM)*

Grants

Travel & meetings

Postgraduate Student Conference Grants

All postgraduate student associate members are eligible to apply for a grant to support their attendance at one SGM meeting each year. Grants contribute to 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. Applicants must be Student Members resident and registered for PhD in an EU country. Closing date for the Edinburgh meeting: **28 March 2008**.

President's Fund for Research Visits

Up to £3,000 is available 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: **20 March** and **26 September 2008**.

Retired Member Grants

Contribute toward accommodation and the Society Dinner at one SGM meeting a year. Closing date for the Edinburgh meeting: **28 March 2008**.

Scientific Meetings Travel Grants

This scheme aims to support early-career microbiologists wishing to present work at a scientific meeting in the UK or overseas. Graduate research assistants and lecturers, and postdoctoral researchers (within 3 years of first appointment in all cases), and postgraduate students are eligible to apply. Retrospective applications are not considered.

SfAM/SGM Short Regional Meeting Grants

Contribution of up to £2,000 towards the costs of running a regional microbiology meeting.

SGM has a wide range of schemes to support microbiology. See **www.sgm.ac.uk/grants**

Enquiries should be made to the SGM Grants Office, Marlborough House, Basingstoke Road, Spencers Wood, Reading RG7 1AG (**t** 0118 988 1821; **f** 0118 988 5656; **e** grants@sgm.ac.uk).

Technician Meeting Grants

All associate members who are technicians are eligible to apply for a grant to support their attendance at one SGM meeting each year. Applicants need not be presenting work at the meeting. Some microbiology technicians who are not members of SGM may also apply for a grant to attend a Society Meeting. Closing date for the Edinburgh meeting: **20 March 2008**.

Studentships

Elective Grants

Funding for medical/dental/veterinary students to work on microbiological projects in their elective periods. The closing dates for applications in 2008 are **20 March** and **26 September**.

Education & development

National

Education Development Fund

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. Up to £1,000 is also available to support science promotion activities.

GRADSchool Grants

Postgraduate Student associate members who are not eligible for a free place on a UKGrad (www.grad.ac.uk) personal development course (National GRADSchool) may now apply for a grant from SGM to cover full course fees. Retrospective applications are not considered.

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

These cover the travel and other expenses of up to two speakers on microbiological topics per society each year at student society meetings.

International

IUMS-SGM Fellowships

These provide funding for young microbiologists from developing countries to pursue, or complete, part of an on-going research programme in a laboratory in a developed country and/or acquire theoretical or technical knowledge in their particular area of research. See **www.iums.org/outreach/outreach-fellowships.htm** for details.

International Development Fund

The Fund exists to provide training courses, publications and other help to microbiologists in developing countries. Applications for 2008 are invited. Closing date: **26 September 2008**.

The Watanabe Book Fund

Members who are permanently resident in a developing country may apply for funding to acquire microbiology books for their libraries. These annual awards are available as a result of a generous donation from Professor T. Watanabe of Japan. The following awards were made in 2007: *Edgar Sevilla–Reyes* (Mexico) and *Wasu Pathom–aree* (Thailand). Applications for 2008 are invited. Closing date: **26 September 2008**.

microshorts

Lucy Goodchild takes a look at some stories that have hit the headlines recently.

Bird 'flu transmitted to foetus

Scientists at Peking University, Beijing, China, studied the bodies of people who had died from H5N1 avian influenza and found that the virus infects organs other than the lungs and can even be transmitted from placenta to foetus. They detected genetic material from the virus as well as antigens in the lungs, trachea, lymph node T-cells, neurons and placental cells. Viral genetic material was also found in the intestinal mucosa. In an infected foetus, the lungs, circulating immune cells and liver all contained evidence of viral presence. Scientists are unsure about whether an infected foetus would survive if its mother survived infection, but they said '*this study has shown the capacity for human vertical transmission of the H5N1 virus*'.

http://multimedia.thelancet.com/pdf/press/H5N1.pdf

Hope for dengue fever patients

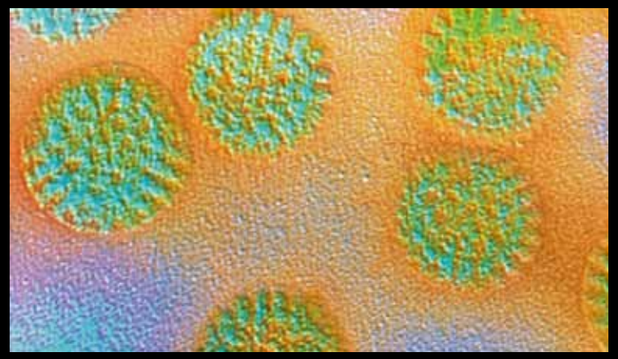
Dengue fever is caused by the most prevalent mosquito-borne virus that infects humans. Despite around 100 million people being infected, there is currently no treatment available. Scientists at the Novartis Institute for Tropical Diseases and the Genome Institute of Singapore have monitored the genetic responses of the dengue virus to analyse host–virus interactions and identify potential drug targets. Three pathways have been noted. Researchers hope to develop drugs that inhibit these pathways, in order to halt virus replication.

http://ntds.plosjournals.org/perlserv/?request=get-document&doi=doi/10.1371/journal.pntd.0000086

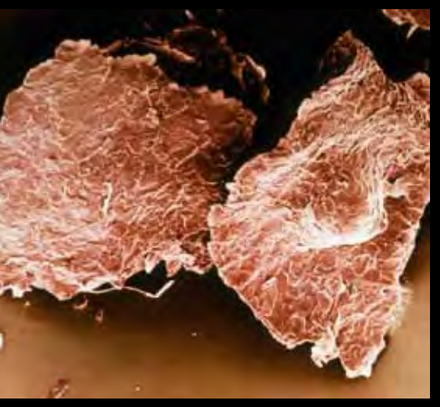
Probiotics to treat viral diarrhoea

Every year, more than half a million infants worldwide die as a result of diarrhoea caused by rotavirus. Probiotic bacteria, beloved of yoghurt advertisers, could soon be used to treat these infections. Even in industrialized countries rotavirus disease costs $1 billion per year. Researchers from the Karolinska Institute and the University of Linkoping in Sweden, and the Nestlé Research Center in Switzerland tested the effect of *Lactobacillus rhamnosus* in animal models. 59 % of the animals given probiotics before infection did not develop diarrhoea, compared with just 7 % in those without probiotics. Hyperimmune bovine colostrums (HBC), an antibody used to treat rotavirus infections, is expensive. If probiotics are used, the required dose of antibodies can be reduced by 90 %, which will be particularly beneficial in developing countries.

www.biomedcentral.com/bmcmicrobiol/



▲ False-colour transmission electron micrograph of rotavirus particles, a cause of viral diarrhoea. *CNRI / Science Photo Library*



Dandruff fungus genome sequenced

Scientists from Procter & Gamble have sequenced the genome of *Malassezia globosa*, the fungus that causes dandruff. As much as 50 % of people are affected by dandruff and similar disorders caused by fungi. Researchers looked at the most common type, which was stored at a fungus bank in Holland, and identified new targets for dandruff treatment. *M. globosa* uses 50 enzymes to digest the products of the skin's sebaceous glands, leading to the telltale symptoms of itching and flaking. The fungus is also involved in eczema, atopic dermatitis and psoriasis. Experts hope the research, which has been published in the *Proc Natl Acad Sci USA*, will lead to treatments for a closely related pathogenic plant fungus that infects crops like wheat and corn.

Proc Natl Acad Sci U S A (2007) **104**, 18370–18735

◀ False-colour scanning electron micrograph showing two human skin scales from a mild case of dandruff. *Dr Jeremy Burgess / Science Photo Library*

▶ Endoscope view of ulceration and inflammation in the ileum of a 42 year-old woman with Crohn's Disease. *David M. Martin MD / Science Photo Library*

New treatment for African sleeping sickness

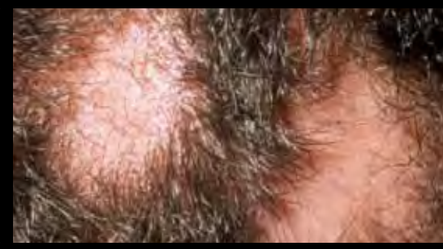
The standard treatment for human African trypanosomiasis, or African sleeping sickness, is highly toxic and kills a large percentage of patients every year. The disease-causing parasites have also started to become drug-resistant. Clinical trials in Uganda of a new drug combination have been successful, offering hope to over 15,000 people who are infected every year. The combination of nifurtimox and eflornithine caused no treatment-related deaths and it was successful in all patients. Although eflornithine must be administered intravenously and is a strain on health services, this is the best treatment available for the next decade, while new drugs are being developed.

http://ntds.plosjournals.org/perlserv/?request=get-document&doi=doi/10.1371/journal.pntd.0000064

Dental flora cause alopecia

Alopecia areata, or localized alopecia, affects 1 in 1,000 people. It is a dermatitis that causes bald patches, and was thought to be an autoimmune condition. Scientists at the University of Granada have found that the bald patches, which can occur on the scalp, beard and even eyebrows, are caused by 'infection outbreaks on the teeth'. Many people experience recurrences after regrowth because the hair follicles are not destroyed. Patches are usually on a line projected from the dental infection. The researchers advise people exhibiting alopecia areata to visit the dentist.

http://prensa.ugr.es/prensa/research/index.php



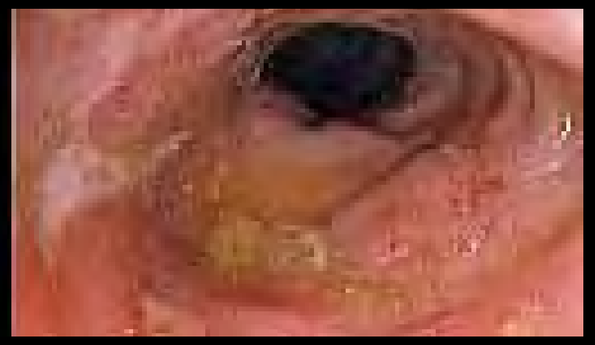
▲ False-colour scanning electron micrograph of *Trypanosoma brucei*, the cause of African sleeping sickness, amongst red blood cells. *Eye of Science / Science Photo Library*

▶ Alopecia areata. *Dr P. Marazzi / Science Photo Library*

Crohn's disease clue

'Sticky' *Escherichia coli* is known to be present in patients with Crohn's disease. Scientists have identified a bacterium that releases a molecule containing mannose, which in turn prevents macrophages from killing *E. coli* in the intestine. *Mycobacterium paratuberculosis*, which causes a disease called Johne's disease in cows, is transmitted to humans in dairy products. Researchers at the University of Liverpool suggest that this weakens the body's immune response to intestinal bacteria, resulting in symptoms like bleeding and diarrhoea. The team is planning to carry out clinical trials using antibiotics to target *M. paratuberculosis* in patients with Crohn's disease.

www.liv.ac.uk/newsroom/press_releases/2007/12/crohns-disease.htm



HIV helper identified

More than 90 % of HIV-1 infections result from sexual intercourse, but factors influencing the infectiveness of the virus in semen have been poorly understood until now. Researchers at the University Clinic of Ulm have discovered that prostatic acidic phosphatase (PAP), a molecule that is plentiful in semen, builds a sort of ferry for virus particles to help them infect cells. Amyloid fibrils (known as semen-derived enhancers of virus infection, SEVI) are formed, which capture HIV particles and help them penetrate target cells. SEVIs are so effective that they can enhance infection rates by several orders of magnitude; in some cases over 100,000-fold. The researchers have published their study in *Cell* and plan to explore how the fibrils allow the viruses to enter their target cells. Future findings could lead to drugs that block the process, which could be added to microbicide gels that are being developed.

Cell
14 December 2007

Melanin may be antimicrobial

In humans, the skin pigment melanin helps to protect the body against harmful UV rays producing the painfully familiar sunburn. Scientists know that, in invertebrates, melanin encapsulates invading parasites and fungi, which appear as black-brown spots on the shell. However, its effect on bacterial infections has been hotly debated. Writing in *J Biol Chem*, scientists say melanin protects crayfish from bacterial infections. The bacterium *Aeromonas hydrophila* is dangerous in crayfish; researchers found that effective melanin production is crucial to the survival of those infected.

J Biol Chem (2007) **282**, 21884–21888; doi:10.1074/jbc.M701635200

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Life on us

▶ *Adam and Eve*, 1526 (oil on panel, 117x80 cm) by Lucas Cranach I (1472–1553). Did the microbes of hunter–gatherers in Eden differ from those of modern humans? *Courtauld Institute of Art Gallery, London*

This issue of *Microbiology Today* takes a look at some of the microbes who share our bodies. SGM President **Robin Weiss** wonders just how human we are.

More than 30 years ago, Michael Andrews wrote an entertaining book entitled *The Life That Lives On Man* (London: Faber & Faber, 1976), concerning human ectoparasites such as fleas, lice, mites and ticks, and the diseases they carry: plague, typhus and lyme disease. This issue of *Microbiology Today* is devoted to the microbes that live on or in us. Humans are mobile ecosystems that harbour numerous bacteria, viruses, fungi and single-cell eukaryotes. Even our own genome has been repeatedly colonized by retroviral genomes constituting some 8 % of human DNA sequences (see Voisset & Griffiths, p. 30), and that's not counting other transposons and parasitic DNA.

Articles on the skin (Farrar & Bojar, p. 14, oral cavity (Spratt, p. 22), large bowel (Tannock, p. 26) – a veritable chemostat for *Escherichia coli* – illustrate what a wonderful habitat we provide for microbes. Relatively few of them are routinely pathogenic, but in specially susceptible hosts such as the immunocompromised individual, viruses and fungi that we carry for decades without ill effects can suddenly run wild. There is also the danger of MRSA leaving its commensal niche in our nostrils to invade surgical wounds with devastating effects (as Foster describes on p. 18).

As an ecosystem, it has become clear that we are only part human, because a significant amount of our biomass is microbial. In demographic terms, microbes outnumber our own cells. While there are 10^{14} human cells in the average adult, there are probably ~10^{15} bacteria and >10^{17} viruses associated with the human body. In terms of genetic diversity and complexity, the microbial metagenome of humans may be greater than the 3×10^{9} base pairs of human DNA.

Are the microbes that live on us on to a good thing? Yes, if we consider that for a large mammalian host, our current population of 6 billion is substantial, and widely distributed. Even viruses that do not actually infect us may find that humans serve as a useful vector for dispersal. At the September 2007 SGM meeting we were reminded by Mya Breitbart that the most prevalent virus to be found in human faeces is pepper mild mottle virus. This curious finding is thanks to our diet of solanacaceous vegetables (tomatoes, potatoes, peppers and so on), all of which came from the Americas. It is ironic that the post-Columbian exchange resulted in the introduction of novel foods (*Solanaceae* and *Zea mais*) from the New World to the Old, whereas almost all the pathogens such as smallpox, measles, yellow fever and tuberculosis made the transatlantic journey in the other direction. Since human numbers and activity affect the global ecosystem, it would seem sensible from a microbial point of view to adopt the attitude 'if you can't beat them, join them'. Humans certainly provided the SARS coronavirus with a brief world tour before it retreated to its fruit bat reservoir with occasional forays into civet cats.

Zoonoses in turn raise the question, how similar is our microbial flora to that of our closest living relative, the chimpanzee? It would be interesting to compare, say, the oral flora of chimpanzees to that of humans. Considering viruses, many of the ubiquitous, endemic not highly pathogenic viruses have co-evolved with the human host, e.g. herpesviruses. In contrast, most of the highly pathogenic, epidemic viruses came to humans from diverse animal sources, long after we left the forest for the savannah and eventually spread out of Africa. Many of them, such as measles, smallpox and influenza, became part of the human microbial scene only during the last 12,000 years or so, when we domesticated livestock, and when other animals, such as dogs, cats, rats and mice, chose to colonize human settlements. Thus while we share >98 % host DNA sequence similarity with the chimpanzee, the microbial and viral species that live in or on us are only ~50 % shared with the great apes.

Does life on us represent a harsh and challenging environment? It may sometimes seem so, given our modern habits of washing with soap, brushing our teeth, taking antibiotics and separating drinking water from sewage. But, as the articles in this issue illustrate, we still abound with microbes. One interesting human ecological niche (not explored here) is the vagina. Together with the uterine cervix, it is home to the symbiont *Lactobacillus acidophilus* and frequently to the more irritating *Candida* and *Gardnerella*, not to mention sexually transmitted papilloma viruses, herpes simplex virus type II, *Chlamydia, Gonococcus, Trepanema, Haemophilus ducreyi* and *Trichomonas*. The lactobacilli help to maintain the vaginal mucosal surface at pH 4.2, but after ejaculation of semen, this can rise to pH 8.0. This sudden shift from an acid to an alkaline milieu might strike an environmental microbiologist as dramatic, although I would not go as far as calling sexually transmitted microbes extremophiles!

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Skin microbes

Human skin may not be the most ideal habitat for micro-organisms, but as **Mark Farrar** and **Richard Bojar** report, the skin microflora is more species-rich than we think.

As a microbial habitat, human skin is somewhat inhospitable. Resident micro-organisms must be able to withstand a much drier and nutritionally limited environment than that found, for example, in the gut. As a consequence, there are relatively few microbial groups capable of colonizing human skin. The dominant micro-organisms belong to the genera *Staphylococcus*, *Propionibacterium* and *Corynebacterium*. Skin commensals can colonize both the skin surface and hair follicles. The distribution and density of these micro-organisms varies greatly over the body and is influenced mainly by nutrition and humidity. For example, total bacterial numbers per cm^2 of skin in the armpit or on the face can reach 10^7, whereas on the forearm numbers may only reach 10^2. On the whole our resident microflora exists without any detriment to human health and may actually play a protective role in preventing colonization by pathogens. However, occasionally our microflora can cause problems, some of which will be addressed in this article.

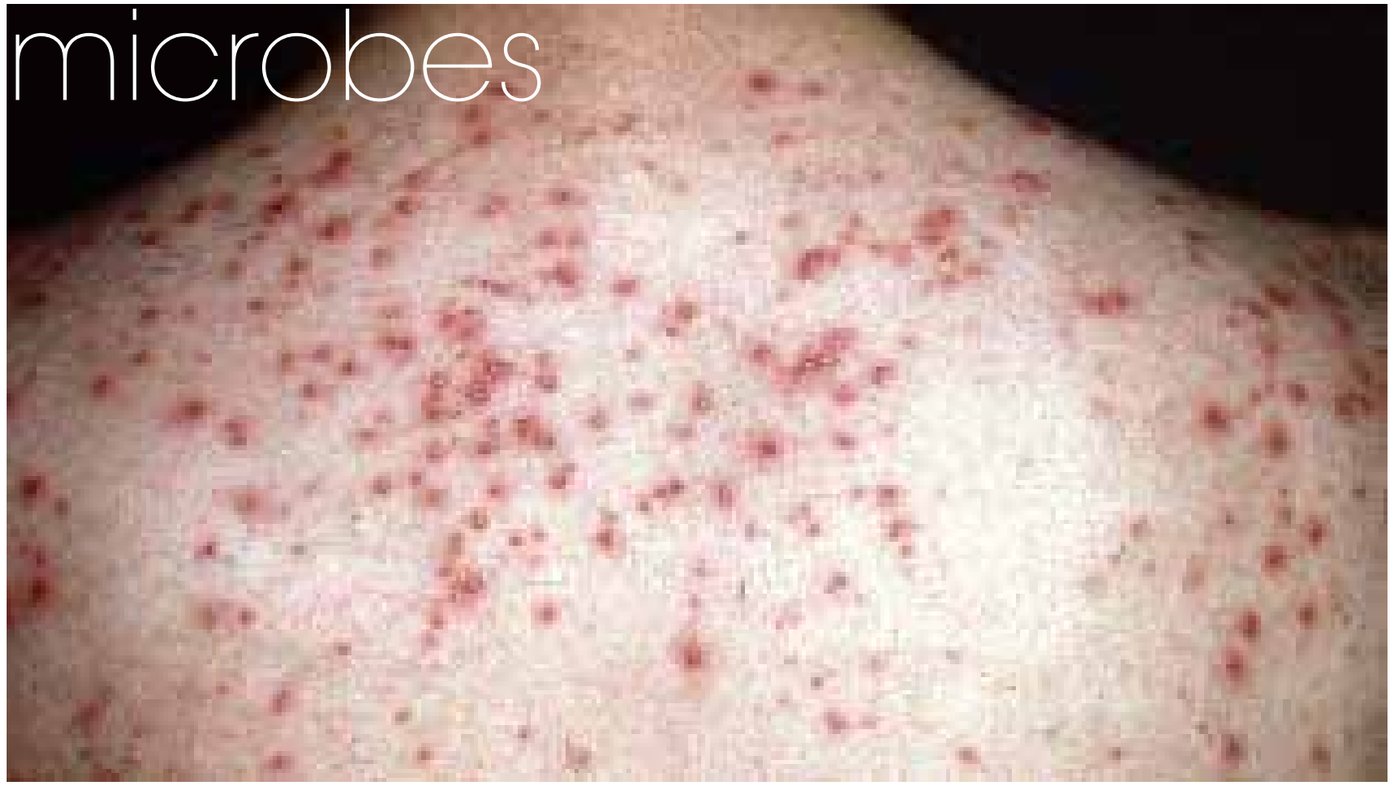
Staphylococci

Over 40 species of *Staphylococcus* are currently recognized, of which at least ten can be found on human skin. *Staphylococcus epidermidis* is the most prevalent member of the human skin microflora on most body sites. Other common skin residents belonging to this genus include *S. hominis*, *S. haemolyticus* and *S. capitis*. These are all coagulase-negative staphylococci. The coagulase-positive and more infamous *S. aureus* is not usually considered a resident of human skin but is found in the nose of approximately 30 % of the population. When found on skin it is most likely to be a transient colonization. Staphylococci are found in the highest numbers on the face and chest, and in some individuals in the armpit.

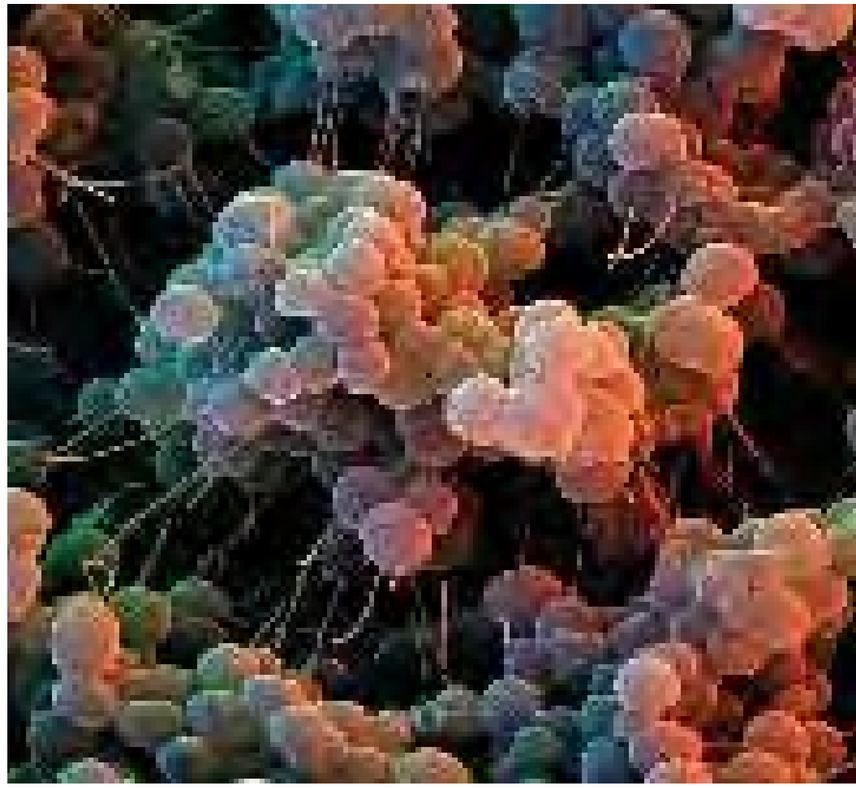
As pathogens, cutaneous staphylococci are most commonly associated with infections of catheters and prosthetic implants. *S. epidermidis* is by far the most common cause of such infections. The most significant virulence factor of coagulase-negative staphylococci is the production of extracellular polysaccharide or 'slime'. This is produced in large amounts by around 50 % of *S. epidermidis* isolates and enables the bacteria to adhere to and colonize medical devices. It also contributes to antibiotic resistance and interferes with removal of invading bacteria by the immune system. Consequently, staphylococcal infections of medical devices are difficult to treat.

Propionibacteria

Propionibacteria are most prevalent and found in the greatest numbers in lipid-rich areas of human skin, i.e. the face, chest and back. In the laboratory, propionibacteria are routinely isolated under anaerobic conditions. However, they are aerotolerant and growth has been shown to be increased in the presence of low concentrations of air, making them microaerophilic. Propionibacterial numbers on the face and back can reach 10^7 per cm^2 of skin. *Propionibacterium acnes*



▼ Inflammatory acne vulgaris on the back. *M.D. Farrar*

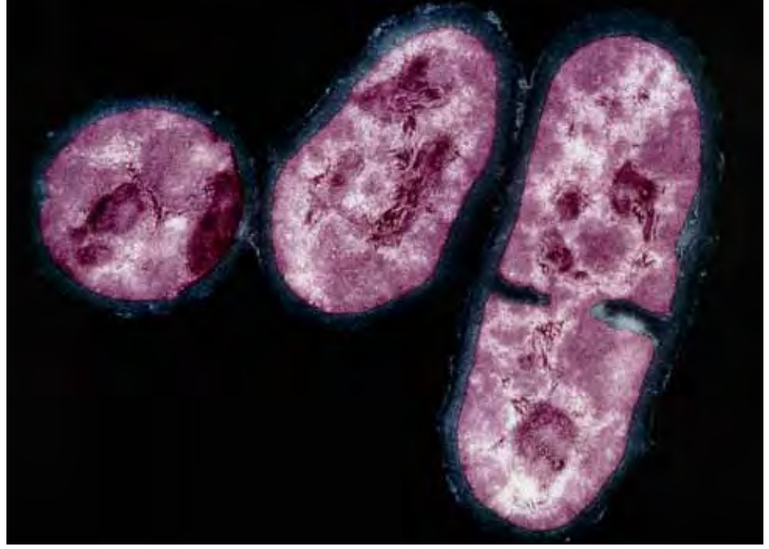


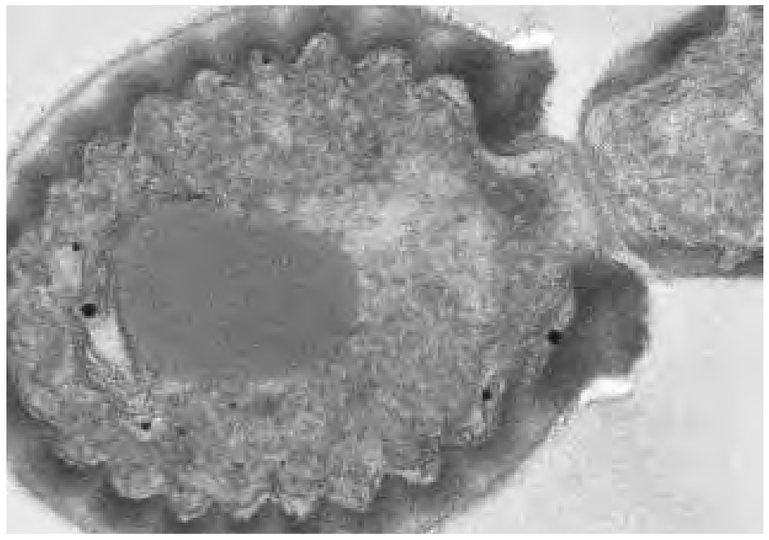
► Coloured scanning electron micrograph of clusters of *Staphylococcus epidermidis* bacteria. *David Scharf / Science Photo Library*

is the dominant member of the genus on skin. Other members of cutaneous propionibacteria include *P. granulosum*, *P. avidum* which is found in more humid areas such as the armpit, *P. propionicum* and *P. lymphophilum*. Propionibacteria have been associated with the common skin disease acne vulgaris for over 100 years. Although propionibacteria do not cause this disease, they are thought to be a significant factor in the development of inflammation. *P. acnes* has been the main focus of research into propionibacteria and acne due to its higher densities, but involvement of other propionibacteria and even members of other genera cannot be discounted. Skin propionibacteria are becoming an increasing cause of infections in immunocompromised individuals, particularly following surgical procedures. Endocarditis, eye infections and tissue infections have all been reported. Publication of the genome sequence of *P. acnes* in 2004 has helped to accelerate research, but the actual role of this organism in acne and the factors involved in the transition from harmless commensal to inflammatory stimulus are still unknown.

Corynebacteria

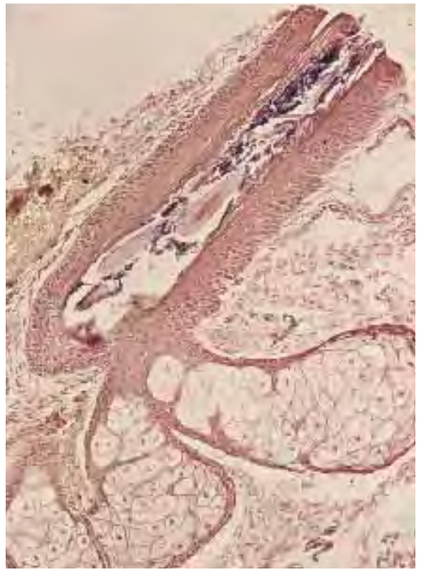
Corynebacteria are related to propionibacteria, but grow under aerobic conditions. This genus is less well characterized than *Staphylococcus* or *Propionibacterium*, but ribosomal RNA typing is helping to distinguish species. Skin residents include





▲ Coloured transmission electron micrograph of a section through *Propionibacterium acnes* bacteria. *Kwangshin Kim / Science Photo Library*

▲ Transmission electron micrograph of a budding *Malassezia* cell. *M.D. Farrar*



▶ Biopsy section of a human hair follicle showing Gram-positive micro-organisms within the follicle. *M.D. Farrar*

Molecular typing has shown there to be many more bacterial species present on human skin than previously thought

Corynebacterium bovis, *C. jeikeium* and *C. xerosis*. Corynebacteria are also found in lipid-rich areas and many are lipophilic. Recent sequencing of the genome of *C. jeikeium* has shown it to have a nutritional requirement for monounsaturated fatty acids, which it cannot synthesize. Corynebacteria are of great interest to the personal hygiene industry as they are thought to be major contributors to underarm body odour. Secretions from sweat and other skin glands can be metabolized to odorous compounds by this group of organisms. Many deodorant products contain antibacterial agents that aim to reduce bacterial numbers and therefore reduce the potential for odour formation. Corynebacteria may also cause opportunistic infections. *C. jeikeium* is recognized as a significant nosocomial pathogen and is resistant to several antibiotics, including erythromycin, tetracycline, kanamycin and chloramphenicol.

Malassezia spp.

From there being just one recognized species several years ago, molecular typing has led to the description of 13 species in this genus, 11 of which have been found on human skin. As with some of the bacterial groups already described, *Malassezia* spp. are also lipophilic. Members of this genus, in particular *M. globosa* and *M. restricta*, are thought to be involved in the pathogenesis of seborrheic dermatitis, a severe form of dandruff. Although their role is not exactly clear, disease is believed to develop due to an inappropriate immune response to the organism.

Other skin residents

Other micro-organisms are found on human skin and are recognized as commensals, although they are found in much lower numbers than the groups described above. True commensals include members of the genera *Micrococcus*, *Brevibacterium*, *Kytococcus* and *Dermacoccus* (both formerly classified as micrococci), and the Gram-negative *Acinetobacter*. All these can be regularly isolated, albeit in low numbers, from human skin. Less prevalent and probably considered transients rather than true commensals are species of *Streptococcus* and *Peptostreptococcus*.

Is the skin microflora more diverse than we thought?

With advances in molecular typing and classification of micro-organisms through the use of ribosomal RNA sequencing, the microbial diversity of numerous habitats has been shown to be greatly underestimated. Recently, such techniques have been applied to human skin. This has shown there to be many more bacterial species present on human skin than previously thought. This is mostly due to previous studies relying solely on culture techniques. However, a note of caution should be applied; to date, molecular studies have looked at samples at only a single point in time. This makes it difficult to distinguish those organisms that are true residents from transiently colonizing micro-organisms and environmental contaminants. If we are to truly understand the diversity of the human skin microflora, more long-term studies are required where multiple samples are taken from individuals over a period of time. However, those studies carried out to date are important as they have shown the skin to be a potential habitat for a diverse range of micro-organisms.

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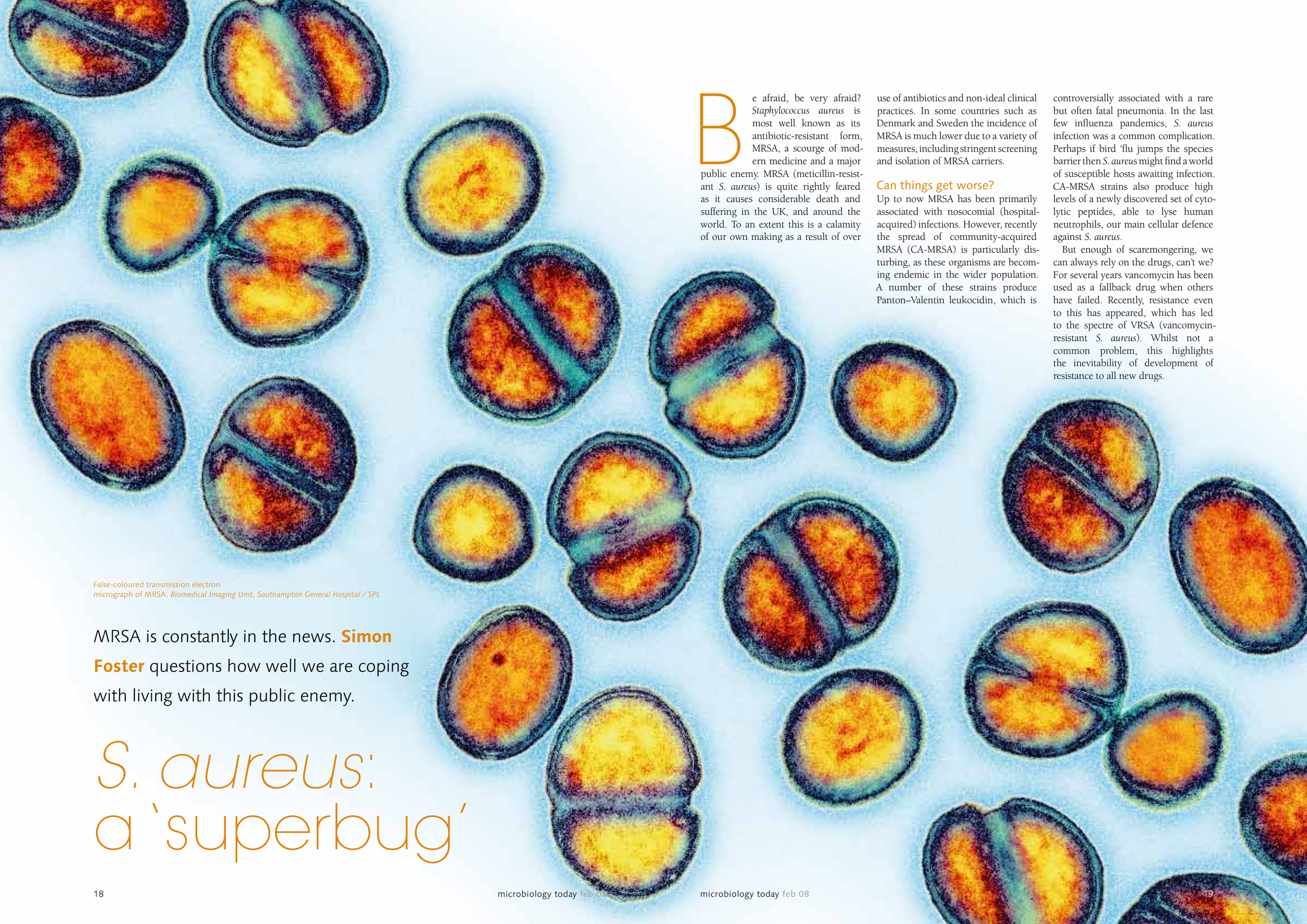


False-coloured transmission electron micrograph of MRSA. *Biomedical Imaging Unit, Southampton General Hospital / SPL*

MRSA is constantly in the news. **Simon Foster** questions how well we are coping with living with this public enemy.

S. aureus: a 'superbug'

Be afraid, be very afraid? *Staphylococcus aureus* is most well known as its antibiotic-resistant form, MRSA, a scourge of modern medicine and a major public enemy. MRSA (meticillin-resistant *S. aureus*) is quite rightly feared as it causes considerable death and suffering in the UK, and around the world. To an extent this is a calamity of our own making as a result of over use of antibiotics and non-ideal clinical practices. In some countries such as Denmark and Sweden the incidence of MRSA is much lower due to a variety of measures, including stringent screening and isolation of MRSA carriers.

Can things get worse?

Up to now MRSA has been primarily associated with nosocomial (hospital-acquired) infections. However, recently the spread of community-acquired MRSA (CA-MRSA) is particularly disturbing, as these organisms are becoming endemic in the wider population. A number of these strains produce Panton–Valentin leukocidin, which is controversially associated with a rare but often fatal pneumonia. In the last few influenza pandemics, *S. aureus* infection was a common complication. Perhaps if bird 'flu jumps the species barrier then *S. aureus* might find a world of susceptible hosts awaiting infection. CA-MRSA strains also produce high levels of a newly discovered set of cytolytic peptides, able to lyse human neutrophils, our main cellular defence against *S. aureus*.

But enough of scaremongering, we can always rely on the drugs, can't we? For several years vancomycin has been used as a fallback drug when others have failed. Recently, resistance even to this has appeared, which has led to the spectre of VRSA (vancomycin-resistant *S. aureus*). Whilst not a common problem, this highlights the inevitability of development of resistance to all new drugs.

It can't be that bad?

It is all too easy to fear and loathe *S. aureus* and with such antipathy, to gloss over the special relationship which has evolved between us and one of our most faithful microbes. We all have a high titre of circulating antibodies against *S. aureus* and so we must be challenged subclinically on a regular basis. Getting a serious *S. aureus* infection is actually remarkably difficult and mostly requires immense effort on our part via injury, surgery, indwelling medical devices, etc. *S. aureus* is an opportunist pathogen for which many of the diseases it causes are distinctly inopportune for the bacterium. Endocarditis and other deep-seated infections give little chance for reintroduction into the environment. Superficial and minor skin lesions are the primary infections caused by *S. aureus* and the flow of golden pus gives relief to the host and the prospect of dispersal to the pathogen.

That is not to say that the interaction between *S. aureus* and the human host during infection is not exquisite and highly evolved. *S. aureus* has a myriad of surface and secreted components able to react in the most intricate ways with almost every facet of the human immune and other bodily systems. The organism is also extraordinarily adaptable in being able to cause a wide range of different infections. *S. aureus* has a large arsenal of virulence determinants, including toxins, enzymes and adhesins. Apart from a very few specific syndromes (such as toxic shock) it is impossible to label individual components as primary virulence determinants. It is more the skilful wielding and interplay of a variety of determinants drawn from its repertoire that allows *S. aureus* to succeed so well across different infections. This in itself requires extraordinary powers of regulation in response to the host environment. Many regulators have been identified but how these interact, particularly in response to the host environment has remained largely elusive.

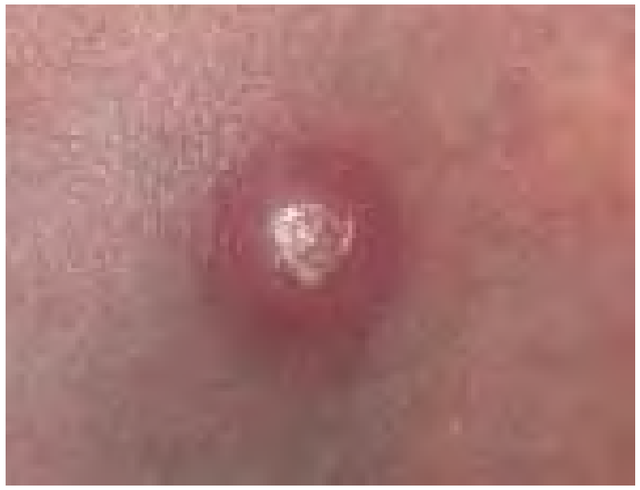
Living in harmony?

The primary niche for *S. aureus* is as a commensal living in our noses, on our skin and in the nasopharynx. In fact 20 % of the human population are permanent carriers and 60 % are transient carriers, which attests to the highly successful nature of the bacterium. Some people actually carry the same strain of *S. aureus* for years and such a long-term commitment to a relationship is laudable. Carriage cannot be looked upon, however, as an easy life for *S. aureus* as it not only has to survive and proliferate in a rather harsh physical environment, but also resist human innate defences and compete with other microflora. Understanding the basis for carriage is beginning to not only define the roles for several *S. aureus* components, but also give us clues as to the host environment. Surface components such as wall teichoic acids and the proteins ClfB and IsdA are required for nasal carriage and act as adhesins to nasal squamous cells. ClfB and IsdA, like many other *S. aureus* surface proteins bind to more than one human ligand and so their true *in vivo* target cannot as yet be defined (if a single target actually exists). The *isdA* gene is only expressed under conditions of iron deprivation and so the nose must be iron-limited.

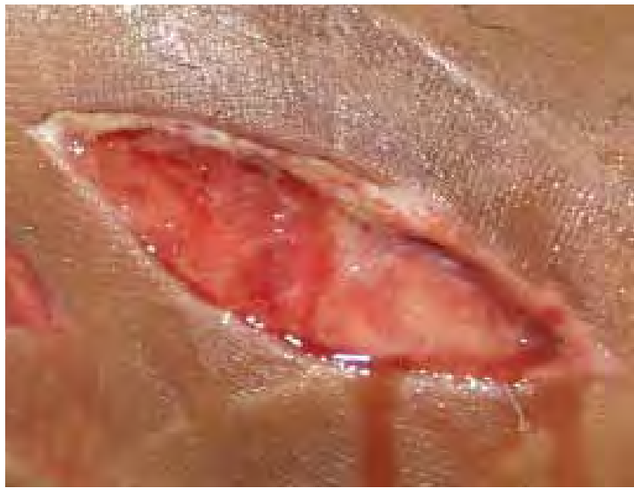
We also control *S. aureus* on our surfaces via the production of bactericidal fatty acids and peptides. IsdA is required for resistance to these factors and survival on live human skin. It is interesting that patients with atopic dermatitis often have altered fatty acid metabolism resulting in reduced levels of anti-staphylococcal fatty acids and this correlates with enhanced *S. aureus* colonization. In fact, application of anti-staphylococcal fatty acids results in reduced colonization in these patients. Thus by harnessing our own defence mechanisms we may reduce the prevalence of *S. aureus* carriage. The interaction of *S. aureus* and our skin fatty acids is even more intense, as at sub-growth inhibitory concentrations the fatty acids are able to inhibit virulence determinant production and so potentially allow survival of the organism but render it benign.

The nose and skin are also inhabited by a range of other flora (see the article by Farrar & Bojar on p. 14) and it is interesting to note that nasal carriage by *Staphylococcus epidermidis* or corynebacteria does not coincide with *S. aureus*, suggesting that our resident flora is battling it out for the prize of a human host. Thus *S. aureus*, whilst it is a formidable foe, can be beaten by other lowly microbes.

> *Although* S. aureus *can be a killer, one cannot but admire the tenacity and versatility of one of the most intimate cohabitees of the human body.*



▶ *S. aureus* abscess. *Emma Nickerson, Mahidol University*



▶ Open incision to drain pus from a thigh in a case of *S. aureus* pyomyositis. *Emma Nickerson, Mahidol University*

Man against microbe

The golden age of antibiotic discovery is over and the inevitable rise in resistance levels does not bode well for the future. *S. aureus* soon acquires resistance to new drugs, including linezolid and daptomycin. However, antibiotics will always remain a key tool in our armoury against *S. aureus* and it is essential that industry is encouraged to keep new compounds flowing into the clinic. Alternative approaches such as vaccines and therapeutic/prophylactic antibody development have continued apace, but all Phase III trials so far have ended in failure. MRSA is high on the UK political agenda and the favoured approach for control rests on cleaner hospitals. In truth, there is no one measure that will remove the MRSA problem. It is by a concerted and integrated package of approaches for the present, and the future, that will at least alleviate some of the burden. *S. aureus* cannot be eradicated from the human environment and it does not seek to eradicate us. By understanding more of the basis of our interaction with *S. aureus* we are beginning to discover possible breakpoints to reduce the incidence of infections and also to treat those once established. Although *S. aureus* can be a killer, with associated suffering, one cannot but admire the tenacity and versatility of one of the most intimate cohabitees of the human body.

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Further reading

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Microbial life in the mouth

The variety of surfaces in the oral cavity offers a home to a whole range of microbial communities, as **Dave Spratt** describes.

◀ A healthy human mouth. *BananaStock / JupiterImages*

The oral cavity forms the top section of the gastrointestinal tract and provides a large number of diverse surfaces on which a wide variety of complex biofilms is able to form. These surfaces include soft shedding tissues of the buccal mucosa, papillae and crypts of the tongue and hard non-shedding surfaces of the teeth. Dental plaque is the term commonly used for the biofilm formed on teeth; however, the term plaque has now been extended to encompass biofilms on all the oral surfaces. These biofilms consist of a complex microbial community embedded in a matrix of polymers of bacterial and salivary origin.

More than 700 bacterial taxa have been recorded in the oral cavity (although only 100–200 occur in any particular mouth) and this rich and diverse flora contains bacterial species principally found only in this habitat. The soft tissues in the oral cavity lack significant plaque accumulation due to the rapid rates of epithelial cell turnover, the exception being the dorsum of the tongue which is associated with a significant and characteristic microbiota. The hard non-shedding surfaces of the oral cavity, i.e. teeth; provide a far more stable substratum for the colonization of bacteria.

Acquiring the oral microbiota

Colonization of the oral cavity begins at birth. Neonates are usually sterile, despite encounters with the maternal resident microbiota during birth. The acquisition of the oral microbiota is via passive transmission from a variety of sources including food, milk, water and particularly saliva from the mother. However, the majority of these bacteria are present only transiently and only a limited range actually colonize. Streptococci predominate the primary colonization, especially *Streptococcus salivarius* and *S. mitis*. The richness and diversity of the microbiota increase rapidly with age and by the time the infant has teeth (6–18 months) numerous species are present. The microbiota becomes more complex during puberty (12–16 years) and notable increases are observed in Gram-negative anaerobes and spirochaetes. The presence of sex hormones in the gingival crevicular fluid (serum-like exudate that bathes the tooth/gum interface) is thought to drive this. Few changes have been observed with further increases in age, except to note that loss of teeth and therefore habitat will influence the microbiota.

Dental plaque

In a healthy mouth the only non-shedding surface available for colonization is enamel (a hard, highly calcified tissue) of the tooth surface. This surface is covered with a conditioning film or pellicle, derived from the saliva, within seconds of cleaning and it is this surface which is rapidly colonized by the bacteria in saliva (up to 10^8 per ml). The colonization can be split into two broad processes, the initial attachment of bacteria to the pellicle and the secondary attachment of other cells to those already present. The process is far from random and cell-to-cell recognition of genetically distinct partner cell types plays an important role in development of micro-colonies and subsequent biofilm architecture. Co-aggregation interactions are thought to contribute or perhaps drive plaque development. Early plaque accumulation is facilitated by intrageneric co-aggregation among *Streptococcus* and *Actinomyces* species, as well as intergeneric

co-aggregation between these species. The partnerships between dental plaque bacteria are highly specific and lead to complex biofilms. These processes are likely to benefit the individuals involved and may have nutritional or protective roles. The plaque is therefore rich and diverse and contains numerous microenvironments with a variety of gradients present, including nutrients, oxygen, redox potential and pH. The plaque is not only heterogeneous in nature but differs depending on location on the tooth surface and indeed with time. Mature plaque is therefore an extremely complex and highly dynamic community.

Unknown quantities

It has been estimated that only about 50 % of the oral microbiota can be cultivated. While this is a high percentage compared to some other microbial systems, it still means that we have very little knowledge about half of the oral microbiota. Culture independent techniques have clearly made inroads in determining the richness of this proportion (mainly via comparative 16S rRNA gene sequencing) of the microbiota. However, it has only been very recently that techniques have allowed the metagenome and functions of the whole community to be studied.

Oral infections

Small alterations in an environment can lead to ecological shifts and subsequent population changes, and in certain specific cases this may predispose to a more 'pathogenic' microbial community. This concept is termed the 'ecological plaque hypothesis' and can be used to understand the microbial aspects of a range of oral infections.

Dental caries and periodontal disease are some of the most prevalent infectious diseases of humans and are due to the accumulation of dental plaque on the tooth surface and at the tooth gum interface respectively.

Dental caries. This is the localized demineralization of the tooth tissue by various acids produced by bacterial fermentation of dietary carbohydrates and is arguably the most common, chronic infectious disease in humans. Approximately 90 % of all dentate adults in the UK have at least one restored tooth as a result of caries with a mean frequency of seven per person. Caries can be simply and conveniently split into two categories: coronal (crown) caries and root-surface caries. Coronal caries can occur on all surfaces of the crown where the plaque biofilm is allowed to develop and mature. Demineralization occurs due to a shift in the microbiota brought about by an increase in the amount and frequency of dietary fermentable carbohydrates. The increase in acids, such as lactic acid, reduce the pH to a level which only favours the growth of acid-loving microbes such as *Streptococcus mutans*, which is additionally highly acidogenic. Root surface caries, as the name implies, occurs on root cementum or dentine and is secondary to recession of the gums. This is clinically and microbiologically distinct from crown caries. The disease has been shown to have a definite progression, each with its own characteristic microbiology. In brief, initially the lesion is described as 'soft' and consists of a highly demineralized tissue replete with bacteria (increased numbers of lactobacilli and Gram-positive pleomorphic rods, *Actinomyces israelii* and *A. gerencseriae*, but fewer streptococci). The progression of the lesion leads to a change in appearance and is categorized as 'leathery', consisting of a re-mineralized surface overlaying a heterogeneous mix of bacteria, de-mineralized tissue and re-mineralized tissue. A further progression is to a 'hard' lesion which is fully re-mineralized and inactive with respect to caries (reduced numbers of *S. mutans*).

Root canal infections. Structures present in the mouth not normally exposed to the microbiota are usually sterile, for example, the endodontium – the pulp and root canal system within teeth. The root canals of teeth are complex systems of interconnecting channels containing the blood vessels and nerve tissue leading from the tooth apex to the pulp chamber. Endodontic infections are therefore defined as infections of the pulp and periapical tissues. Bacteria and bacterial products can gain access to the pulp chamber, often as a consequence of caries (demineralization of enamel and dentine). The resulting inflammation will lead to pulpal necrosis and progress to resorption of bone supporting the tooth, finally leading to tooth loss. The bacteria associated with individual lesions are surprisingly limited given the number of taxa potentially able to colonize and the large number of taxa associated with periodontal lesions. This reduced diversity implies special selective pressures operating within the root-canal system. Root-canal infections are invariably polymicrobial in nature and typically 4–12 bacterial isolates can be cultured. This microbiota is often diverse with respect to growth atmosphere, nutritional needs and virulence determinants and may be regarded as an 'infection team'. For example, primary colonization and adherence to dentine is carried out by streptococci which additionally utilize oxygen, thus making the environment more anaerobic and therefore suitable for the colonization and growth of strict anaerobes. Commonly, isolates from infected root canals include streptococci, *Actinomyces* spp., *Prevotella* spp., *Peptostreptococcus* spp. and *Fusobacterium nucleatum*.

Periodontal diseases. This broad group of diseases affects the periodontal tissues (gums and supporting bone). The most common of the periodontal diseases is gingivitis; this is usually brought about by poor oral hygiene. The microbiota and their extracellular products present at the gum margin cause a reversible (with good oral hygiene) non-specific inflammation of the gums. The plaque microbiota shifts from a streptococci-dominated community to one dominated by *Actinomyces* species.

Periodontitis refers to a group of more advanced and related diseases defined as 'an apical extension of gingival inflammation to involve the tissues supporting the tooth (periodontal ligament and bone)' and results in a periodontal pocket. By far the most common is chronic periodontitis which is the major cause of tooth loss in adults. The microbiota present in the periodontal pocket is extremely diverse, with up to 100 culturable species from a single pocket. Since such a rich microbiota is present, trying to identify the particular species responsible for disease initiation and progression is a very complex and difficult undertaking (especially as probably only half of the taxa are culturable). However, the World Workshop on Clinical Periodontology has designated three species as aetiologic agents of periodontitis in a susceptible host: *Aggregatibacter* (formerly *Actinobacillus*) *actinomycetemcomitans*, *Porphyromonas gingivalis* and *Tannerella forsythia* (formerly *Bacteroides forsythus*). Numerous other types of periodontitis exist and the microbiology of each is thought to be different.

The oral microbiota is also responsible for a number of other oral problems including oral malodour, thrush, angular cheilitis, denture-associated erythematous candidosis, *Candida* leukoplakia and median rhomboid glossitis.

In summary

Life on us and especially our oral microbiota is complex, dynamic, rich and diverse. Changes in the community structure brought about by environmental alterations cause a range of diseases, the ecology and pathology of which remain, largely, unknown.

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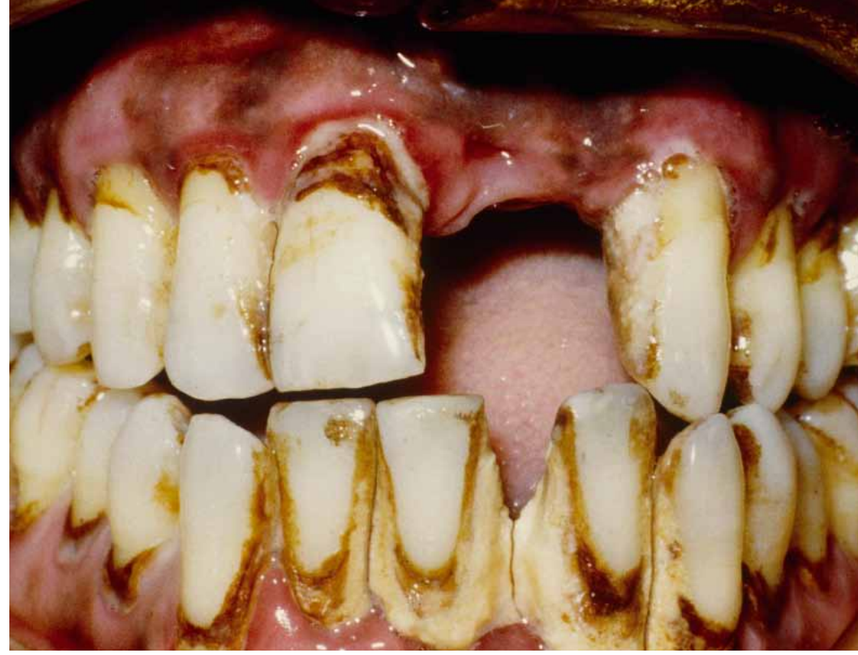


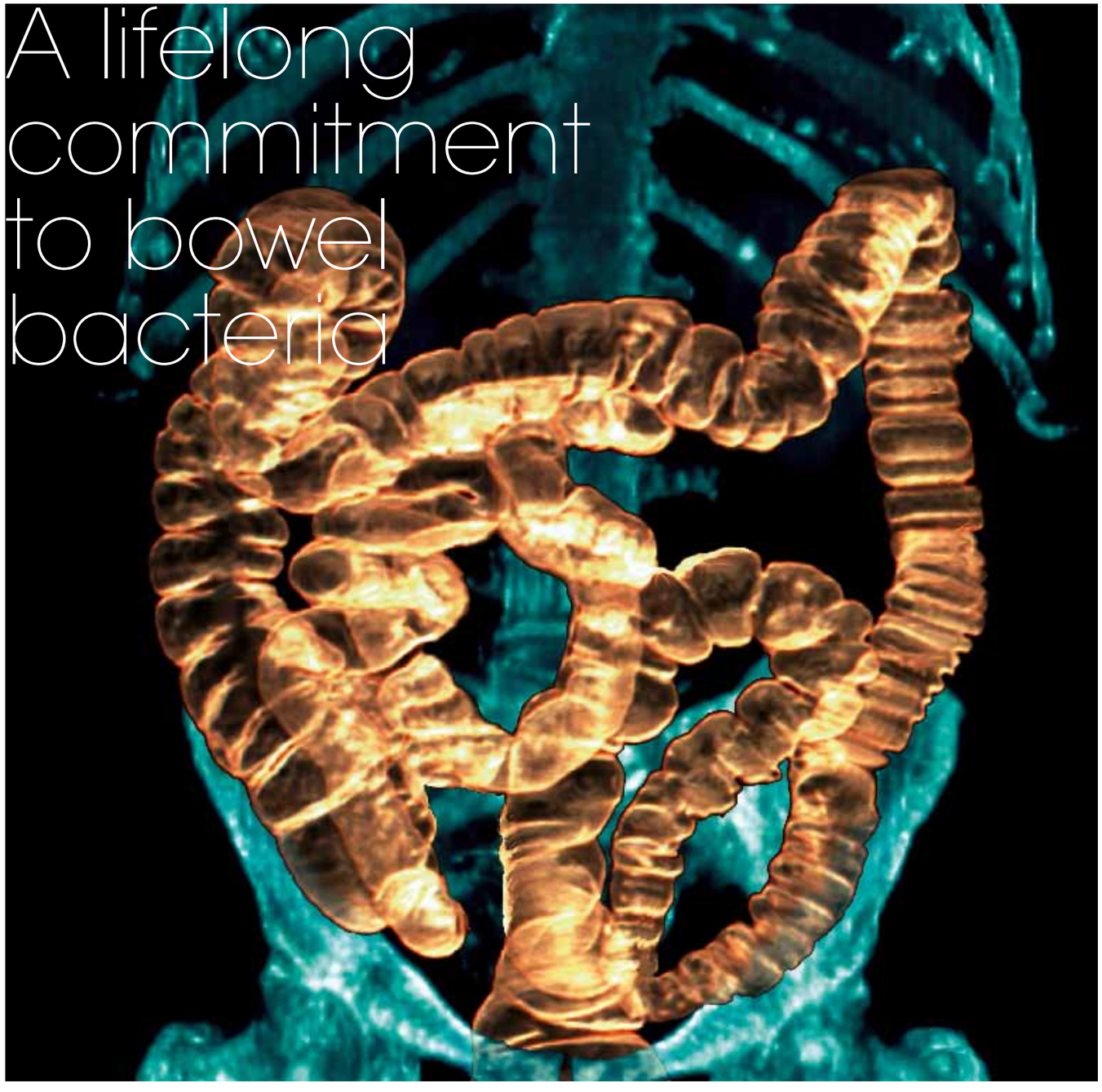
▲ Top. Mouth of a person reflecting a history of poor oral care: the gums are inflamed (gingivitis) and the lower incisor teeth are surrounded by deposits of tartar. *Science Photo Library*

▲ Lower left. False-coloured scanning electron micrograph of a cavity (lower centre) in a human incisor. Cavities are caused by dental plaque (brown), a film of bacteria embedded in a glycoprotein matrix. *Steve Gschmeissner / Science Photo Library*

▲ Lower right. False-coloured scanning electron micrograph of *Streptococcus mutans* bacteria (pink) in dental plaque. *Manfred Kage, Peter Arnold Inc. / Science Photo Library*

There are about as many bacterial cells in our bowel as there are human cells in our body. **Gerald Tannock** takes a look at the amazing community of bacteria that helps to set the very young on a fit and healthy life.

A lifelong commitment to bowel bacteria



► A false-coloured 3D CT scan of the abdomen of a 53-year-old patient, showing healthy intestines. *Zephyr / Science Photo Library*

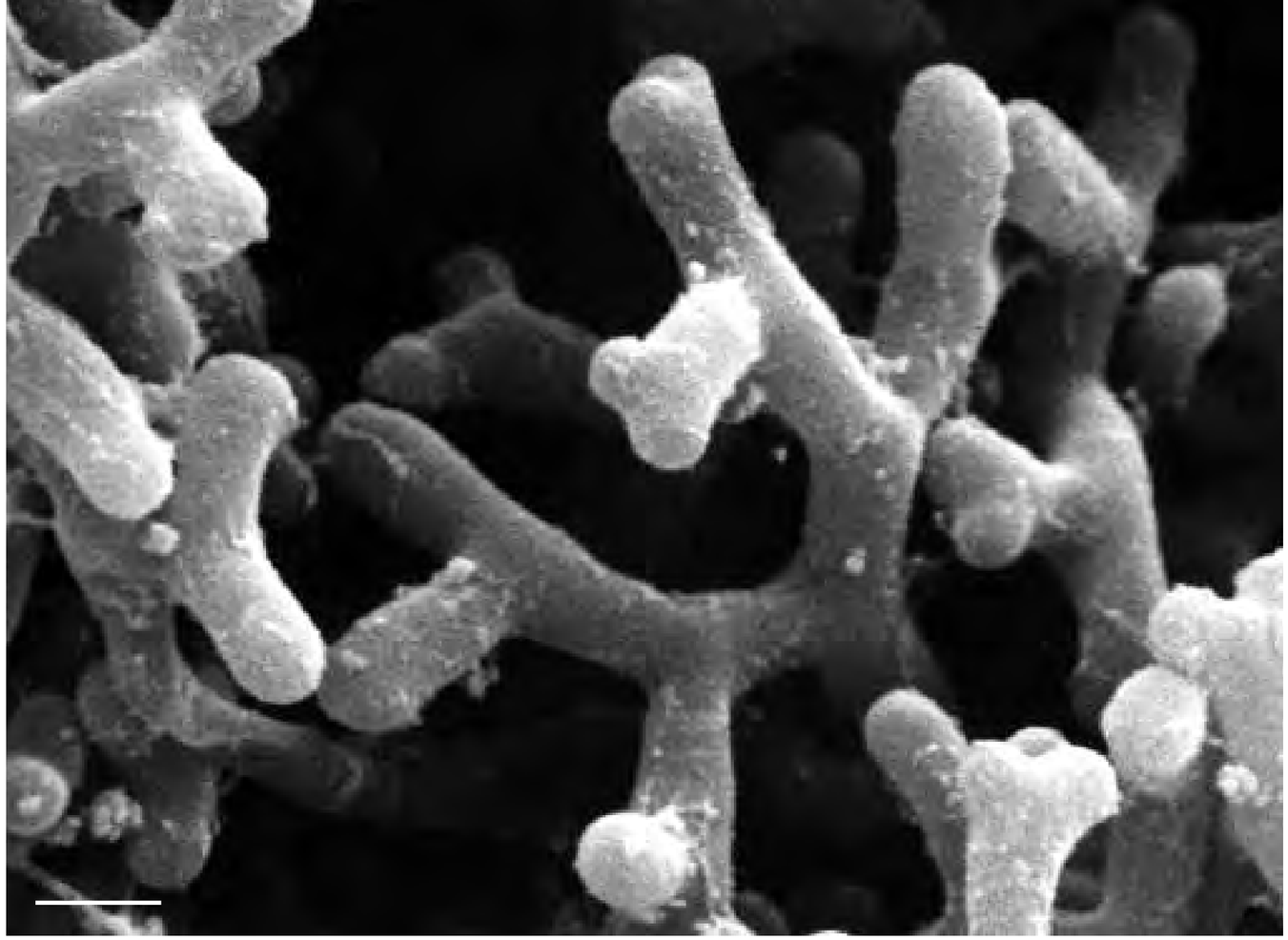
I have finally cum to the konklusion that a good reliable set ov bowels iz worth more to a man than enny quantity of brains.

Josh Billings (Henry Wheeler Shaw, American humorist, 1818–1885)

One can empathize, if not completely concur, with the sentiment expressed in the quotation attributed to the fictional, poorly educated, but commonsensical character Josh Billings. Constipation and diarrhoea are both unpleasant conditions reflecting bowel dysfunction, the latter often due to the activities of pathogenic microbes. Little appreciated, however, are the legions of bacteria that normally reside in the large bowel of humans and which constantly toil to digest complex molecules derived from the diet (such as fibre) and alimentary secretions (such as mucins in mucus). In doing so, the bacteria produce short-chain fatty acids, gases, indoles, phenols and amines as fermentation products. The bowel bacteria, sometimes referred to as commensals because they share the banqueting table that we lay in the large bowel, form a complex community about which relatively little is yet known. Hundreds of bacterial species are probably able to inhabit the human bowel, but not more that 50 % of the bacterial cells, based on comparisons of microscopic and plate counts, have been cultivated so far in the laboratory. Much of our knowledge of the taxonomy of these bacteria has been gained by the use of nucleic acid-based analytical methods, most of which target 16S rRNA gene sequences that form a cornerstone of bacterial phylogeny. From the application of these methods, which are culture-independent, we know that four bacterial phyla dominate the bowel community (*Firmicutes*, *Bacteroidetes*, *Actinobacteria*, *Proteobacteria*) and that there are about 10,000-fold more bacterial cells in the average large bowel than there are human beings on Earth.

Early days

Our lifelong commitment to feeding this large bacterial mass starts soon after birth when the bowel is apparently adventitiously inoculated with bacteria of environmental and maternal origins. Soon, a regulated and predictable succession of bacterial groups occurs in the bowel. Alone among mammalian species, the bowel community of human infants is dominated by *Actinobacteria* belonging to the genus *Bifidobacterium* which, by the time the baby is aged 3–6 months, comprise, on average, at least 40 % of the total bacterial community of stool, regardless of alimentation. These bacteria are especially endowed with the ability to detect and hydrolyse oligosaccharides, such as those found in human milk, using tightly regulated catabolic pathways.



◀ Bifidobacterial cells viewed by scanning electron microscopy. Bar 1 µm. *G. Tannock*

Thus, during the first few months of life, while immunological germinal foci develop in the bowel mucosa, the bowel of infants is colonized by large numbers of bifidobacteria.

Benefits for the bowel mucosa

Commensals interact with the bowel mucosa, at least in experimental animals, and in so doing influence the expression of mammalian genes. The up- or down-regulation of gene transcription as a result of bacterial exposure may be transient, but the succession of bacterial types to which the infant bowel mucosa is exposed could, in some instances, have long-lasting physiological, including immunological, ramifications as a result of molecular communications between bacterial and human cells. This conditioning of the bowel mucosa during infancy, which may have lifelong consequences, is integral to the concept of 'biological Freudianism': the biological (bacteriological) past is alive in the physiological present.

Impact on allergies

Allergies (eczema, hayfever, asthma) are more common today among the inhabitants of affluent countries than they were 60 years ago, yet remain uncommon in poorer nations. What has changed in affluent countries that could be responsible for this phenomenon? Doubtless there are thousands of possible correlations, including red herrings such as an increase in the number of telephones per head of population, but there may be bacteriological possibilities worthy of investigation. The kinds of bifidobacteria that babies are exposed to now may differ compared to 60 years ago. This might be because of better hygiene, different obstetrical practices and the relatively common use of antibiotics in modern paediatrics. We cannot investigate this possibility because we do not have bowel samples collected from infants born long in the past. Even if we did, the accuracy of the assay results might be compromised by the storage of the samples over decades. Comparisons of the bifidobacterial species present in stool collected from infants born in countries with low or high prevalence of allergies, however, have revealed differences. The exposure of infants to different kinds of bacteria in different geographical regions of the World could be influenced by ethnicity and maternal diet, as well as the factors mentioned above. The bifidobacterial species detected in the stool of allergic babies compared to healthy infants has also been reported to differ. The varying compositions of bowel communities in human populations are meaningless, however, unless functional links to disease or health can be made. Fortunately, preliminary observations point to a differential response of immune cells to bifidobacterial species residing in the bowel of infants, opening extensive opportunities for the investigation of bifidobacterial–eukaryotic cell cross-talk in relation to health and disease.

Bowel commensals and health

Medical science continues to be more concerned with defining the abnormal in relation to the normal in order to discover the means to heal. Thus medical knowledge focuses on the pathogenesis of diseases and the derivation of intervention strategies. These are noble causes, but while we know much about diseases, one wonders whether we really understand 'health'. Defining the functional roles of bifidobacterial species in early life might be illuminating in the contexts of both disease and health.

Francis Xavier believed that a Jesuit education until the age of 7 prepared a child to live a useful Christian life in no matter what circumstances they later ended up in. Modern educationists also understand that there are optimal periods during childhood in which the ability to process certain information (visual, sound, numbers, language) can be acquired. While the commitment to maintaining a bowel community is lifelong, critical beneficial interactions with commensals may occur during early childhood. Later, the presence of bowel bacteria may be incidental or, with increasing age and the development of chronic diseases, even malign.

Whatever your quantity of brains, you may find that the bowel community, together with its activities, an exciting and important field of research. The study of bowel commensals should concentrate on community function and interactive mechanisms, avoiding subversion by the wiles of high throughput sequencers and the creation of phylogenetic catalogues. Note could be taken of the advice given by the naturalist and putative 'father of ecology', whose comments concerning botany are equally relevant to modern microbial ecology in general, and the bacteriology of the bowel in particular.

The standing objection to botany (microbial ecology) has always been, that it is a pursuit that amuses the fancy and exercises the memory, without inspiring the mind, or advancing any real knowledge; and, where the science is carried no further than a mere systematic classification, the charge is but too true … Not that system is by any means to be thrown aside – without system the field of Nature would be a pathless wilderness – but system should be subservient to, not the object of, pursuit.

Gilbert White, curate of Selborne, 1778

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Human endogenous retroviruses: from ancestral pathogens to bona fide genes

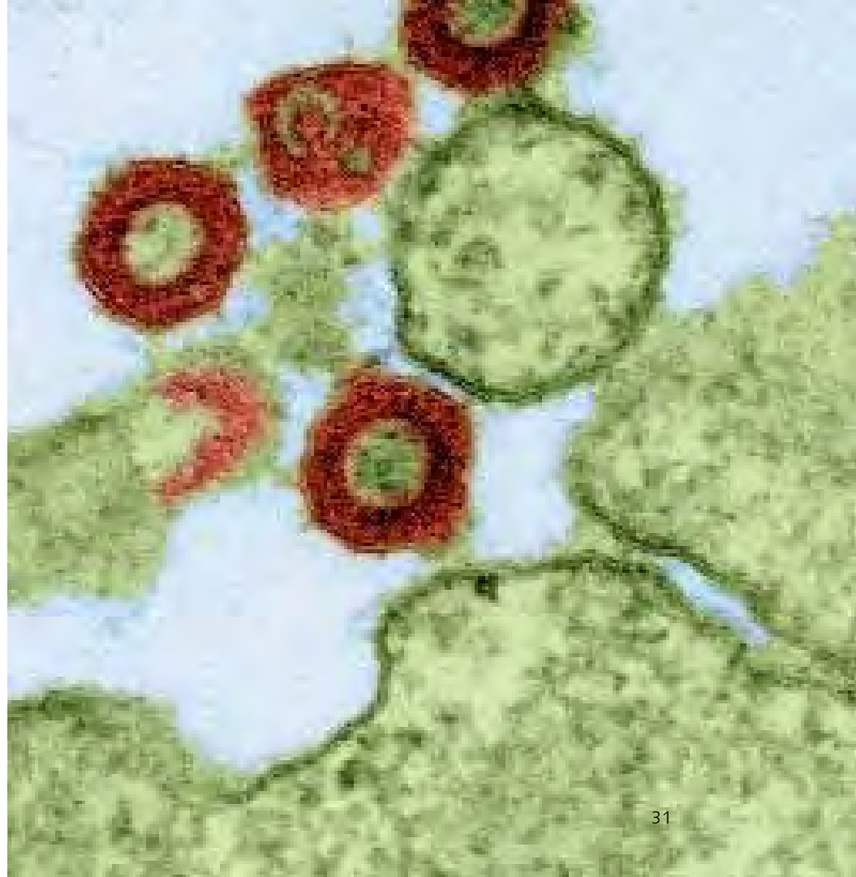
How many of our genes are actually human in origin? **David Griffiths** and **Cécile Voisset** explore the fascinating impact of endogenous retroviruses in and on our bodies.

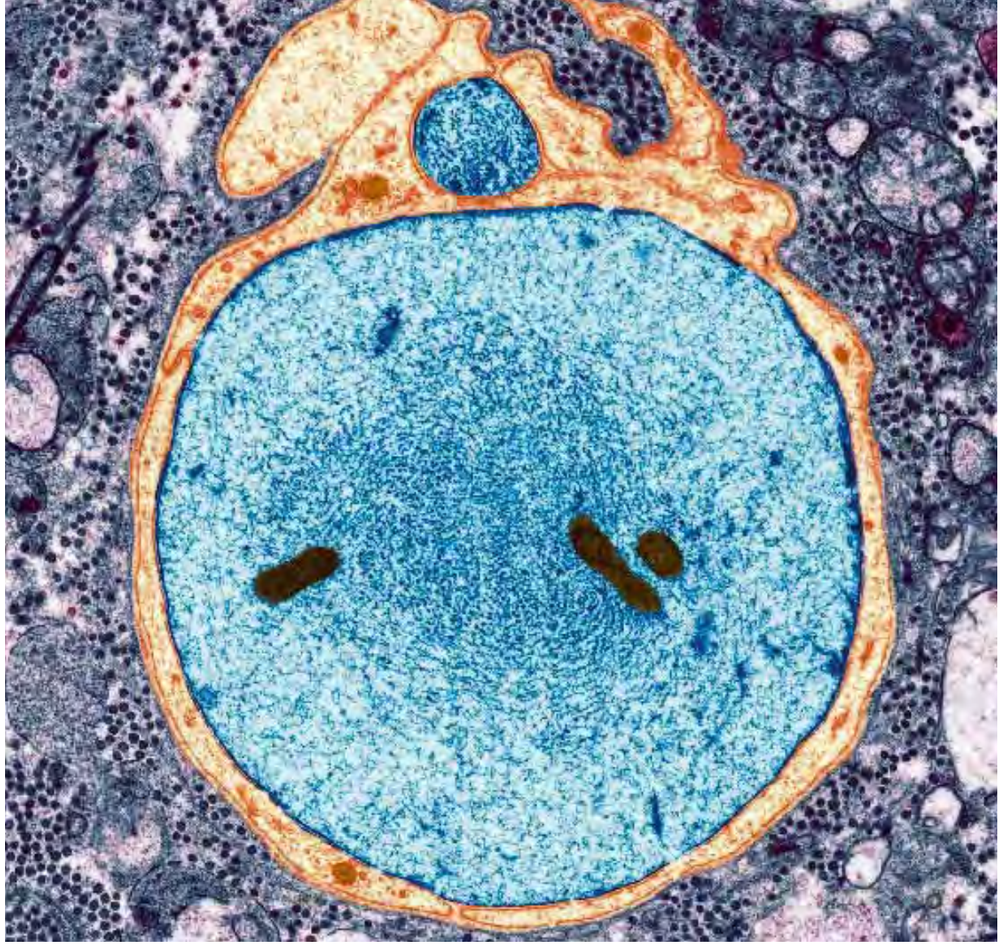
Retrovirus infection represents perhaps the most intimate of all host–pathogen relationships. The replication of these viruses requires the insertion of a DNA copy of their genome into the chromosomal DNA of the infected cell. This process, known as integration, is essentially irreversible and provides a means for stable, usually lifelong, infection of the host. Moreover, if a retrovirus infects a sperm or egg cell, or their germ cell precursors, its genetic material can become permanently fixed in the germ-line DNA of any resulting offspring. In this way, the retrovirus can persist in the host and its descendents for millions of years. Such inserted sequences are known as endogenous retroviruses (ERV) and they are replicated and inherited in a Mendelian fashion along with all other nuclear DNA.

Over the course of evolution, retroviruses have invaded the germ-line of our ancestors on numerous occasions such that human ERVs (HERVs) now comprise ~8 % of our genome. These can be divided into around 30 different families, each representing a different ancestral infection event. The timing of their introduction into the genome ranges from over 30 million years ago up to less than 1 million years ago, depending on the family. Since HERVs represent ancient infections, they are not closely related to retroviruses currently circulating in humans, such as HIV. Instead, they have greater sequence similarity with ERVs of animals.

◀ Common Gibbon (*Hylobates lar*). Many HERV integrations are also present in the same genomic locus in other primates, a feature which has been used to date the time of germ-line infection. HERV-W is common to Old World primates (e.g. chimpanzees, gibbons and African green monkeys) but is absent from New World primates (e.g. squirrel monkey) and is therefore thought to have entered the germ-line around 25–40 million years ago, the divergence time estimated from fossil records. *Art Wolfe / Science Photo Library*

▼ False-coloured TEM of an ultrathin section showing a group of HERV-K particles budding from a cultured human teratocarcinoma cell. HERV-K is the only HERV family known to encode assembled virions. These non-infectious particles are well characterized in cell lines derived from testicular tumours. Some recent studies have suggested that HERV-K has been capable of re-infecting the human germ-line relatively recently (<100,000 years), raising the question of whether a relative of this virus stills circulates as an infectious virus today. *Klaus Boller, Paul-Ehrlich-Institut, Langen, Germany*





◀ Coloured TEM of a section through a demyelinated nerve in multiple sclerosis. The axon (blue) has only its Schwann cell (brown) surrounding it. The Schwann cell would normally produce the myelin sheath. A nerve's myelin sheath helps it conduct electrical impulses and when the myelin sheath is lost, nerve function is impaired. *Steve Gschmeissner / Science Photo Library*

Consequences of endogenization for the virus

Because retrovirus infection is frequently pathogenic, any new introduction of ERVs is likely to be highly detrimental for the host. Therefore, to become fixed in a genome, a newly endogenized retrovirus would probably be inactivated to prevent its expression, for example through mutation or truncation of genes encoding viral proteins or by cellular mechanisms such as the silencing of ERV gene expression by DNA methylation. In this way, endogenization of retroviruses leads ultimately to their inactivation and a reduction in pathogenicity. Contemporary HERV families therefore consist of numerous heterogeneous elements, ranging from full-length defective proviruses to isolated long terminal repeats (LTRs) derived from recombination events. (LTRs are regions of the retroviral genome containing gene promoter and enhancer elements.) However, inactivation of ERVs may take many generations and the immediate effect of germ-line integration can therefore be increased pathogenesis, which could potentially lead to the extinction of the host. In humans, the effects of HERV acquisition on our ancestors millions of years ago can only be a matter of speculation. However, an epidemic of neoplastic disease currently afflicting koalas in Australia represents a modern example of retrovirus endogenization in action and provides a rare opportunity to study this process and its effect on the host.

While the vast majority of HERVs are defective, many are nevertheless transcribed as RNA in various tissues and in a few instances HERV proteins or particles may be produced. The abundance of HERVs raises the question of what effect their presence has had on the evolution of our genome and whether they have any function today. These topics have proved to be rather controversial, but recent work has provided some tantalizing evidence supporting roles in normal human physiology and also in disease.

HERVs as bona fide human genes

Among the rare HERV-encoded proteins, syncytin-1 and syncytin-2 are particularly interesting examples of how new functional genes may be acquired from HERVs in the human genome. Syncytin-1 and syncytin-2 are glycoproteins encoded by the envelope genes of specific proviruses of the HERV-W and HERV-FRD families, respectively. Of note, the other viral genes in these proviruses are highly mutated, suggesting that the genes encoding these envelope proteins may have been positively selected by providing some benefit to humans. The key biological function of these two proteins is their ability to mediate the fusion of cellular membranes to produce multinucleate cells or syncytia. Importantly, it appears that this function has been co-opted by the host to serve an important physiological function.

Syncytin-1 is expressed predominantly in placental cytotrophoblast cells where it participates in their fusion to form the syncytiotrophoblast. This is a fused cell layer that forms the direct border between maternal blood and foetal tissues, and its formation and maintenance is crucial for a healthy pregnancy. Dysregulation of syncytin-1 expression could impair placental morphogenesis and has also been linked with pre-eclampsia. Syncytin-1 induces the formation of cell-to-cell fusion by interacting with its specific receptors, the amino acid transporters ASCT1 and ASCT2. In non-placental tissues, syncytin-1 expression is usually kept silent through promoter methylation because inadvertent cell fusion could be deleterious for tissue organization and integrity. Syncytin-2, which has been characterized only recently, is also expressed in cytotrophoblastic villous cells and may serve a similar function in placental development. The HERVs encoding the syncytin proteins are only present in primates; however, unrelated ERV envelope proteins in mice and sheep have also been implicated in facilitating placental cell fusion. Given the divergent placental structures between these species and humans, this is a startling example of parallel evolution where unrelated retroviruses have been co-opted to serve a conserved host function.

Are HERVs still pathogenic?

Although individual HERV proteins, such as syncytin-1 and 2, may serve a physiological role for the host, expression of HERVs has also frequently been linked with diseases, notably autoimmune diseases and cancer. However, due to the ubiquity and abundance of HERVs, it has proved difficult to confirm or refute a role for them in pathogenesis. Nevertheless, recent evidence has implicated syncytin-1 in the aetiology of multiple sclerosis (MS). MS is a chronic inflammatory disease of the central nervous system, characterized by the loss of the myelin sheath that surrounds and protects neurons. The pathogenic mechanisms involved in MS are still unclear, but inappropriate expression of syncytin-1 in astrocytes has been proposed to exacerbate the inflammatory events within the brain and spinal cord by inducing inflammatory mediators such as interleukin-1β (IL-1β) and reactive oxygen species. These factors are cytotoxic for oligodendrocytes, the cells involved in myelin formation. What triggers the expression of syncytin-1 in the astrocytes is unknown but it has been shown that cytokines detrimental to MS, such as tumour necrosis factor, interferon-γ, IL-6, and IL-1 can activate the syncytin-1 promoter, while the MS-protective cytokine interferon-β inhibits syncytin-1 expression. Why this should occur in some individuals and not others is also unclear but common herpes virus infections of the central nervous system have been proposed as a potential initiating factor.

Research on other HERV proteins has focussed on their potential role in cancer because a number of malignancies are accompanied by increased production of HERV RNA and proteins. Some members of the HERV-K family, which integrated into our genome relatively recently (~1–5 million years ago), can express non-infectious particles. These defective viruses are expressed in melanoma, testicular tumours and myeloproliferative diseases, but whether they are actively involved in triggering or promoting tumour development appears doubtful. It is perhaps more likely that they are up-regulated as a result of other genetic lesions in the tumour cell. An exception may be teratocarcinoma where a HERV-K protein called Rec has been directly implicated in activating transcription factors involved in cellular proliferation. As with syncytin-1 and MS, additional research is necessary to determine its importance in tumour development.

An infectious HERV?

In some species, such as pigs and chickens, a few ERVs have escaped deleterious mutation and remain capable of infectious transfer. In humans no such virus has yet been described. However, recent work by laboratories in France and the USA has reconstructed infectious forms of HERV-K that represent the consensus sequence of the most recently acquired HERV-K viruses. The original HERVs are defective for replication but the consensus sequences, in which the effects of individual mutations have been 'averaged out', are infectious. These viruses therefore provide important tools for further analysis of the function of this ancient virus and its potential role in disease.

The future for HERVs

HERVs have been with us and our ancestors for millions of years and it is only recently that their abundance has been recognized and their functional significance begun to be elucidated. Whatever their function today, at the time that they entered our germ line, their infectious counterparts may have been pathogenic viruses. As noted above, there are no HERVs closely related to the infectious retroviruses currently infecting humans, although ERVs related to lentiviruses have recently been described in rabbits. Could it be possible in the future that HIV will be tamed by endogenization?

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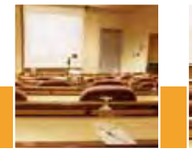
meetings



Spring**08** | Edinburgh International Conference Centre

31 March–3 April 2008 | 162nd Meeting

Plenary

Bacterial secretion systems: commonality and diversity

31 March–1 April 2008

Hot topic

Microbes and climate change

2 April

Programme Booklet

A booklet giving full details of the programme is enclosed with this issue of *Microbiology Today*. Any changes will be posted on the SGM website.

Registration

Registration is through the SGM website (www.sgm.ac.uk/meetings).

Registration fees per day (*incl. lunch, refreshments, abstracts book, conference literature, welcome reception*)

Earlybird (up to 29 February 2008)

Ordinary Members*	£45
Student/Associate Members*	£25
Non-members	£115
Retired/Honorary Members	Free

Full (after 29 February 2008)

Ordinary Members*	£55
Student/Associate Members*	£35
Non-members	£125
Retired/Honorary Members	£10

Please note: to qualify for earlybird rates, 2008 membership fees must be paid by the deadline of **29 February.*

Postgraduate Conference Grants

These will be available, subject to the usual conditions. See **www.sgm.ac.uk/grants/pg.cfm**

Offered poster presentations

Delegates whose offered posters have been accepted should note that an area of 90 cm x 90 cm only is available on the poster boards for their display.

Microscence Noticeboard

At the meeting, a board will be set up with notices of job, postdoctoral positions, studentships, courses, conferences, etc. Contributions are welcome and may be brought to the meeting or sent beforehand to Janet Hurst (j.hurst@sgm.ac.uk)

Special events

Communicating microbiology

31 March – *Workshop*

1 April – *Display*

Postdoc and beyond – planning for a research career

2 April

Are you a PhD student or first postdoc wondering how to plan for your future? At this event short talks on strategies to improve your chances of a career in research will be followed by Q&A, a buffet and wine. Entry is free, but by ticket only, so make sure you tick the box on the booking form. If applying for a PG conference grant, attending the workshop qualifies you for overnight accommodation on Wednesday.

Social events

Monday 31 March

Welcome Reception

Get to know your fellow delegates over a glass of wine on the first evening of the conference.

Ceilidh & Supper (*entry by ticket only*)

Tuesday 1 April

Society Dinner (*at the Hub*)

A 3-course meal with inclusive wine and pre-dinner drink.

Autumn**08** | Trinity College Dublin

8–11 September 2008 | 163rd Meeting

Plenary

Behaviour of biofilm bacteria: from cooperation and communication to control

8–9 September 2008

Organizers: G.M. Gadd, P.S. Handley, P.R. Langford, H.M. Lappin-Scott, M.M. Tunney, M. Upton & J. Verran

Speakers

Biofilm basics

J. Verran Manchester

H.M. Lappin-Scott Exeter

A. Mc Bain Manchester

M. Upton Manchester

A.H. Rickard USA

Bacterial biofilm structure and organization

P.B. Rainey New Zealand

P. Stoodley USA

K. Sauer USA

J. Webb Southampton

H.-C. Flemming Germany

T. Tolker-Nielson Denmark

Biofilm communication, resistance and control

S. Molin Denmark

M.R. Parsek USA

P. Kolenbrander USA

D.A. Spratt London

G. Seymour New Zealand

M. Givskov Denmark

P. Gilbert Manchester

Other symposia

Innate immunity systems

Cells & Cell Surfaces/Microbial Infection Groups

Organizers: I. Henderson & A. Cunningham

Biofilm infection of medical devices

Clinical Microbiology Group

Organizers: D. Mack & M. Tunney

Infective endocarditis

Clinical Microbiology Group

Organizers: S. Lang & D.R. Ready

Industrial bioremediation: from contamination to clean-up

Environmental Microbiology Group / Irish Branch

Organizers: R. Howarth, C. Whitby & E.M. Doyle

Packaging of nucleic acids

Eukaryotic Microbiology Group

Organizers: M.L. Ginger & S.K. Whitehall

Fuels and chemicals from renewable feedstocks

Fermentation & Bioprocessing Group

Organizer: G.M. Stephens

Bacterial adhesion within the food and beverages industry

Food & Beverages Group

Organizers: C.E. Rees & T.G. Aldsworth

Sealed membranes: the structural basis of transport and energetic processes

Physiology, Biochemistry and Molecular Genetics Group

Organizers: F. Sargent & G.M. Fraser

Contact details of organizers are included in the meeting programme on the SGM website. Deadline for receipt of titles and abstracts for offered presentations: **9 May 2008**.

A poster to promote the meeting is enclosed with this issue. Please display it in your department.

Irish**Branch**

Regulatory mechanisms in host–pathogen interactions

National University of Ireland, Galway – 27–28 March 2008

Abstract submission – 29 February

Organizer Conor O'Byrne (e conor.obyrne@nuigalway.ie)

For details of Irish Branch activities contact Evelyn Doyle (e evelyn.doyle@ucd.ie).

Other**Events**

Federation of Infection Societies Conference

Cardiff – 1–3 December 2008

e pjmccoy@bsac.org.uk

Microbes: then, now and hereafter

IUMS Istanbul

Meetings of the three divisions of the International Union of Microbiological Societies 2008

5–9 August

XII International Congress of Bacteriology and Applied Microbiology

XII International Congress of Mycology

10–15 August

VIV International Congress of Virology

Deadlines

Abstract submission: 31 January 2008

Early registration: 1 April 2008

www.iums2008.org

Molecular Biology of Archaea

St Andrews – 19–21 August 2008

www.biochemistry.org/meetings

Biochemical Society / SGM

Meetings on the web

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

Meetings organization

The SGM meetings programmes are organized by the committees of the special interest groups, co-ordinated by the Scientific Meetings Officer, **Professor Hilary Lappin-Scott**. Suggestions for topics for future symposia are always welcome. See p. 10 for contact details of Conveners.

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

Offered papers & posters

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

Offered posters

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

Abstracts

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

Abstracts Book

The full text of the abstracts book for the Federation of Infection Societies Conference 2007 is available as a PDF file at **www.sgm.ac.uk/meetings/past.cfm**

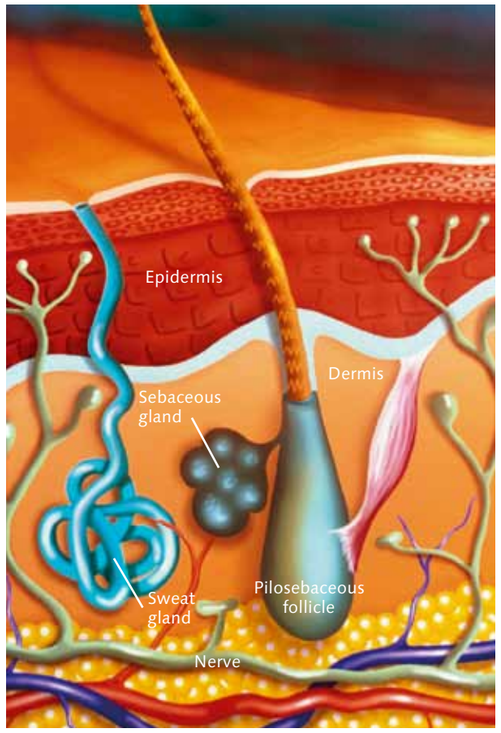
Schools Membership costs only £10 a year. Benefits include *Microbiology Today*, advance copies of new teaching resources and discounted fees on SGM INSET courses. To join see **www.sgm.ac.uk/membership**. Enquiries: **education@sgm.ac.uk** or go to **www.microbiologyonline.org.uk** for full details of resources and activities.

In recent months science teacher Gemma Sims has been working at the SGM to produce multimedia microbiology resources for the new Biology A-levels coming on stream in England and Wales this September. Most of her efforts have been devoted to explaining the microbial science that students will need to understand to follow the courses, but the resource also aims to have 'added value' and put microbes into the context of real life. Further information about the whole teaching pack will be available in the May issue of *Microbiology Today*.

The following article is a taster for a resource we are developing for Key Stage 4 and 5 Personal, Social Health and Economic Education (PSHE). 'What your mum might not know and probably hasn't told you' will deal with some of the health issues that affect teenagers.

Microbes and puberty: a teenager's guide

Being a teenager can be so horrible that many adults (especially teachers and parents) have wiped its ghastly memories from their minds. It is easy to feel lonely and isolated, but don't worry, you are not alone: there are over 10 times the numbers of microbes living in and on you than there are human cells in your body. You are home to a complex community of bugs such as bacteria, viruses, fungi and protozoa. They live in your gut, mouth, skin, vagina, upper respiratory tract and urethra, and each of us has our own unique collection. They help digestion, synthesize vitamins, boost immunity and occupy niches that would otherwise be filled by pathogens. Puberty is a time of change, both physically and emotionally, and this affects your microbes too…



◀ Artwork of a cross-secion through human skin. *BSIP Estiot / Science Photo Library*

The skin

Acne

The skin is home to 10^{12} bacterial cells, the most common being *Propionibacterium acnes*. Just before puberty your body starts to release sex hormones. One of their effects is to stimulate the production of an oily substance called sebum from sebaceous glands below the surface of the skin. In some people this can lead to the most common skin disease: acne. Adults may tease you about your zits, but acne is no laughing matter. 80 % of adolescents suffer from it and it can cause scarring and serious emotional distress. Inflammatory acne, with its unsightly pustules is by far the worse type.

But what causes it?

Three factors conspire: first, there is an over-production of sebum. Second, pilosebaceous follicles (each containing a sebaceous gland and a single hair) become blocked, often due to the over-proliferation of skin cells called keratinocytes. Sebum and bacteria become trapped in the follicle and the bacteria cause an inflammatory immune response, resulting in a spot.

We all produce sebum and have bacteria on our skin, so why are some people more prone to acne than others? The answer is not clear, but influential factors may include genetics (something you can blame your parents for), hormones (girls often find it is linked to their periods) and stress.

The good news is that acne is treatable and usually goes away in time (only 10 % of acne sufferers still have it after the age of 25). Ask your pharmacist about an effective over-the-counter cream called benzoyl peroxide and, if this does not help, your doctor can prescribe a variety of suitable medications.

Acne myths

- Acne is not caused by poor hygiene ('blackheads' contain the pigment melanin, not dirt).
- Acne is not linked to any specific foods, e.g. chocolate.
- Wearing makeup is OK (and is sometimes necessary for your self confidence), but make sure you use non-greasy ('non-comedonal') products and that you remove it fully.

To squeeze or not to squeeze?

If there is an obvious white 'head' that resembles an erupting volcano then a gentle squeeze is fine. But remember, your hands can be a source of infection (wash them first) and incorrect squeezing can push the sebum further into the follicles, exacerbating the inflammation and leaving a scar.

Body odour

During puberty hair starts to grow in your armpits and, as if this isn't bad enough, body odours start wafting around. BO is caused by volatile waste products released by bacteria such as *Corynebacterium jeikeium* that feed off the dead cells, sebum and sweat that stick to the skin and hairs. The problem can easily be alleviated by daily washing and the use of deodorants (which are often antibacterial).

Fungi

Fungi can also colonize the skin, generally causing no harm, but some species can cause infections such as 'athlete's foot'. This can cause itchy and cracked skin, but can easily be treated with an antifungal cream. The fungi are spread by direct contact and they particularly like the warm, moist, sweaty bits between your toes, so make sure you dry them thoroughly, wear clean socks and avoid sharing towels. The fungus *Malassezia globosa* lives on the scalp and can cause dandruff, but don't worry, an antifungal shampoo will soon clear it up. Ask your pharmacist for advice about suitable products.

The vagina

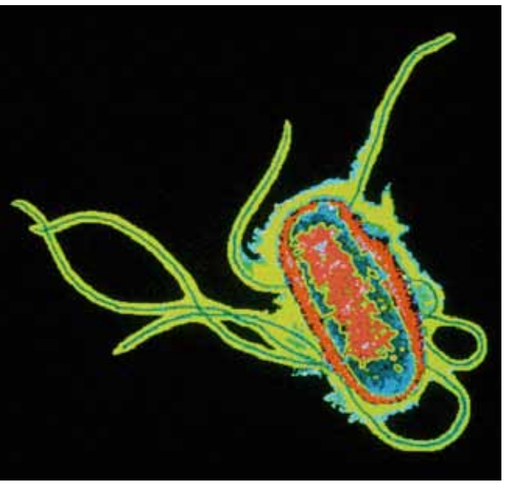
Before puberty the vagina is alkaline and contains bacteria such as enterococci and coliforms, but during puberty the environment of the vagina changes. Secretions of sticky mucus encourage the growth of the bacterium *Lactobacillus acidophilus* which feeds on glycogen and produces acidic waste. This results in a vaginal pH of 4 which deters other bugs, but if the natural flora of the vagina is disrupted (e.g. after taking antibiotics), less welcome visitors can thrive. Vaginal thrush, for example, is caused by the fungus *Candida albicans*. Symptoms include a burning or itching sensation and a

thick yellowy-white discharge. It is extremely common (75 % of women get it) and it can be easily treated by over-the-counter antifungal drugs.

The volume and consistency of mucus changes throughout the menstrual cycle, and each woman is different. If you notice secretions which are different for you, especially if they are accompanied by an unpleasant odour, itchiness or pain, then see your doctor immediately. These could be symptoms of thrush, bacterial vaginosis, a sexually transmitted infection or a forgotten tampon (yes, it does happen!).

The urinary tract

The urethra (the tube that carries urine from the bladder) contains bacteria such as staphylococci, enterococci and corynebacteria. Sometimes other bacteria invade, causing the condition cystitis (urethritis). Sorry girls, but you are more likely to suffer than males. Women have a shorter urethra and because its opening is close to the anus it is easily infected by faecal bacteria. To put it bluntly, the symptoms feel like you are weeing concentrated sulfuric acid while a herd of elephants tap dance on your bladder. You also need to urinate frequently (but not much comes out), your urine may be dark or bloody and you could have a fever and pain in your lower back or pelvis. The immune system usually clears the infection, but your pharmacist can give you medication to ease the symptoms and drinking cranberry juice really does work (there is evidence that it prevents bacteria sticking to the walls of the urethra). If it is your first attack of cystitis or if symptoms last more than 3 days, you must see your doctor. If not treated the infection could move up to the bladder or kidneys.



Preventing cystitis

Girls

- After a poo, wipe from front to back (to avoid spreading faecal bacteria);
- Don't let yourself become dehydrated – drink 2 litres of water per day;
- Avoid perfumed hygiene products that can irritate the urethra;
- Urinate after sex (as soon as is polite!) to 'flush out' any bacteria.

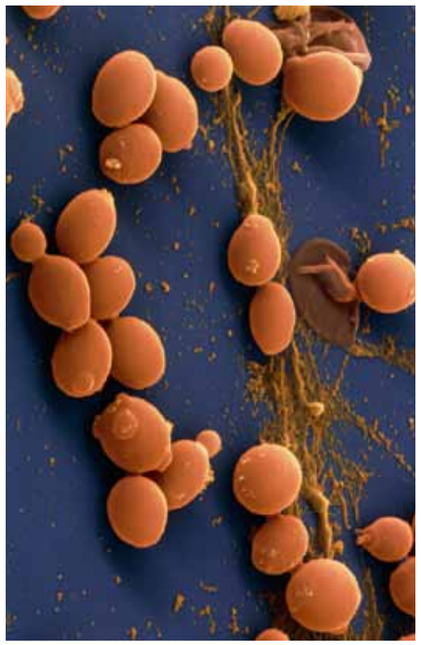
Boys

- You can get cystitis too, but it is less common. It could be linked to dehydration or a problem with the urinary system. Go to the doctor!

Conclusion

Despite the fact that your microbes can cause embarrassing and irritating conditions, most of the time their presence is beneficial. If you are concerned about any health matter, don't suffer in silence and hope it will go away: talk to someone! Your GP, pharmacist or school nurse will know what to do and the links on the right may be useful.

Gemma Sims
(e g.sims@sgm.ac.uk)



▲ The thrush fungus, *Candida albicans*. *Eye of Science / Science Photo Library*

◀ False-coloured transmission electron micrograph of *Escherichia coli*. *Dr Linda Stannard, UCT / Science Photo Library*

Taking it further

Eady, A. & Bojar , R. (2001). Spotting the onset of puberty – the secret's in the skin. *Microbiology Today* **28**, 178–181. (www.sgm.ac.uk/pubs/micro_today/pdf/110104.pdf)

Spinney, L. (2007). Bugs R Us. *New Scientist* **2617**, 34–38.

Websites

General health:
www.nhsdirect.nhs.uk
www.embarrassingproblems.co.uk

Acne:
www.stopspots.org

Cystitis and urinary problems:
www.cobfoundation.org

Sex and sexual health:
www.brook.org.uk
www.ruthinking.co.uk
www.mariestopes.org.uk

Summer in the city

To foster the interest of young people in science and to encourage their study of microbiology, 15 talented A-level students were invited to take part in a three-week Summer School at The University of Manchester. **Sue Crosthwaite** describes the event.

Students investigated the 'one gene–one enzyme' model organism *Neurospora crassa*, a key eukaryotic microbe that plays an important role in elucidating gene function. By monitoring and recording the growth, morphology and colouration of 100 *Neurospora* strains for which genes of unknown function are deleted, the students contributed to a community effort that aims to characterize phenotypically all available *Neurospora* knockout strains. Excitement grew when several of the strains displayed interesting and unexpected phenotypes. The students captured images of the colonies as well as high magnification pictures of the hyphae, which occasionally revealed stunning changes in morphology. All the data were uploaded to The BROAD Institute database, from where they now can be accessed worldwide.

The practical work was accompanied by lectures on various topics revolving around fungal biology. Dr Geoff Robson (Manchester) introduced the students to the use of microbes in biotechnology and showed some astonishing pictures of enormous fermenters in which *Aspergillus niger* is grown for the production of citric acid. This was followed by a memorable talk given by Dr Christian Heintzen (Manchester) centred around the use of model organisms in basic research, in particular the use of *Neurospora* for the study of circadian clocks. A highlight was a visit from Professor Nick Read (Edinburgh) whose presentation on *The Dynamic Hypha* included some stunning movies of modern molecular imaging techniques and the use of laser tweezers in the study of cell–cell communication.

Dr Jayne Brookman's *Bioscience for Business* talk on fungal disease, which incorporated some particularly unsavoury images of the symptoms of fungal infections, was especially well-received! The lectures concluded with a talk from Dr Eileen Paul (Northbank Communications) on the role communicating science to different sectors of society can play in promoting the success of biotechnology and life-science-based industries.

Judged by the students' comments and from the highly reproducible data obtained during the 3 weeks, the summer school was a great success. The young scientists enjoyed learning about fungi, carrying out a project that would help and inform other scientists and, perhaps most importantly, meeting like-minded students. To keep their interest burning, the SGM sent each of the students an information pack on careers in microbiology, and a copy of the latest issue of *Microbiology Today*.

The project was supported by the National Institute of General Medical Sciences (USA), Leica Microsystems UK Ltd and The University of Manchester.

Sue Crosthwaite
(e susan.k.crosthwaite@manchester.ac.uk)

In brief

Education Show

Birmingham NEC, 28 February–1 March 2008

SGM will be showcasing some new resources at the Education Show this year. Representatives from member schools must be sure to visit Stand U29, where a warm welcome will await. There will be something for everyone, with a completely new version of *The World of Microbes* for KS 2 and 3 available to take away and demonstrations of the multimedia A-level microbiology teaching pack. To register for the event, see **www.education-show.com**

Science in School

The latest issue of this quarterly European magazine, supported by the EU, is now available.

It is published by EIROforum and based at the European Molecular Biology Laboratory in Heidelberg. The content includes teaching materials, cutting-edge science, education projects, interviews with scientists, resource reviews and much more. The print version is in English, but multilingual versions are available online. Subscriptions are free at **www.scienceinschool.org**

Gradline aims to inform and entertain members in the early stages of their career in microbiology. If you have any news or stories, or would like to see any topics featured, contact **Jane Westwell** (**e** j.westwell@sgm.ac.uk).

The Career Word

You are busy with your research and if you focus all your energy onto the work in hand the future will look after itself – right? … Well not really, unless you have a fairy godmother who specialises in careers and, to be honest, they are fairly thin on the ground.

Luckily, universities, funding bodies, learned societies and professional associations all recognize the need to support scientists early in their careers and between them they provide a whole range of activities to support early-career scientists. We take a look at a few here that are particularly relevant to microbiologists.

R is for Roberts…

…or more precisely the late Sir Gareth Roberts who led a government-commissioned review of UK science and published a report in 2002. The Roberts review identified, among other things, a mismatch between the actual skills of graduate and postgraduate scientists and the skills employers wanted. The report recommended a range of measures to improve this situation. In response the Government made £20 million funding (Roberts money) available to implement a programme of activities. The emphasis for postgraduates being on employability and on career planning for postdoctoral scientists.

So much for the brief history lesson – how does this affect you? Some of the Roberts money was directed towards centrally funded activities, the rest was allocated to institutions with research council funded staff and postgraduate students. Your university almost certainly offers postgraduate skills training that was developed in response to the Roberts Review. Do you take full advantage of the courses and workshops that are on offer? Or do you reluctantly traipse along to one or two of them to keep your supervisor quiet? Maybe the programme in your university is not well publicised – if not, it is a good idea to check out what is available. A quick survey of university websites, revealed workshops on a range of topics including science communication skills, getting the most out of conferences, managing your supervisor and surviving your viva. These workshops may not seem directly relevant to your current situation and perhaps you view them as a distraction from your research. However, a PhD does not guarantee you a job and you can improve your employability by recognising and developing your transferable skills to present alongside your valuable research experience.

As a postdoctoral researcher, in an established and well-funded laboratory, it is easy to drift from one contract to another. Sometimes last-minute funding comes through and you can put off the inevitable decision for another few months or years, but postdoctoral research is not a permanent career and has a fairly short shelf life. For this reason careful career planning is essential for contract researchers. If you work in a poorly funded area, you may not have the cushion of a well-resourced lab and it is even more important to develop a clear career strategy. Roberts money has funded a range of activities for contract researchers in universities. Some supervisors recognize their responsibility to research staff in their teams; others may be less encouraging. Whatever your situation, you owe it to yourself (and it is your right) to take every opportunity to develop your career.

UKGrad

A major initiative to support UK-based postgraduate researchers is UKGrad (www.grad.ac.uk). One of its most successful activities is the long-running programme of personal development courses for postgraduate students, commonly known as GRADschools. There are two types of course: those organized on a regional basis and tailored to the needs of local students who can attend free of charge and, alternatively, national GRADschools that are organized by the central office and take place at a number of locations throughout the UK. The courses last 3 or 4 days and offer a chance to take a step back from your research and focus on transferable skills such as networking, communicating and team-working. The courses give vital breathing space and participants have a chance to reflect on their experience and to start to think about career options. Most students return to their research feeling revitalized and with increased motivation.

Students who are funded by a research council or the Wellcome Trust are entitled to a funded place on a national GRADschool. The SGM is aware that this leaves a large number of microbiology researchers with no obvious means of financing a place. As a result, the Society launched the GRADschool grant scheme and since 2007 we have been offering postgraduate student members of the society grants to cover the full fees of a national GRADschool course. Eligible students must have been members of the SGM for more than three calendar months, in the final or penultimate year of their studentship and be funded by organizations other than the Wellcome Trust or a research council. We allocated a handful of grants in 2007 and are hoping to see an increase in uptake in the coming year. It is a golden opportunity to attend a fabulous course, if you are eligible to apply for a grant you have nothing to lose – go for it!

GRADschool participant comment:

'I was able to put my own research into perspective. I found working and exchanging ideas with fellow students really rewarding.'

SGM and you

SGM is committed to supporting career development of early career microbiologists and offers a range of activities.

For many years we have been closely involved with the Life Science Careers Conferences. In recent years SGM has taken a lead role in organizing the conferences and carries out much of the background work. In addition to this, last November Jane Westwell and Lucy Goodchild from the External Relations Office travelled to University of Leeds, KCL and University of Bristol where the events took place.

Undergraduate and postgraduate students from all branches of bioscience had the chance to listen to speakers from a variety of career pathways. Talks included R&D in the pharmaceutical industry, teaching in schools, IP management, planning for an academic career, clinical sciences and CVs, interviews and job hunting. Refreshment and lunch breaks gave an opportunity to network with the speakers, learned societies and employers. Some delegates took part in a CV clinic where they received feedback on their CV in a one-to-one session with an experienced reviewer. The SGM exhibition stand was laden with copies of *Microbiology Today* and our careers information sheets.

Regular attendees at the SGM Spring meeting will know that we always arrange a skills development session for early career microbiologists. 2008 will be no different and we will be running a session entitled *Postdoc and beyond – planning for an academic career*. We will focus on three main themes: getting published, getting funded and getting that elusive lectureship. After the presentations the speakers will be glad to answer questions from the audience and we expect that the usual lively discussion will follow. The evening will end with drinks and a buffet.

As you can see, there is plenty of opportunity to attend courses and workshops tailored to the needs of 21st century research microbiologists. These are only a starting point; there are also a number of websites offering advice, shared experiences and points of view. University career advisory services are often undervalued by postgrad and postdocs, but can be an excellent resource.

There is lots of support and advice out there just waiting to be used so why not make time for yourself in 2008 to think about where you are heading in your career.

Jane Westwell, External Relations Office

Useful websites

www.biocareers.org.uk – microbiology careers website

www.grad.ac.uk – UKGrad, resources for postgraduate students including information on GRADschools

www.npc.org.uk – National Postgraduate Council

sciencecareers.sciencemag.org – articles, advice on all aspects of science careers

Science vs science communication – a fine balance

Gemma Walton is a postdoc at University of Reading. She has a keen interest in science communication and public engagement but hasn't yet made the decision to abandon research. In the meantime she takes every opportunity to have fun with the communication activities and gain useful experience.

Profile

Name Gemma Walton
Age 27
Present occupation Research Fellow (gut microbiology), University of Reading
Education University of Reading, *PhD Food Bioscience* (looking at whether prebiotics may offer some protection against colorectal cancer through changing the bacteria within the colon and their metabolic activities); Coventry University, *BSc Biological Sciences*.

What appeals to you about communicating science?

It's a great way to help people to understand why things happen. It's about taking concepts that people may not grasp and trying to relate them to life in a way that can be understood.

How do you balance your research commitments with science communication activities?

In a way the two can go hand in hand. The communications I have done to date have related to my research field – you have to find a little extra time to do these; but it's an enjoyable part of the work.

You appeared on TV – how did that come about?

My PhD supervisor was approached by the BBC to help with a programme in *The Truth About Food* series. He suggested I do it since I had some spare time after completing my thesis.

What was it like being on TV?

It was amazing! I was involved in the design of an experiment which essentially was feeding pre- and probiotics to a bunch of cowboys to look at their effect on gut health. I had to fly out to Colorado for some of the filming; everyone was really friendly, so I was very much at ease and it was great to see what goes on behind the scenes on a production. Back in Reading they did some more filming in the lab. Fiona Bruce interviewed me; she was also very friendly and down-to-earth, making it a great experience. I had plenty of opportunity to develop communication skills, especially in the US where pre- and probiotics are not well known.

What else have you been involved with?

In 2007, our research group was selected to display our research at the Royal Society Summer Science Exhibition. I was heavily involved in putting the stand together – from working with designers, editing the publicity leaflets, liaising with suppliers to actually putting the display together on set-up day. The visitors were from all walks of life – from school children to retired professors. We had a lot of interest with many people wanting to know more about their guts. The great thing about promoting this topic is that everyone can relate to it.

What transferable skills did you gain from this?

The obvious ones are organization and communication skills, but I would say that I really developed my project management skills. Co-ordinating the timely production of the display material, freebies and accompanying literature was a massive task although my colleagues made a big contribution too.

Have you taken up any training opportunities?

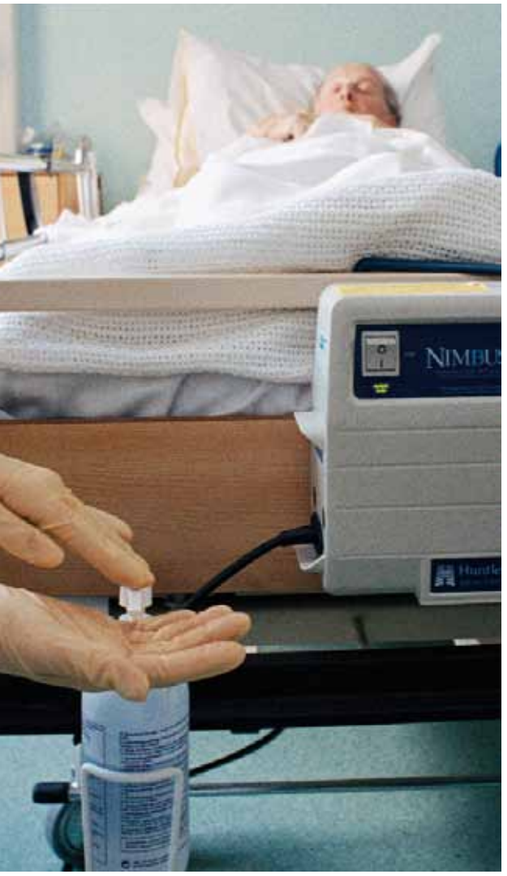
During my PhD, I attended a Life Science Careers Conference to find out more about career options. I also took part in Royal Society science communication training course prior to the summer exhibition.

What aspect of your work gives you the most job satisfaction?

In terms of my research getting results in projects is very satisfying. But I do also really enjoy science communication tasks. Being able to do both of these things in one job is great.

hotoffthepress

Science writer **Meriel Jones** takes a look at some recent papers in SGM journals which highlight new and exciting developments in microbiological research.



▲ A hospital hand wash. How effective are alcohol-based hand rubs against *Acinetobacter baumannii*? *Mark Thomas / Science Photo Library*

Alcohol hand rubs increase growth of *Acinetobacter*

Edwards, J., Patel, G. & Wareham, D.W. (2007). Low concentrations of commercial alcohol hand rubs facilitate growth of and secretion of extracellular proteins by multidrug-resistant strains of *Acinetobacter baumannii*. *J Med Microbiol* **56**, 1595–1599.

The medical profession continues to be haunted by serious bacterial infections acquired within hospitals. If the bacteria can survive antibiotics, as in the so-called multidrug-resistant strains, treatment can be very difficult. The classic measure of good hand hygiene can reduce infection rates by 10–50 %, so hospitals have introduced ways for staff to clean their hands frequently without suffering from chapped skin. These include using alcohol-based hand rubs (ABHRs) dispensed from a pump adjacent to patients' beds. These are solutions of ethanol or isopropanol, often in combination with an antiseptic and moisturiser which are less irritating to skin than soap.

Although studies have shown that ABHRs reduce infection rates, they are not effective against all bacteria. One example is the spores of *Clostridium difficile* that can cause very serious gastrointestinal infections. Now, Justin Edwards, Geeta Patel and David Wareham from Barts and The London NHS Trust and the MRC Clincal Sciences Centre of Imperial College London have found evidence that low concentrations of ABHRs enhance *in vitro* growth of *Acinetobacter baumannii*, an opportunistic pathogen of critically ill patients.

A. baumannii has emerged in recent years as an important cause of ventilator-associated pneumonia and blood infections in patients with burns, immunosuppression and critical illness. Many strains also turn out to be resistant to many antibiotics so that treatment is extremely difficult. The bacteria have a remarkable ability to persist on surfaces in the hospital environment and are spread by the hands of hospital staff so that hand hygiene is the key factor in preventing these infections.

The researchers therefore tested whether low concentrations of commercially available ABHRs could affect the growth of multidrug-resistant *A. baumannii*. Depending on the growth medium, the presence of 0.01–1 % ABHR resulted in increased growth of *A. baumannii*. In contrast, a hand-cleaning product that was free of alcohol and relied on strong detergents to kill bacteria did not support the growth of *A. baumannii* at all.

The researchers then investigated the proteins secreted by *A. baumannii* as it grew because previous work has shown that low concentrations of ethanol increase the virulence as well as the numbers of *A. baumannii*. They discovered that OmpA was a major secreted protein, along with another protein with an unknown role. OmpA is well known and may help the cells take up ethanol as a food source when other nutrients are in short supply. It may also help the bacterial cells sense each other and form films on surfaces.

The findings of this paper are certainly interesting, but their clinical significance remains unclear. However, if low concentrations of ABHRs do indeed exist in the clinical environment, this work may have implications for those hospitals currently experiencing outbreaks of *A. baumannii*.

A novel amniotic pathogen

Lawson, P.A., Moore, E. & Falsen, E. (2008). *Prevotella amnii* sp. nov., isolated from human amniotic fluid. *Int J Syst Evol Microbiol* **58**, 89–92.

Researchers from Göteborg, Sweden, and Oklahoma, USA, have identified a novel species of bacterium from human amniotic fluid. The bacteria were first recovered in 1999 from fluid that was described as turbid and ill-smelling. Similar bacteria were found again in amniotic fluid in 2006 and the authors have now worked out exactly what sort of bacteria they are. The cells will only grow in the absence of oxygen, which makes working with them rather difficult. The researchers first tried out the standard biochemical identification tests used with anaerobic bacteria, then commercial bacterial identification kits and finally they analysed the fats within the bacteria and the sequence of one gene.

A comparison of the sequence of the rRNA gene from the two isolates proved they were highly related to each other because the genes were 99.5 % similar. This gene is chosen for taxonomic studies because it changes very slowly over time. As a result, although researchers cannot give a precise correlation between a species definition and rRNA similarity, they consider that any difference over 3 % is significant. The similarity between the two strains and the closest named bacterium was 95.3 %. These bacteria were members of the *Bacteroides–Prevotella–Porphyromonas* group and the two isolates were especially close to members of the genus *Prevotella*. Many species of *Prevotella* have been isolated from people, from the mouth and from both healthy and infected tissues in the pelvic region. The rRNA sequence of the two isolates and the range of sugars and other chemicals that they were able to synthesize were different from all known *Prevotella* species. The closest relative was *Prevotella bivia* which was first identified in the endometrium and has frequently been detected in patients with pelvic inflammatory diseases.

In addition to these two bacterial strains, the researchers realised that several studies that had simply isolated DNA from the vagina of healthy women had recovered the same DNA sequence. It therefore looks like *Prevotella amnii* may be present all the time among the bacteria that inhabit the healthy vagina. However, in some circumstances its numbers increase and it can become a pathogen. The fact that the researchers have now discovered ways to identify this species means that routine hospital laboratories can be more confident in their identification of new opportunistic bacterial pathogens.

Something in the air

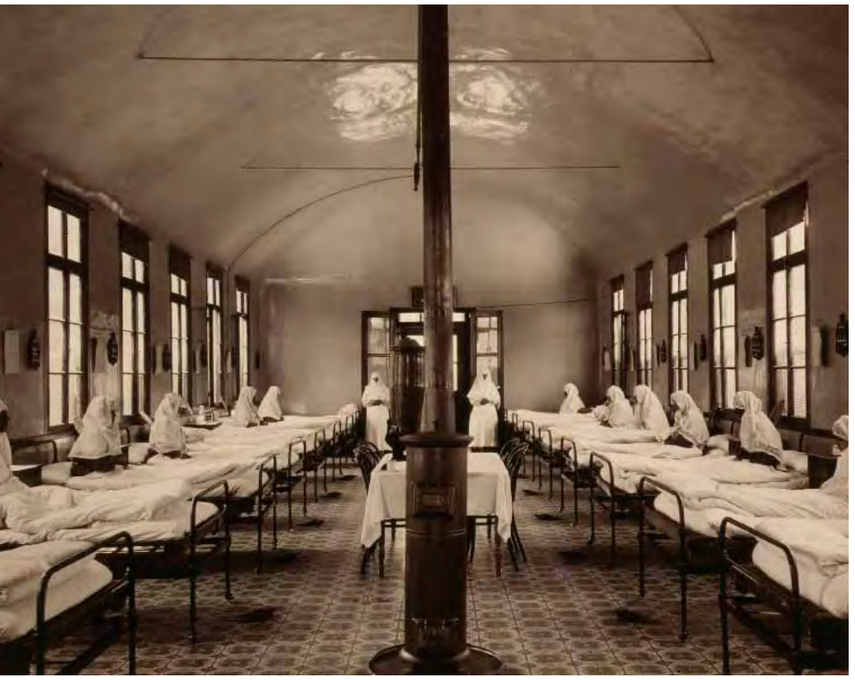
Ogata, N. & Shibata, T. (2008). Protective effect of low-concentration chlorine dioxide gas against influenza A virus infection. *J Gen Virol* **89**, 60–67.

'Flu, caused by the influenza A virus, is a continuing health problem. It infects around one-fifth of the world's population yearly, causing symptoms that range from very minor to a severe illness. Indeed, 'flu is a factor in the death of around half a million people every year. Some pandemic strains have caused more severe mortality, such as the 1918 pandemic that killed 20–50 million people worldwide. Measures to counteract 'flu include vaccination, which only has short-term and partial success, and antiviral drugs, which all have drawbacks, such as toxicity to the sufferers and the emergence of resistant influenza strains.

The 'flu virus is spread in the air as patients cough and sneeze. Researchers at the Taiko Pharmaceutical Co. Ltd in Japan have been wondering whether there is a way to deal with the virus when it is in this airborne form, so reducing the opportunities for infection. There are many chemicals that are very effective at killing viruses, but most are also toxic to people. The researchers investigated chlorine dioxide (ClO_2) gas, which has a well-known and very effective antimicrobial action when dissolved in water. It has been used to disinfect public supplies of drinking water since the 1950s and is also used in food industries. Air containing low levels of ClO_2 is considered safe to breathe, with the US Occupational Health and Safety Administration setting an upper limit of 0.1 parts per million (p.p.m.) for ClO_2 in the workplace. Norio Ogata and Takashi Shibata wondered whether even this low level could be useful against the 'flu virus.

▶ A crowded public space. Could the addition of chlorine dioxide to the air in public buildings help to prevent the spread of influenza? *Jupiter Images*

They arranged for small groups of mice to inhale a 'flu aerosol for 15 minutes, counted the number of virus particles in the lungs of the mice and recorded their health 16 days later. The air breathed by half of the mice also contained about 0.03 p.p.m. ClO_2 gas. This gas reduced the number of virus particles by over 1000-fold 3 days later and all the mice were alive 16 days later. In contrast, seven out of the 10 mice that had inhaled the viral particles alone had died. The ClO_2 gas had to be present at the same time as the virus in the air; breathing it even 15 minutes later completely cancelled the protective effect. The reason was that the gas damages proteins on the outer surface of the virus that are essential for attachment to mammalian cells to start an infection. The authors suggest that ClO_2 gas could therefore be used to disinfect the air in places such as airports, hotels, offices and schools without the need to close the buildings and thus disrupt the flow of normal life.



◀ A 19th-century tuberculosis ward. This is the Haskoy Hospital for Women in Constantinople (present-day Istanbul), then the capital of the Ottoman Empire. Tuberculosis (TB) is a bacterial infection, usually of the lungs, that was a widespread cause of death before a vaccine was developed in the 20th century. In the late 19th century it was discovered that the disease was contagious, and hospitals were created to quarantine patients and prevent the spread of the disease. This photograph, by the Abdullah Brothers, was taken in the period from 1880–1893.*Library of Congress / Science Photo Library*

Export blockade

McCann, J.R., McDonough, A., Pavelka, M.S. & Braunstein, M. (2007). *β*-Lactamase can function as a reporter of bacterial protein export during *Mycobacterium tuberculosis* infection of host cells. *Microbiology* **153**, 3350–3359.

Tuberculosis continues to kill millions of people slowly every year. It is caused by the bacterium *Mycobacterium tuberculosis*. Few drugs are effective against this species and the bacteria have very effective strategies to thrive within the body. For example, they colonize macrophages, a type of human cell that usually destroys invading bacteria. To do this the bacterium secretes proteins to ensure survival through interference with the body's defence systems. *M. tuberculosis* has at least four systems to export proteins from the cells. The one that is probably responsible for most protein secretion is the general secretion pathway (Sec), but the twin-arginine translocation pathway (Tat) is also important. The pathways recognize signals on the proteins to identify which ones to export, but also appear to export proteins that lack conventional signals. *M. tuberculosis* secretes proteins when grown alone in laboratory cultures, but a different set appears to be made and exported during infection of the host. Researchers think that vaccines or drugs to prevent export of these proteins could be very effective therapies. Unfortunately, it has proved difficult to even identify them since it is difficult to collect bacterial proteins from infected human tissues.

Researchers in the USA have now devised a system to identify and study these elusive proteins by turning an antibiotic resistance mechanism against the bacterial cells. The trick is to use a test where the exported protein, in this case *β*-lactamase, is essential for the bacteria to survive. *β*-Lactamase is an enzyme that many bacteria use as protection against the antibiotic penicillin. Since penicillin interferes with synthesis of the bacterial cell wall, the *β*-lactamase must be outside the cell to provide protection. *M. tuberculosis* is normally resistant to penicillin so the researchers created a sensitive strain by removing the protective gene, called *blaC*. They then linked the *β*-lactamase gene to any gene where they suspected that the encoded protein might be exported. Mammalian cell cultures were infected with the bacteria and penicillin was added. If the cells really did export the protein, the *β*-lactamase was exported as well and protected the bacteria from the antibiotic. If the protein was not exported, the bacteria died.

The idea was tested with several *β*-lactamase genes using proteins known to be exported by the Tat and Sec pathways to come up with a reliable system. Shortened versions of the *Escherichia coli* TEM-1 *β*-lactamase gene as well as *M. tuberculosis* BlaC worked well with several export signals. The cultured mammalian cells were very like macrophages, so the researchers think that they now have a way to identify the most interesting category of proteins, namely those that are exported by *M. tuberculosis* during intracellular growth.

goingpublic

The Society has a broad programme of activities to promote microbiology to the public, the media, opinion-formers and policymakers. 2007 has seen some great successes, as **Lucy Goodchild** reports. Contact Lucy at **l.goodchild@sgm.ac.uk**

Microbiology in the Media

SGM September 2007 meeting, Edinburgh

For every SGM meeting, the public affairs office produces press releases to get the research presented to the public. The plenary session this time was '*Food, fluids, fingers, faeces and flies: food- and water-borne pathogens*', so we knew there would be some newsworthy stories.

We selected over 30 abstracts to be the subjects of press releases. After asking the authors whether they would like to be involved, just 15 abstracts remained. Topics ranged from Hepatitis E in European pork to the self-assembly of the *Clostridium difficile* protein coat and its applications in nanotechnology. The press releases were produced in close collaboration with the authors and sent to our extensive list of reporters a week before the meeting. We also made them available to journalists on EurekAlert and AlphaGalileo, two online science news centres.

The meeting started on Monday 3 September 2007. As I was the main contact for the press releases on site, I made sure I had my mobile by my side from the beginning. Sure enough, media calls began to pour in. We had enquiries from all over the world about the research being presented and a variety of publications were interested in the stories, from *Grocer* magazine through to BBC online and *Nature*. I was kept rather busy over the next few days and the calls kept coming for weeks after the meeting.

I am still keeping tabs on the coverage of the meeting. The Society was mentioned in hundreds of places, both in print and online. Some of my favourite coverage included articles in *Le Monde*, *De Standaard*, www.thepoultrysite.com, United Press International and Ten to the Minus Nine.

FIS 2007

The Federation of Infection Societies holds an annual conference. In 2007, SGM was the conference's host society, and was responsible for publicizing the research to the media.

At first glance, there was a plethora of brilliant news stories. Research covered new and existing superbugs, which always make the front pages. However, we encountered two problems. The first was selecting relatively few stories from so much newsworthy research and the second was finding authors willing to participate. Medics seem to be strangely reticent in this respect. Despite all our efforts, only four scientists agreed to press releases.

I was unable to attend the conference, so yet again I made sure my contact details were clear on the press releases. Although I had received a taster during SGM's September meeting, nothing could have prepared me for the response. One of the press releases, about a new strain of community-acquired MRSA, was selected for promotion by the Press Association.

On Tuesday 27 November 2007, the day before the conference was due to start, the phone began to ring first thing in the morning. The pace increased and before long I was answering three phones. We had calls from UK broadsheets and tabloids, as well as television channels. The journalists wanted to speak to the author, Dr Marina Morgan from the University of Exeter. Marina was on her way to the conference in Cardiff, but she took all the calls nonetheless and was exactly what the press were looking for.

On the first day of the conference, news cameras appeared and the story made almost all of the national newspapers, as well as many regional ones. Dr Chris talked about it on *This Morning* on TV and I was happy to hear a news bulletin on Marina's research while I was on my way to the airport. With Marina's help, we certainly reached a wide audience with the research!

Press briefing at the Science Media Centre

Microbes and climate change

Climate change has been at the top of the public agenda for a while and we had become increasingly concerned that the important role of microbes was hardly mentioned. To redress this, we decided to run a press briefing on the topic.

The microbial aspect of climate change is notoriously complex, so we first had to break it down into manageable chunks. We split the briefing into four sections, based on climatically important gases: microbial methane production, carbon dioxide and ocean acidification, nitrous oxide and microbes, and oceanic dimethyl sulphide production.

The briefing was held on 10 December 2007 at the Royal Institution in London. The national news media were invited, as were press officers from contributing universities. The speakers – James Chong (University of York), Ian Joint (Plymouth Marine Laboratory), Mark Trimmer (Queen Mary, University of London) and Michael Steinke (University of Essex) – gave exceptional presentations; everybody was enthralled. Four journalists attended, from *Science*, the *Daily Mail*, the Press Association and BBC Radio 4. We had chosen a 'busy news day' for our briefing, so the turnout was less than we had hoped.

However, the coverage was exciting. During the briefing, we had enquiries from the *Guardian* and the *Telegraph*, both unable to attend but interested in the stories. The speakers were interviewed immediately after the briefing for Radio 4's *Farming Today*, which aired the following morning at 5.45am, and the *Daily Mail* ran a fantastic piece on methanogens. The stories were a hit online, and were chosen as blog topics in several languages. Perhaps most exhilarating was the subsequent appearance of a £1 million anaerobic digester on a farm in *The Archers*, exactly what Dr Chong had been talking about!

Science and the Parliament 2007

The Royal Society of Chemistry (RSC) hosts 'Science and the Parliament' annually, to encourage scientists and politicians to debate key scientific issues. The 2007 event focussed on 'Energy and Climate Change – The Science Behind the Energy Debate'. SGM has a stand at the event every year, and this year Executive Secretary Ron Fraser and I attended the event at Our Dynamic Earth in Edinburgh.

Since microbes and climate change had featured so heavily in our activities in 2007, we were very excited about this event; it gave us the opportunity to bring the subject to life. SSERC very kindly lent us a digital microscope and laptop to allow delegates to see the display and helped us to set up. We showed a mix of algae and protozoa, which is lively and exciting, to highlight the climatic importance of photosynthetic microbes.

Even before the delegates arrived, people were transfixed. Staff at Our Dynamic Earth were creeping over hesitantly and asking is 'that happening right now?' They were fascinated by the protozoa, and surprised to hear about the role of algae in climate change. Many people commented that they were pleased to see us acknowledging the importance of microbes in climate change.

The Scottish Government will consider a Climate Change Bill this year and we hope that micro-organisms will be taken into account, both as an important aspect of modelling and as a key to tackling greenhouse gas emissions.

SGM could not carry out these promotional activities without input and help from members. If you would like to be included in our database of experts, contact Lucy, or click on the link for the form at the bottom of the SGM website noticeboard page (www.sgm.ac.uk/noticeboard.cfm).

Microbiology in social media

www.micropodonline.com

'New media' is increasing in popularity all the time. Podcasts pop up every day and every other person has a blog. There is a small group of websites dedicated to microbiology, with *Microbiologybytes* and *Microbeworld* leading the way. However, there was no such website dedicated to providing microbiology news, information and entertainment for the general public… until now.

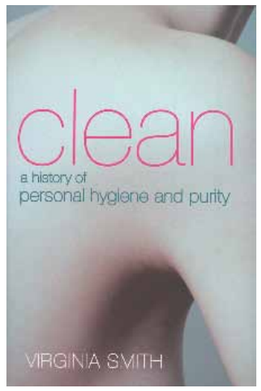
I have been working with Dr Lucy Harper, Communications Officer at the SfAM, to develop a brand new portal brimming with news, views, information and fun – all with a microbiological theme. Our aim is to engage with the public about microbiological issues, covering bird 'flu, home hygiene and giant fungi among many others.

Micropod was launched on 17 December with the theme 'The microbiology of Christmas'. In the first podcast, we asked residents of Reading, 'will you be eating turkey this Christmas?' We discussed the many and varied microbiological aspects to the festive season and I interviewed astrobiologist Lewis Dartnell about life in the snow. Our first blogs were about bird 'flu and the festive increase in STIs.

This year's topics will include hygiene and cleanliness, bugs in space and GM. So far, we have an editorial board of four people, but we are looking for additional helpers. If you are interested in getting involved and you are willing to attend the occasional virtual meeting (just doing our bit to save the planet), please drop us a line.

reviews

If you would like your name to be added to our database of book reviewers, please complete the book reviewer interests form at **www.sgm.ac.uk**. A classified compendium of reviews from 1996 to the present is also available on the website.



Clean: A History of Personal Hygiene and Purity

By V. Smith
Published by Oxford University Press (2007)
£16.99 pp. 457
ISBN 978-0-19-929779-5

Clean tells the story of hygiene from ancient Egypt to the modern world. Virginia Smith documents the changes in practices and investigates the motivations behind these changes. It is so easy to see hygiene and cleanliness as a necessity, especially with the rise in antibiotic-resistant infections like MRSA, but this has not always been the case. Prior to the public acceptance of germ theory, hygiene and cleanliness had several roles. In ancient Egypt, the focus was on cosmetics and beauty, and in Rome, the new Galenic medicine advocated cleanliness to avoid illness. Religious asceticism followed, compelling people to remain clothed and avoid washing to remain morally pure. Hygiene was not simply influenced by disease, but also by religion, fashion, politics and technical advances. The book is a comprehensive study of the history of hygiene. It is written beautifully and is not only accessible but also utterly fascinating. The notes are informative and useful but do not detract from the story. This is a must read for anybody interested in history and especially for those who have an interest in public health.

Lucy Goodchild, SGM

Parasites and Infectious Disease Discovery by Serendipity, and Otherwise

By G.W. Esch
Published by Cambridge University Press (2007)
£23.99/US$45.00 pp. 355
ISBN 0-52167-539-0

The 15 chapters or essays in this book vary greatly. Some tell stories, for example how the cause of malaria was discovered, and how a toad and its fluke parasite, water-loving creatures, survive in a desert. Other chapters discuss important issues. Why is vaccination highly effective for some diseases but has no prospect of success with others? Did the elimination of a highly debilitating disease (hookworm) from an area lead to an increase in prosperity, or did improvement in living conditions eradicate the disease? How can medication be delivered to an impoverished and ill-educated population?

Many of the essays are interesting and lucidly written. The book is, however, a very unusual one and has shortcomings. The essay section of about 240 pages comes after a prologue of over 100 pages describing meetings with the 18 experts consulted and their life stories. I feel that cogent comments of the experts in these pages should have joined their comments in the essays, and that brief biographical details would have been better at the end of the book. For seven essays, which concern diseases caused by animal parasites such as flukes and hookworms, a microbiologist needs to have a textbook of parasitology or invertebrate zoology handy. A glossary and a few life cycle diagrams would have made these chapters easier for microbiologists. The writing is informal and colloquial, which is not always helpful – in one chapter I was more than usually surprised by the prescience of Darwin, and had to remind myself that 'Darwin' is the given name of one of the experts – nor did I need frequent information along the lines of 'The taxi fare to Chelsea was £10'. The index of the book is strangely constructed and not very helpful. Amazingly, considering that the author is the editor of a scientific journal and the distinguished publishers have over 400 years experience of book production, typos are numerous. I suspect from a text comment that the typos and the occasional less well-organized chapter are the result of an excessive rigidity with respect to deadlines, and a final rush to complete the book.

In spite of the above criticisms the book is valuable for some well-told stories and the thoughts of the author and his experts. It is appropriate for purchase by libraries of universities and institutes with microbiology departments; this would also permit individual microbiologists to peruse and consider whether they wanted a copy.

Finally, what about the 'serendipity' featuring in the subtitle? Having started by considering it to be a major factor in discovery, in the end the author settles for 5–10 %, with well-planned investigations being the major route to discovery. A conclusion perhaps not too far from the aphorism on genius attributed to Thomas Carlyle – 1 % inspiration and 99 % perspiration.

Michael Carlile, Bridgwater

Manual of Clinical Microbiology, 9th edn, vols 1 and 2

Edited by P.R. Murray, E.J. Baron, J.H. Jorgensen, M.L. Landry & M.A. Pfaller
Published by American Society for Microbiology (2007)
US$209.95 pp. 2,488
ISBN 1-55581-371-0

While the size and weight of each of these two volumes are a strong disincentive to carrying them around in one's briefcase, they assuredly deserve a place on the shelf of every medical microbiology department. The first volume deals broadly with bacteriology and related subjects such as antibiotic susceptibility testing, while volume 2 covers virology, fungi and parasites. Despite the use of the word 'clinical' in the book's title, a number of chapters are more 'academic', covering detailed molecular and biochemical information on the mechanisms of resistance to antibiotics, antivirals, antifungals and antiparasitic agents. In addition to detailed information on a comprehensive range of pathogens, the initial chapters deal with the complementary and important issues of laboratory management, design, information technology and storage of micro-organisms. Other general chapters of value to those working at the bench cover disinfection and sterilization, and the control of laboratory-acquired infections. Furthermore, in the current climate where there is much media coverage and political interest in the subject of healthcare-associated infections, it is reassuring to note that there are two short but well-written and focussed chapters on infection control epidemiology and laboratory procedures for the epidemiological analysis of micro-organisms.

The format of the book, which comprises dense text in a fairly small font, will not attract the casual reader. Indeed, I would not feel generally inclined to recommend it to first-year microbiology students or those just starting in the field of clinical microbiology as there are other more accessible tomes available for the beginner, although one notable exception to this was the short chapter on the taxonomy and classification of viruses. Nonetheless, it is the very intensity of the information provided that gives this book its strength. For those seeking a ready source of information on matters relating to the practice of clinical microbiology, this surely should be one of the first ports of call. Despite the density of the text, the judicious use of headings, subheadings and tables makes the retrieval of specific information fairly straight forward. This is also helped by an extensive and detailed subject index. A number of the chapters also contain helpful diagrams, with several in colour. The latter are particularly valuable when showing features such as the appearance of bacterial colonies growing on agar plates, Gram-stained films or pathological material. Although it was not available for me to review, a CD-ROM with close to 500 illustrations from the book is also available for purchase through the publisher.

Despite the fact that there were over 250 contributors to these two volumes, the text appears remarkably cohesive. Indeed, in the Preface the Editor-in-Chief candidly reveals that he and the other Editors had their work cut out trying to achieve consistency. However, they are to be congratulated on achieving their objective. Interestingly, approximately 30 % of the authors for this edition were from outside the USA. This is a welcome development in terms of moving away from a purely American perspective of the field and is likely to broaden the potential readership of the book.

In summary, this book is highly recommended to those involved in the field of clinical microbiology, be their specialty bacteriology, virology, mycology or parasitology. The price makes it unlikely that many individuals will purchase personal copies, although given the amount of information contained in each volume, there is a case to be made that the book is good value for money. With this in mind I would strongly recommend that microbiology departments purchase copies for their staff.

Alan Johnson, Health Protection Agency

Reviews on the web

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

- *Genomics and Evolution of Microbial Eukaryotes*
- Listeria*, Listeriosis, and Food Safety, 3rd edn*
- *Enzyme-mediated Resistance to Antibiotics Mechanisms, Dissemination, and Prospects for Inhibition*
- *Molecular Genetics of Bacteria, 3rd edn*
- *Modern Soil Microbiology, 2nd edn*
- *Adenovirus Methods and Protocols, Vols 1 and 2, 2nd edn*
- *Bacterial Pathogenomics*
- *Exploitation of Fungi*
- *Gene Cloning and Manipulation*
- *Virology Principles and Applications*
- *Hospital Acquired Infections Power Strategies for Clinical Practice*
- *Crash Course: Infectious Diseases*
- *Master Medicine. Microbiology and Infection, 3rd edn*
- *The Microbiology Bench Companion*
- *Advances in Food Diagnostics*
- *Superantigens: Molecular Basis for Their Role in Human Diseases*
- *Encyclopedia of Infectious Diseases: Modern Methodologies*
- *Methods for General and Molecular Microbiology, 3rd edn*
- *Virulence Mechanisms of Bacterial Pathogens, 4th edn*
- *Microbial Biodegradation: Genomics and Molecular Biology*
- *The Cyanobacteria: Molecular Biology, Genomics and Evolution*
- *Models of Exacerbations in Asthma and COPD*
- *Animal Viruses: Molecular Biology*
- *Coronaviruses: Molecular and Cellular Biology*
- *Sex in Fungi: Molecular Determination and Evolutionary Implications*
- *Methods for Computational Gene Prediction*
- Acinetobacter*: Molecular Biology*
- *Antimicrobial Susceptibility Testing Protocols*
- *Segmented Double-stranded RNA Viruses: Structure and Molecular Biology*

comment

microbes as climate engineers

Human activity is a big factor in global warming and politicians around the world are trying to agree some control measures. But as **Dave Reay** reveals, they cannot afford to ignore the role played by microbes in climate change.

'The Anthropocene' is the name given by Nobel Laureate Paul Crutzen to the epoch we now live in. Ours is an age where global climate is increasingly determined by humankind, one where our species continues to alter the composition of the atmosphere and the energy balance of the planet. Arrogant organisms that we are, it is easy to view this as something entirely novel in Earth's history – evolution's newest top consumers breaking the environmental shackles and dictating global climate. In truth of course, micro-organisms have been at it for billions of years. From the first molecule of oxygen released by a cyanobacterium in the turbulent oceans of a young Earth, to the methanogen-made CH_4 belched from the warm bogs of the Carboniferous, microbes have long helped determine the composition of Earth's atmosphere and its climate.

Both natural and human-induced fluxes of CO_2, CH_4 and N_2O are dominated by microbiology. In the sea, CO_2 uptake by phytoplankton provides the pump for the annual drawdown of 90 billion tonnes of carbon from the atmosphere, with microbial decomposition and respiration returning much of this to the atmosphere. On land, the huge amount of carbon stored in soils and vegetation is under continual attack from microbes, the balance between primary production, respiration and decomposition resulting in the uptake of around 120 billion tonnes of carbon each year, and the loss of about 119 billion tonnes.

For CH_4, the flagella-print of micro-organisms on the atmosphere is even more apparent. The world's wetlands pour over 100 million tonnes of CH_4 into the atmosphere each year as a result of microbial methanogenesis. This amount would be far greater if it was not for the significant proportion used by methanotrophic bacteria before it can escape into the atmosphere. The CH_4 that does escape is still not free from their attentions; high affinity methanotrophs utilize atmospheric CH_4 at a rate of ~30 million tonnes each year. Along with additional CH_4 release from gas hydrates and microbial methanogenesis in the oceans, the world's termite population produces an additional 20 million tonnes of CH_4 every year courtesy of the methanogenic bacteria in their guts. Aside from energy-related sources like fossil fuel extraction, most human-induced CH_4 emissions come from ruminant livestock, rice cultivation and landfill, adding around 150 million tonnes to the atmosphere annually, all derived from the methanogens that thrive in these carbon-rich environments.

N_2O has a sobering global warming potential of 298 (this is the warming produced per kg of a gas over a 100-year time horizon relative to 1 kg of CO_2). Again, it is microbes that dominate global emissions. For every tonne of reactive nitrogen that human activities add to the biosphere, between 10 and 50 kg end up being emitted into the atmosphere as N_2O.

It is not only via reactive nitrogen that we are changing microbial greenhouse gas emissions. Through our post-industrial emissions, we have enhanced global warming. Average temperatures have risen by 0.7 °C in the last 100 years, with a projected increase in the 21st century of between 2 and 4.5 °C. From enhanced soil carbon decomposition rates to elevated wetland methanogenesis, the impact of these microbially mediated feedbacks on further climate warming in the 21st century is potentially huge.

The role of microbes as climate engineers is evident, as is their potential to exacerbate the problems of enhanced global warming driven by our burning of fossil fuels. But all is not lost in the world of climate microbiology. In the abundance and diversity of microbial life on our planet may lie the roadmap by which we can better navigate the Anthropocene. Through a better understanding of microbial decomposition of organic matter, there is the potential to alter land-management practices to conserve or even enhance soil carbon storage. By inducing increased primary production in the oceans, by adding iron or reactive nitrogen, there exists the possibility of sequestering more CO_2 from the atmosphere. Already the methanotrophs in landfill cover soils are playing a vital role in intercepting the vast quantities of CH_4 produced below, cyanobacteria are being explored as providers of hydrogen fuel, and vats of phytoplankton are being grown as the feedstock for biofuels.

Microbes will continue as climate engineers long after humans have burned that final barrel of oil. Whether they help us to avoid dangerous climate change in the 21st century or push us even faster towards it is dependent on just how well we understand them.

▲ The author measuring methane emissions from a peatbog in northern England. *D Reay*

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